Kinetic Control
The Management of Uncontrolled Movement

Mark Comerford, BPhty, MCSP, MAPA
Director, Movement Performance Solutions

Sarah Mottram, MSc, MCSP, MMACP
Director, Movement Performance Solutions
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This book presents a comprehensive system for the assessment and retraining of movement control. It has been in evolution for the last 25 years.

Uncontrolled movement has a significant impact on the development of movement disorders and pain. The scientific support for the process of the assessment and retraining of uncontrolled movement has been steadily expanding particularly in the last 10 years. The influence of uncontrolled movement on symptoms, especially pain, movement function, recurrence of symptoms and disability is now well established. We believe that in the next 10 years the literature will support that the presence of uncontrolled movement will also be recognised as a predictor of injury risk and as having an influence on performance.

Uncontrolled movement can be identified by movement control tests. People with pain demonstrate aberrant movement patterns during the performance of these movement control tests. A growing body of evidence supports the use of movement control tests in the assessment and management of chronic and recurrent pain. The identification of uncontrolled movement in terms of the site, direction and threshold of movement impairment is a unique subclassification system of musculoskeletal disorders and pain. The movement testing process proposed enables the classification of uncontrolled movement into diagnostic subgroups that can be used to develop client-specific retraining programs. This process can determine management priorities and optimise the management of musculoskeletal pain and injury recurrence. Subclassification is now recognised as being the cornerstone of movement assessment and the evidence for subclassification of site, direction and threshold is growing. This book details a structured system of testing, clinical reasoning and specific retraining. This system does not preclude other interventions as it is designed to enhance the management of musculoskeletal disorders.

The Kinetic Control process has come a long way in last 25 years. The motivation for the development of the Kinetic Control process was to find a way to blend the new and exciting concepts in movement dysfunction into an integrated clinical process, built on the foundation of a solid clinical reasoning framework. Our aim is to gain a better understanding into the inter-relationship between the restrictions of movement function and movement compensations. The breakthrough came with the realisation that some compensation strategies are normal adaptive coping mechanisms and do not demonstrate uncontrolled movement, while others are maladaptive compensation strategies that present with uncontrolled movement. This led us to develop the structured assessment process detailed in this text including the Movement Control Rating System (Chapter 3). This clinical assessment tool can identify movement control deficiencies and be valuable for reassessing improvements in motor control efficiency.
Recurrent musculoskeletal pain has a significant impact on health care costs, employment productivity and quality of life. Uncontrolled movement can be identified by observation, and corrective retraining of this uncontrolled movement may have an influence on onset and recurrence of symptoms. To date, outcome measures in terms of changes in range and strength, have not influenced the onset and recurrence of injury. The ability to assess for uncontrolled movement and to retrain movement control is an essential skill for all clinicians involved in the management of musculoskeletal pain, rehabilitation, injury prevention, and those working in health promotion, sport and occupational environments. Preventing the recurrence of musculoskeletal pain can both influence quality of life and have an economic impact.

Movement control dysfunction represents multifaceted problems in the movement system. Skills are required to analyse movement, make a clinical diagnosis of movement faults and develop and apply a patient-specific retraining program and management plan to deal with pain, disability, recurrence of pain and dysfunction. The mechanisms of aberrant movement patterns can be complex, so a sound clinical reasoning framework is essential to determine management goals and priorities. We present an assessment framework which will provide the option to consider four key criteria relevant to dysfunctional movement: the diagnosis of movement faults (site and direction of uncontrolled movement), the diagnosis of pain-sensitive tissues (patho-anatomical structure), the diagnosis of pain mechanisms and identifying relevant contextual factors (environmental and personal). This clinical reasoning framework can help identify priorities for rehabilitation, where to start retraining and how to be very specific and effective in exercise prescription to develop individual retaining programs.

Uncontrolled movement can be reliably indentified in a clinical environment and related to the presence of musculoskeletal pain, to the recurrence of musculoskeletal pain and to the prediction of musculoskeletal pain. We hope this text will enable clinicians worldwide to effectively identify and retrain uncontrolled movement and help people move better, feel better and do more.

Mark Comerford
Sarah Mottram
2011
Comerford and Mottram are to be commended for their extensive and comprehensive presentation of factors involved in movement dysfunctions. This book shares several of my own strong beliefs that have implications for the management of musculoskeletal pain conditions. Those beliefs are: 1) recognising and defining the movement system; 2) identifying and describing pain syndromes based on movement direction; 3) identifying the primary underlying movement dysfunction; 4) describing the various tissue adaptations contributing to the movement dysfunction; and 5) developing a treatment program that is comprehensive and based on the identified contributing tissue adaptations. I also share with the authors a belief that the treatment program requires the patient’s active participation, which can range from control of precise, small, low force requiring movements to total body large force requiring movements. Historically – and still prevalent – is the belief that tissues become pathological as an inevitable outcome of trauma, overuse and ageing. The result is a focus on identifying the patho-anatomical structure that is painful rather than on identifying the possible contributing factors, or even how movement faults can be an inducer. We are all aware that movement is necessary to maintain the viability of tissues and bodily systems. Almost daily, studies are demonstrating the essential role of movement, in the form of exercise or activity, in achieving or maintaining health. Yet there is very little recognition that there are optimal ways of moving individual joints and limb segments as well as the total body. Similarly there is little recognition that painful conditions can be treated by correcting the movement rather than resorting to symptom-alleviating modalities, drugs or surgery. Optimal alignment when maintaining prolonged postures, such as sitting, is not considered to be necessary. I believe the situation is analogous to that of diet. For many years, no one worried about the effect on a person’s health of the type or amount of food that was consumed. Indeed, more money is still spent on the alignment of the teeth than on the alignment of the body, though the function of the body is more affected by alignment faults than eating is by poor alignment of the teeth.

This book serves to reinforce and define the characteristics of the movement system and how they contribute to movement dysfunctions associated with pain syndromes. The authors have done an extensive review of the relevant literature describing the dysfunctions of the nervous and muscular systems. They have provided a detailed description of a key underlying factor, designated as uncontrolled movement, which then provides a basis for the treatment program. The detailed descriptions of the syndromes, key observations and examination forms should be most helpful in guiding the clinician. Building upon the information taken from the examination, the treatment program is also described in detail. What is particularly noteworthy is the incorporation of most of the perspectives and methods used by the best known
approaches to musculoskeletal pain. The authors have organised the rationale and methods from these varying approaches into a comprehensive approach. Comerford and Mottram have done a thorough job of describing all aspects of what could be considered the ‘psychobiosocial’ model of analysis and treatment of musculoskeletal pain. The timeliness of this book is reflected by the incorporation of their concepts to the International Classification of Functioning, Disability, and Health. As stated previously this book has its particular value in the comprehensiveness and detailed descriptions of possible tissue dysfunctions as reported in the literature, methods of analysis and treatment. The reader will be truly impressed by the many complexities of the movement system and the rigorous analysis that is required to understand, diagnose and treat the dysfunctions that can develop and contribute to pain syndromes. The authors have truly provided an outstanding text in its inclusive and thorough discussion of the topic of movement dysfunction.

Shirley Sahrmann, PT, PhD, FAPTA
Professor Physical Therapy, Neurology, Cell Biology and Physiology
Washington University School of Medicine – St. Louis
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Reviewers

Technical Reviewer
Prue Morgan  M.App.Sc (Research), B.App.Sc (Physio)
Grad Dip Neuroscience
Specialist Neurological Physiotherapist, FACP
Lecturer, Physiotherapy
Monash University

Philippa Tindle  BSc. BA. MCSP
Member of Chartered Society of Physiotherapy and
Registered with the Health Professions Council (HPC)
Uncontrolled movement

The key to managing movement dysfunction is thorough assessment. This includes the determination of any uncontrolled movement (UCM) and a comprehensive clinical reasoning process by the clinician to evaluate contributing factors which influence the development of UCM. This first chapter details the concept of UCM and the clinical reasoning process which is the framework for assessment and rehabilitation.

UNDERSTANDING MOVEMENT AND FUNCTION

Normal or ideal movement is difficult to define. There is no one correct way to move. It is normal to be able to perform any functional task in a variety of different ways, with a variety of different recruitment strategies. Optimal movement ensures that functional tasks and postural control activities are able to be performed in an efficient way and in a way that minimises and controls physiological stresses. This requires the integration of many elements of neuromuscular control including sensory feedback, central nervous system processing and motor coordination. If this can be achieved, efficient and pain-free postural control and movement function can be maintained during normal activities of daily living (ADL), occupational and leisure activities and in sporting performance throughout many years of a person’s life.

The movement system comprises the coordinated interaction of the articular, the myofascial, the neural and the connective tissue systems of the body along with a variety of central nervous system, physiological and psycho-social influences (Figure 1.1). It is essential to assess and correct specific dysfunction in all components of the movement system and to assess the mechanical inter-relationships between the articular, myofascial, neural and connective tissue systems. This chapter will describe a systematic approach to evaluation of the movement system and identification of the relative contributions of individual components to movement dysfunction.

Movement faults

Identifying and classifying movement faults is fast becoming the cornerstone of contemporary rehabilitative neuromusculoskeletal practice (Comerford & Mottram 2011; Fersum et al 2010; Sahrmann 2002). In recent years clinicians and researchers have described movement faults and used many terms to describe these aberrant patterns. These terms include substitution strategies (Richardson et al 2004; Jull et al 2008), compensatory movements (Comerford & Mottram 2001a), muscle imbalance (Comerford & Mottram 2001a; Sahrmann 2002), faulty movement (Sahrmann 2002), abnormal dominance of the mobiliser synergists (Richardson et al 2004; Jull et al 2008), co-contraction rigidity (Comerford & Mottram 2001a), movement impairments...
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- The management of uncontrolled movement syndromes are seldom caused by isolated events; and that habitual movements and sustained postures play a major role in the development of movement dysfunction. These statements have been fundamental in the development of the movement dysfunction model. Clinical situations which have a major component of movement dysfunction contributing to pain include: postural pain; pain of insidious onset; static loading or holding pain; overuse pathology (low force repetitive strain or high force and/or impact repetitive strain); recurrent pain patterns; and chronic pain.

- It is important to identify UCM in the functional movement system. It is our hypothesis that the uncontrolled segment is the most likely source of pathology and symptoms of mechanical origin. There is a growing body of evidence to support the relationship between UCM and symptoms (Dankaerts 2006a, 2006b; Luomajoki et al 2008; van Dillen et al 2009). The direction of UCM

(Sahrman 2002; O’Sullivan et al 2005) and control impairments (O’Sullivan et al 2005; Dankaerts et al 2009). All of these terms describe aspects of movement dysfunction, many of which are linked to UCM.

The focus of this text is to describe UCM and explore the relationship of UCM to dysfunction in the movement system (Comerford & Mottram 2011). Movement dysfunction represents multifaceted problems in the movement system and the therapist needs the tools to relate UCM and faults in the movement system to symptoms, recurrence of symptoms and disability. Skills are required to analyse movement, make a clinical diagnosis of movement faults and apply a patient-specific retraining program and management plan to deal with pain, disability, recurrence of pain and dysfunction.

Sahrman (2002) has promoted the concept that faulty movement can induce pathology, not just be the result of it; that musculoskeletal pain syndromes are seldom caused by isolated events; and that habitual movements and sustained postures play a major role in the development of movement dysfunction. These statements have been fundamental in the development of the movement dysfunction model. Clinical situations which have a major component of movement dysfunction contributing to pain include: postural pain; pain of insidious onset; static loading or holding pain; overuse pathology (low force repetitive strain or high force and/or impact repetitive strain); recurrent pain patterns; and chronic pain.

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relates to the direction of tissue stress or strain and pain producing movements. Therefore it is important in the assessment to identify the site and the direction of UCM and relate it to the symptoms and pathology. The UCM identifies the site and the direction of dynamic stability dysfunction and is related to the direction of symptom-producing movement. For example, UCM into lumbar flexion under a flexion load may place abnormal stress or strain on various tissues and result in lumbar flexion-related symptoms. Likewise, uncontrolled lumbar extension under extension load produces extension-related symptoms, while uncontrolled lumbar rotation or side-bend and/or side-shift under unilateral load produces unilateral symptoms.

**IDENTIFICATION AND CLASSIFICATION OF UCM**

Figure 1.2 illustrates the link between UCM and pain. Abnormal stress or strain that exceeds tissue tolerance can contribute to pain and pathology. The relationship between UCM and pain/pathology will be explored further in Chapter 3.

In this text the identification and classification of movement faults are described in terms of site and direction of UCM. These movement faults will be discussed in Chapter 2 in relation to changes in motor recruitment and strength (Comerford & Mottram 2001b, 2011). Scientific literature and current clinical practice are linking the site and direction of UCM in relation to symptoms, disability, dysfunction, recurrence, risk and performance (Figure 1.3).

**Figure 1.2** Uncontrolled movement: the link to pain and pathology

**Figure 1.3** Factors relating to the site and direction of uncontrolled movement

### Symptoms

Symptoms are what the patient feels and complains of and include pain, paraesthesia, numbness, heaviness, weakness, stiffness, instability, giving way, locking, tension, hot, cold, clammy, nausea and noise. The treatment of symptoms is often the patient’s highest priority and is a primary short-term goal of treatment.

Pain is frequently one of the main symptoms that the patient presents with to the therapist and is inherently linked to movement dysfunction. Contemporary research clearly demonstrates that individuals with pain present with aberrant movement patterns (Dankaerts et al 2006a, 2009; Falla et al 2004; Ludewig & Cook 2000; Luomajoki et al 2008; O’Sullivan et al 1997b, 1998). Research has demonstrated a consistent finding: in the presence of pain, a change occurs in recruitment patterns and the coordination of synergistic muscles. Individuals with pain demonstrate patterns of movements that would normally be used only in the performance of high load or fatiguing tasks (e.g. pushing, pulling, lifting weights) to perform low load non-fatiguing functional tasks (e.g. postural control and non-fatiguing normal movements). Clearly UCM is a feature of many musculoskeletal pain presentations and identifying and classifying these movement faults is essential if therapists are to effectively manage symptoms by controlling movement faults.

### Disability

Disability is the experienced difficulty doing activities in any domain of life (typical for one’s age and sex group, e.g. job, household management, personal care, hobbies, active recreation) due to a health or physical problem (Verbrugge &
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Jette 1994). Movement faults are related to disability. For example, Lin et al (2006) demonstrated that changes in scapular movement patterns (in particular a loss of posterior tilt and upward rotation) correlated significantly with self-report and performance-based functional measures indicating disability. The relationship between disability and movement faults has been identified in many other fields of physical therapy (e.g. neurological and amputee rehabilitation). Indeed, in relation to gait dysfunction, management and retraining of UCM is a key factor in rehabilitation of people with lower limb amputations using a prosthesis (Hirons et al 2007).

Reduction of disability is the primary long-term goal of therapy or rehabilitation. Disability is individual and what one person considers disability another person might consider exceptional function. For example, an elite athlete’s disability may be a function that most people do not have the ability to do, do not want to do or need to do. Movement dysfunction, however, can affect a person’s ability to function independently and therefore decrease quality of life. The disablement process model in disease as well as in rehabilitation is gaining recognition (Escalante & del Rincon 2002; Verbrugge & Jette 1994) and retraining movement faults has been shown to improve function (O’Sullivan et al 1997a; Stuge et al 2004).

**Dysfunction**

Dysfunction can imply disturbance, impairment or abnormality in the movement system. It can be objectively measured and quantified and/or compared against a normal or ideal standard or some validated or calculated benchmark. These impairments may present as weakness, stiffness, wasting, sensory–motor changes (including proprioception changes, altered coordination and aberrant patterns or sequencing of muscle recruitment) or combinations of several impairments. Dysfunction measurements include: joint range of motion (physiological or accessory); muscle strength (isometric, concentric, eccentric, isokinetic, power and endurance); muscle length; flexibility; stiffness; speed; motor control (recruitment, inhibition, coordination and skill performance); bulk (girth, volume, cross-sectional area); and alignment.

A baseline measurement of dysfunction, followed by an intervention with some form of treatment or therapy over a variable timeframe and subsequent reassessment of dysfunction provides the basis of evidence-based practice. Reduction of dysfunction is a primary short-term goal of therapeutic intervention, although the patient is frequently symptom free before dysfunction is corrected. Treatment should not cease just because the symptoms have disappeared, but may need to continue until no more dysfunctions are measurable.

The process of identifying and measuring UCM, and linking UCM to musculoskeletal pain, and to changes in muscle function, is a developing area of active research in the field of pain and movement dysfunction (Gombatto et al 2007; Luomajoki et al 2007, 2008; Mottram et al 2009; Morrissey et al 2008; Scholtes et al 2009; Roussel et al 2009a; van Dillen et al 2009). Muscle dysfunction is most clearly apparent in people with pain (Falla & Farina 2008; Hodges & Richardson 1996; Hungerford et al 2003; Lin et al 2005). The changes in muscle function underlying pain can present in two ways: 1) as altered control strategies (van Dillen et al 2009; O’Sullivan 2000); and 2) as physiological peripheral muscle changes (Falla & Farina 2008). Physiological changes associated with muscle dysfunction are discussed further in Chapter 2, and altered control strategies are discussed further in Chapter 3.

**Recurrence**

The correction or rehabilitation of dysfunction has been shown to decrease the incidence of pain recurrence (Hides et al 1996; Jull et al 2002; O’Sullivan et al 1997a). This reinforces the need for therapy to be aimed at correcting dysfunction in the management of musculoskeletal disorders and not just relieving symptoms.

**Risk of injury**

Evidence suggests history of injury is a predictive factor for re-injury and therefore outcome measures that are defined in terms of normal range of joint motion and muscle strength are inadequate to prevent recurrence (Mottram & Comerford 2008). Making the link between UCM and pain is not new, but the concept of linking it to injury prevention is.

Some recent research has highlighted the potential for linking UCM to risk of injury. A recent study on dancers identified two movement
control tests that may be useful for the identification of dancers at risk of developing musculoskeletal injuries in the lower extremities (Roussel et al 2009a). Athletes with decreased neuromusculoskeletal control of the body’s core (core stability) are at an increased risk of knee injury (Zazulak et al 2007). Indeed, there is now growing evidence that motor control and physical fitness training prevent musculoskeletal injuries (Roussel et al 2009b), highlighting the importance for therapists to be more knowledgeable about movement control and function.

**Performance**

At present there is little published literature to relate UCM to performance. However, anecdotal empirical evidence has shown that retraining movement faults can improve performance in athletes.

The movement dysfunctions associated with pain and disability have been shown to be reversible so there is a developing need to identify UCM in relation to injury risk and performance and to objectively evaluate the outcome of retraining.

**A MODEL FOR THE ASSESSMENT AND RETRAINING OF MOVEMENT FAULTS**

Many clinicians and researchers have made a significant contribution to the body of evidence relating to movement, movement impairments and corrective retraining. Some have described a particular approach to assessment and retraining and most support each other’s philosophies or provide different pieces of the puzzle to enable an understanding of the ‘whole picture’. No single approach has all the answers but the therapist who wants to provide ‘best practice’ for clients can benefit enormously from a synthesis of the different approaches and concepts proposed to date, along with the ongoing development and integration of original ideas and applied principles.

Figure 1.4 illustrates the development of the movement analysis model. The movement analysis model identifies UCM in terms of the site (joint), direction (plane of motion) and recruitment threshold (low or high) and further establishes links to pain, disability, dysfunction, recurrence, risk of injury and performance. This model has been developed through the analysis and synthesis of historical and contemporary research from many sources; however, it is not intended to be a comprehensive summary of the current level of knowledge surrounding movement analysis.

Kendall and colleagues (2005) described muscle function in detail. Their now classic text has been the foundation for assessment of muscle function, especially with reference to the graded testing of muscle strength and analysing the interrelationship of strength and function. Janda (1986) had previously developed the concept of muscle imbalance and patterns of dysfunction by analysing the pattern of movement sequencing. His primary intervention was to increase extensibility of short muscles. Sahrmann (2002) and co-workers further developed the concept of muscle imbalance, again analysing patterns of movement, and have developed a diagnostic framework for movement impairments (direction susceptible to motion).

The 1990s saw a huge advancement in the identification of motor control dysfunction (Jull et al 2008; Richardson et al 2004). Hodges (Hodges & Cholewicki 2007) has developed a large body of evidence linking motor control of deep muscles to spinal stability. O’Sullivan and co-workers have provided objective measurements to support the links between altered muscle recruitment and direction-related musculoskeletal pain (Dankaerts et al 2006a). From this research a classification system based on diagnostic subgroups has been proposed (Vibe Fersum et al 2009).

Vleeming et al (2007) and Lee (2004) have developed the model of form and force closure and have linked this to anatomical fascial slings. McGill’s (2002) research has emphasised the importance of training more superficial muscles to stabilise the core during loaded and sporting function and is often referred to as core strengthening. All these clinicians and researchers have contributed important aspects to a comprehensive and integrated model of movement analysis.

**Alternative therapies**

In the search to identify the defining characteristics of therapeutic exercise, a brief review and analysis of many different approaches and concepts including alternative therapies is appropriate. Some of these approaches are supported by
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clinical evidence (Emery et al 2010; Rydeard et al 2006). Box 1.1 lists some useful approaches to pain management and/or movement dysfunction to explore. Many exercise approaches have either stood the test of time or their popularity suggests that people who practise them feel or function better.

Whilst the various exercise concepts feature distinctive elements that characterise their approach, there are features that are common to all approaches (Box 1.2). These common features may contribute to good function and warrant closer inspection and further investigation. Breathing control is a key feature in many of these therapies. The link between respiratory disorders

Box 1.1 Useful alternative therapies in the management of movement dysfunction

- Tai chi
- The Alexander technique
- Yoga
- Pilates
- Physio ball (Swiss ball)
- Feldenkrais
- Martial arts
- GYROTONIC®
Box 1.2 Common features in alternative therapies

- Multi-joint movements
- Slow movements
- Low force movements
- Large range movements
- Coordination and control of rotation
- Smooth transition of concentric–eccentric movement
- Awareness of gravity
- Concept of a ‘core’
- Coordinated breathing
- Awareness of posture
- Intermittent static hold of position
- Control of the centre of mass of one body segment with respect to adjacent segments
- Proximal control for distal movement
- Positive mental attitude

and increased risk of development of back pain has recently been established (Smith et al 2009) and altered breathing patterns have been noted during lumbopelvic motor control tests (Roussel et al 2009c).

THE ASSESSMENT AND MANAGEMENT OF UCM

Effective intervention requires the therapist to have a thorough understanding of the mechanisms of aberrant movement patterns, an ability to confidently diagnose and classify the movement faults and to manage these dysfunctions. Guidelines for a comprehensive analysis of movement dysfunction have been described with factors the therapist needs to consider in Box 1.3

Box 1.3 Procedure for analysis of movement dysfunction

Uncontrolled movement: assessment and retraining guidelines

1. Assess, diagnose and classify movement in terms of pain and dysfunction from a motor control and a biomechanical perspective.
2. Develop a large range of movement retraining strategies to establish optimal functional control.
3. Use a clinical reasoning framework to prioritise the clinical decision-making challenges experienced in contemporary clinical practice.
4. Develop an assessment framework that addresses the four key criteria relevant to dysfunctional movement:
   a. diagnosis of movement dysfunction
      i. site and direction of uncontrolled movement
      ii. uncontrolled translation
      iii. uncontrolled range of motion
      iv. myofascial and articular restriction
      v. aberrant guarding responses
   b. diagnosis of pain-sensitive tissue(s)
      i. patho-anatomical structure
   c. diagnosis of pain mechanisms
      i. peripheral nociceptive (inflammatory or mechanical)
      ii. neurogenic sensitisation
   d. identification of relevant contextual factors
      (Verbrugge & Jette 1994)
      i. environmental factors (extra-individual) (e.g. physical and social context)
      ii. personal factors (intra-individual) (e.g. lifestyle and behavioural changes, psychosocial attributes, coping skills).
5. Make links between uncontrolled movement and pain and other symptoms, dysfunction, recurrence, risk of injury and performance.
6. Make a link between uncontrolled movement and disability through the disablement process model.
7. Make links between uncontrolled movement and changes in motor control, strength, joint range of motion, myofascial extensibility and functional activities.
8. Identify the clinical priorities in terms of retraining uncontrolled movement and mobilising restrictions of normal motion.
9. Use a clinical assessment tool to identify deficiencies and reassess improvements in motor control efficiency.
10. Integrate non-functional motor control retraining skills with functionally relevant movement.
11. Use other techniques and strategies (e.g. taping to support uncontrolled movement or facilitate motor relearning and strengthening).
12. Use a clinical reasoning framework to identify priorities for rehabilitation, where to start retraining and how to be specific and effective in exercise prescription to develop individual retaining programs.
13. Know which way and how fast to progress, and know how to tell when retraining has achieved an effective end-point independently of symptoms.
7 Consideration of tissues or structures contributing to symptoms

The site and direction of UCM may match the pathology identified. For example, people with a shoulder impingement demonstrate UCM at the scapula (Morrissey 2005). Abnormal quality of motion in spinal lumbar segments has been demonstrated to be associated with spondylolisthesis pathology (Schneider et al 2005). The link between tissue stress resulting in pathology and abnormal range or quality of movement is becoming more evident. The therapist needs to find a link between the UCM and any presenting pathology.

8 Assess for environmental and personal factors

Personal factors (e.g. lifestyle and behavioural changes, psychosocial attributes, coping and activity accommodations) and environmental factors (e.g. medical care and rehabilitation, medications and other therapeutic regimens, external supports, physical and social environment) should also be assessed. Personal factors commonly assessed within the context of physical therapy include items such as depression, anxiety, coping skills and cognition. These can be objectively assessed (and reassessed) with valid and reliable questionnaires such as the Pain Coping Inventory (PCI; Kraaimaat & Evers 2003), Tampa Scale of Kinesiophobia (TSK; Swinkels-Meeuwisse et al 2003; Vlaeyen et al 1995), Fear Avoidance Beliefs Questionnaire (FABQ; Waddell 1998) and the Pain Self-Efficacy Questionnaire (PSEQ; Nicholas 2007; Nicholas et al 2008).

Once the site and direction of UCM have been established, effective rehabilitation should ensure that movement dysfunction is addressed throughout functional tasks. Control of movement during functional activities, and awareness of the UCM during posture, daily activities, sport and training programs should be promoted. For example, a person with uncontrolled scapula downward rotation needs to be aware of this movement fault during daily activities such as reaching for a cup in a cupboard. A person with uncontrolled lumbar flexion needs to be aware of this movement fault when bending forwards to tie up their shoelaces.

9 Integrate other approaches or modalities

There are many other therapeutic modalities that can influence the correction of movement faults. Table 1.5 details some examples. This is not intended to be an exhaustive list but illustrates
Uncontrolled movement

10 Consider prognosis

Although the management of symptoms has been the primary aim in the treatment of musculoskeletal disorders, research has also demonstrated links between UCM and dysfunction, disability and the recurrence of symptoms. It is therefore appropriate that dysfunction and disability are also considered, along with symptoms, when providing a prognosis for recovery in the management of musculoskeletal disorders. The timeframe for expected improvement in symptoms should be considered independently of the timeframes for recovery of dysfunction and disability when making prognostic judgments for recovery.

Physiological tissue repair timelines have been well researched and are reasonably well defined. In more acute (less than 6 weeks) conditions, these provide a useful guideline. In more chronic (more than 12 weeks) conditions, other prognostic factors become more important. A systematic review on prognostic factors in whiplash-associated disorders established that factors related to poor recovery included: female gender; a low level of education; high initial neck pain; more severe disability; higher levels of somatisation and sleep difficulties (Hendriks et al 2005; Scholten-Peeters et al 2003). Neck pain intensity and work disability proved to be the most consistent predictors for poor recovery in these studies.

The relative influence of factors beyond physiological processes is a contemporary research subject and there is a growing body of evidence indicating that socio-demographic, physical and psychological factors strongly affect short- and long-term outcomes. These factors must be taken into consideration when establishing a realistic timeframe for when dysfunction, symptoms and disability could be expected to improve and by how much.

### CLINICAL REASONING IN A DIAGNOSTIC FRAMEWORK

As noted in Box 1.3, when a patient presents with neuromusculoskeletal pain and dysfunction, it is good clinical practice to assess and identify four key criteria:

1. diagnosis of movement dysfunction
2. diagnosis of pain-sensitive or pain-generating structures
3. diagnosis of presenting pain mechanisms – peripheral nociceptive and neurogenic sensitisation
4. evaluation and consideration of contextual factors.

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<thead>
<tr>
<th>OTHER THERAPEUTIC APPROACHES</th>
<th>EXAMPLES</th>
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<tbody>
<tr>
<td>Pathophysiological approaches</td>
<td>Ice, heat, electrotherapy, medication</td>
</tr>
<tr>
<td>Articular approaches</td>
<td>Joint mobilisation and manipulation (Maitland et al 2005; Cyriax 1980; Kaltenborn et al 2003)</td>
</tr>
<tr>
<td>Ergonomic and environmental factors</td>
<td>Work place assessment, postural advice</td>
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<tr>
<td>Neurodynamic approaches</td>
<td>Neurodynamic mobilisation (Butler 2000; Shacklock 2005)</td>
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<td>Sensory-motor approaches</td>
<td>Neuromuscular facilitation (Rood in Goff 1972), Bobath ‘normal movement’ (Bobath 1990), neurofunctional training (Carr &amp; Shepherd 1998), neurosensory approach (Homstøl 2009)</td>
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<td>Soft tissue approaches</td>
<td>Massage therapy (Chaitow 2003)</td>
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<td>Psychosocial approaches</td>
<td>Behavioural evaluation and therapy (Waddell 1998; Woby et al 2008)</td>
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<tr>
<td>Biomechanical approaches</td>
<td>Taping, orthotics, bracing</td>
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useful adjuvant modalities in retraining UCM, managing pain, mobilising restrictions or treating pathology.
1 Diagnosis of movement dysfunction (site and direction of uncontrolled motion)

The initial priority is to identify the site and direction of UCM that best correlates with the patient’s presenting mechanical symptoms. In complex presentations, there is frequently more than one site of UCM. When this is the case, it is useful to identify whether one site is the site of primary dysfunction and whether the other site is compensating for the primary one.

If there are obvious restrictions that are causing compensatory UCM, it is very effective for the therapist to work to achieve normal mobility of these restrictions early in the management plan (see Chapter 4).

The therapist should also identify if there is a priority to retrain local stability muscle function early or if this can be retrained later in the rehabilitation process. Similarly, the therapist should identify any contributing muscle imbalance issues related to the dysfunction, such as altered length and recruitment relationships between mono-articular stabiliser muscles and multi-articular mobiliser muscles. If these imbalances are identified, the global stabiliser muscle recruitment efficiency should be retrained to recover active control through the full available range of motion, and the global mobility muscle extensibility should be restored.

2 Clinical diagnosis of pain-sensitive or pain-generating structure(s)

The therapist should identify the structure or tissue that is the source of the symptoms or pain that the patient complains of. Patients who present with a chronic or recurrent condition frequently report more than one tissue contributing to the pain experience. The clinical reasoning process that identifies a variety of pain-sensitive tissues requires a thorough understanding of tissue anatomy and physiology, a knowledge of the mechanism of injury (if there is one) and an understanding of the typical responses of different tissues to stress and strain and injury. All available therapeutic skills, tools or modalities can be utilised to best provide an optimal environment to allow and promote tissue healing and to control or manage the presenting signs and symptoms. Contemporary clinical reasoning in patients with chronic pain suggests it is more appropriate to explore factors affecting impairment of function and participation than to attempt to diagnose specific structures or tissues as a source of nociception.

3 Clinical diagnosis of presenting pain mechanisms

It is essential to have an understanding of the relevant pain mechanisms contributing to any individual’s pain presentation. In a person with chronic or recurrent pain it is common to find different mechanisms contributing to their symptoms. Melzack’s (1999) neuromatrix theory of pain proposes that pain is a multidimensional experience produced by characteristic ‘neurosignature’ patterns of nerve impulses generated by a widely distributed neural network in the brain. It proposes that the output patterns of the neuromatrix activate perceptual, homeostatic, and behavioural responses after injury, pathology or chronic stress. The resultant pain experience is produced by the output of a widely distributed neural network in the brain rather than solely by sensory input evoked by injury, inflammation or other pathology (Moseley 2003). Therefore, pain is a multi-system output that is produced when a cortical pain neuromatrix is activated.

Ideally, an attempt should be made to determine the relevant proportions of these mechanisms; that is, the degree to which peripheral nociceptive (mechanical/inflammatory) elements contribute to the pain experience and the degree to which neurogenic sensitisation is present. Behavioural, social and psychosomatic influences further contribute to the multidimensional nature of chronic and recurrent pain. The dominant mechanisms need to be addressed as a priority. A multidisciplinary and multidimensional approach can be more effective in managing symptoms, both in the short and long term.

4 Evaluation and consideration of contextual factors

The therapist should assess for the influence of contextual factors – both personal and environmental – on the patient’s signs and symptoms and explore how these might relate to UCM (Figure 1.8).
Researchers and clinicians have become increasingly aware that there is frequently little correlation between pathology and (functional) limitations in activities and participation. This is even more evident for chronic complaints. Contemporary clinical reasoning has seen a paradigm shift from a biomedical to a bio-psychosocial model. For instance, in the analysis of movement dysfunction model presented in Box 1.3, a modified version of a disablement process model (Verbrugge & Jette 1994) is included. Such a disablement assessment model uses the same theoretical construct as a starting point for assessment and treatment (Figure 1.9).

In a disablement process model, the therapist, together with the patient, determines which functions and ADL are limited. These are defined as ‘disabilities’ and can be evaluated by valid and reliable questionnaires and performance tests. This provides the opportunity to reassess the patient in an objective way and evaluate efficacy of interventions. Within the clinical reasoning process the therapist evaluates the four factors in the diagnostic framework criteria (see Figure 1.8), and relates these to the functional limitations. In this partially reversible system, the functional limitations are continuously influenced by extra- and intra-individual factors. These existing and potential risk factors are the reason why pathology presents as or evolves into impairments. Using a clinical decision-making process, the therapist is able to assess and determine if a normal or aberrant course is present.

Different terminology is used in the International Classification of Functioning, Disability, and Health (ICF 2001) model of functioning and disability (Figure 1.10). However, essentially the intra-individual factors in the disablement process model are comparable with the ICF’s personal factors and the extra-individual factors are comparable with the environmental factors.

The rehabilitation problem solving (RPS) form (Figure 1.11) was developed to address patients’ perspectives and to enhance their participation in the decision-making process during their assessment. The RPS form is based on the ICF model of functioning and disability and
In the ICF model the horizontal dimension of a health status or profile is illustrated as being influenced by elements in the vertical dimension. The ICF model could be considered a method of classification, describing a health condition at a particular moment, such as a picture or ‘freeze frame’. In contrast, the disablement process represents constructs within the ICF model under the constant influence of risk factors and hence could be described as more like a ‘film’.

To summarise, ‘health’ can be described in terms of:
- health condition (using ICF terminology)
- course (normal or aberrant)
- prognostic profile
- patient’s perspective.

Clinical decision-making should start from the patient’s perspective and interventions should be primarily aimed at those aspects of impairment that have a direct bearing on disability and/or functional limitations. In the subjective examination, the patients will define their perspective in terms of disability and functional limitations; for example, the inability (due to low back pain) to bend over from standing to tie shoelaces or the inability (due to shoulder pain) to reach into a cupboard above the shoulders. These self-reported symptoms are explored further within the physical examination to inform the clinical decision-making process. For example, if patients with low back pain are unable to actively control movements of the low back, especially flexion control while performing a waiters’ bow (Luomajoki 2008), then clinicians should direct their intervention towards correcting the neuromuscular impairment underpinning this. Similarly, if patients with shoulder pain are unable to actively control the (re-)positioning of the scapula during functional movements (Tate et al 2008; von Eisenhart-Rothe 2005), then clinicians should aim their intervention strategy at regaining this control. Therapists should not solely focus on addressing an isolated pathology, but use frameworks such as the disablement assessment model to facilitate effective intervention. The link between specific movement retraining and improvement in functional tasks is now well supported by evidence (Jull et al 2009; Roussel et al 2009b).

Practitioners need to have the skills to identify and retrain movement faults. These skills should be integrated into current practice and the patient facilitates the analysis of patient problems, focusing on specific targets, and relating salient disabilities to relevant and modifiable variables.

This form can include the diagnostic criteria within the clinical reasoning framework described in Figure 1.8 in order to assess the key criteria that relate to the functional limitations. This process identifies links between factors in the diagnostic framework and subsequent functional limitations so that the mechanism behind the dysfunction can be addressed to optimise efficacy of intervention. The form in Figure 1.11 can include the diagnostic framework as described in Figure 1.8.

The essence of both the ICF model of functioning and disability and the RPS form is that an individual’s (dys-)functioning or disability represents an interaction between the health condition (e.g. diseases, disorders, injuries, traumas and all factors in the diagnostic framework) and the contextual factors (i.e. ‘environmental factors’ and ‘personal factors’). The interactions of the components in the model are two-way, and interventions in one component can potentially modify one or more other components.

In the ICF model the horizontal dimension of a health status or profile is illustrated as being influenced by elements in the vertical dimension. The ICF model could be considered a method of classification, describing a health condition at a particular moment, such as a picture or ‘freeze frame’. In contrast, the disablement process represents constructs within the ICF model under the constant influence of risk factors and hence could be described as more like a ‘film’.

To summarise, ‘health’ can be described in terms of:
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- prognostic profile
- patient’s perspective.

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Practitioners need to have the skills to identify and retrain movement faults. These skills should be integrated into current practice and the patient
managed in a holistic way with consideration of all aspects of the human motion system, and the influence of both intra- and extra-individual factors. An understanding of how UCM can influence pain is essential in the management of neuromusculoskeletal disorders. To assist in this reasoning process the anatomical and physiological principles are reviewed in Chapter 2 and how pain, dysfunction and pathology can effect UCM is explored in Chapter 3.

**REFERENCES**


Coppieters, M.W., 2006. Shoulder restraints as a potential cause for stretch neuropathies: biomechanical support for the impact of shoulder girdle depression and arm abduction on nerve strain. Anesthesiology 104 (6), 1351–1352.


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INTRODUCTION

The complexity of shoulder girdle dysfunction makes diagnosis difficult, with definitions for common diagnoses, such as impingement and frozen shoulder, being unclear, inconsistent and unreliable (Schellingerhout et al 2008). An epidemiological study examining 1960 people successfully identified current and past shoulder problems, but was unable to discriminate between discrete shoulder pathologies (Walker-Bone et al 2004). Therapy for shoulder girdle pain and disability is predominantly concerned with restoration of optimal movement and function rather than applying diagnostic labels, with traditional approaches to clinical diagnosis at the shoulder girdle frequently neglecting to assess dynamic movement faults, a significant factor associated with shoulder dysfunction (Lukasiewicz et al 1999; Ludewig & Cook 2000; Lin et al 2006; Tate et al 2008). Classification of movement control faults at the shoulder girdle is gaining recognition, with Kibler & McMullen (2003) describing a clinical classification of scapular dyskinesis (scapular movement control faults). A classification of dysfunction in terms of site and direction of uncontrolled movement (UCM) has been proposed (Mottram 2003; Mottram et al 2009a; Comerford & Mottram 2011), and diagnosis based on movement impairment (Sahrmann 2002; Caldwell et al 2007) encouraged. The consensus statement at a recent scapular summit (Kibler et al 2009) agreed that the observation of scapular dyskinesis and clinical tests that alter symptoms (UCM in this text) should form the basis for the scapular evaluation.

This chapter sets out to explore the assessment and retraining of UCM in the shoulder. Before details of the assessment and retraining of UCM in the shoulder region are explained, a brief review of function, changes in muscle function and movement and postural control in the region is presented.

Scapula function and glenohumeral joint stability

The ability to control the orientation and movement of the scapula is essential for optimal arm function. The bony, capsular and ligamentous restraints are minimal at the scapulothoracic ‘joint’ so stability is dependent on active muscular control. Movement faults and changes in muscle function of the scapula are associated with shoulder symptoms (Lukasiewicz et al 1999; Ludewig & Cook 2000; Lin et al 2006; Roy et al 2008; Tate et al 2008).

The glenohumeral joint has the greatest range of motion of any human joint. This mobility is
necessary for upper limb functions, which range from weight bearing to high-speed acceleration and deceleration at the extremes of its range. Stability is sacrificed to a significant degree to achieve this mobility function. The scapula provides the base for attachment of muscles that move the glenohumeral joint. The scapula should be orientated to optimise the length–tension relationship of these muscles (van der Helm 1994) and provides the proximal articular surface of the glenohumeral joint (glenoid) and orients the glenoid, to increase the range available to the upper limb. The scapula facilitates optimal contact with the humeral head – increasing joint congruency and stability (Saha 1971). Abnormal scapular kinematics have been identified in people with multidirectional instability (Ogston & Ludewig 2007). Full upward rotation of the glenoid enhances mechanical stability of the joint by bringing the glenoid fossa directly under the head of the humerus (Lucas 1973) and prevents impingement under the subacromial and coracoacromial arch. Glenohumeral function is influenced to a large extent by the position and orientation of the glenoid and hence scapula stability; however, the glenohumeral joint exhibits a number of mechanisms to retain joint congruency during functional movement which include passive stability mechanisms and active stability mechanisms. Passive stability mechanisms include the capsular and ligamentous restraints, labrum and mechanisms such as the creation on negative intra-articular pressure to resist translation.

Changes in shoulder muscle function

Muscle stiffness is required at the scapula-thoracic and glenohumeral to enhance stability. It has been shown that moderate levels of muscle contraction can significantly increase glenohumeral joint stiffness and stability (Huxel et al 2008). A non-specific pre-setting action of the rotator cuff and biceps is seen prior to rotation of the shoulder joint, and this recruitment is aimed mainly at enhancing the joint ‘stiffness’ and hence its stability (David et al 2000). A similar action is seen in upper trapezius (Wadsworth & Bullock-Saxton 1997), suggesting it has a pre-setting role at the scapula. Evidence suggests that muscle function around the shoulder girdle can be impaired by pain and pathology. Altered timing (latency) of electromyographic (EMG) activity has been identified in muscles of the scapula (Wadsworth & Bullock-Saxton 1997; Cools et al 2003; Lin et al 2005; Falla et al 2007; Moraes et al 2008) and the glenohumeral joint (Hess et al 2005). Interestingly, muscle function (or dysfunction) has been associated with movement faults; for example, decreased serratus anterior activity has been associated with an increase in forward tilt of the scapula (Ludewig & Cook 2000; Lin et al 2005). This literature supports the need for specific assessment of movement faults so individual rehabilitation strategies can be implemented. Further research is needed to explore the relationship between movement abnormalities and symptoms and muscle function.

Identifying UCM at the shoulder girdle

Motion analysis studies have identified abnormal movements of the scapula which include scapula internal rotation (Ludewig & Cook 2000; Nawoczenski et al 2003; Tsai et al 2003; Borstad & Ludewig 2005; Borstad 2006); scapular downward rotation (Ludewig & Cook 2000; Tsai et al 2003; Lin et al 2006); scapula anterior tilt (Lukasiewicz et al 1999; Ludewig & Cook 2000; Nawoczenski et al 2003; Borstad & Ludewig 2005; Lin et al 2005; Morrissey 2005); and elevation (Lukasiewicz et al 1999; Tsai et al 2003; Lin et al 2005). UCM of the glenohumeral joint has been identified and includes translation (Baeyens et al 2001; Ruediger et al 2002; von Eisenhart-Rothe et al 2002, Ludewig and Cook 2002) and external rotation (Baeyens et al 2001).

In the current literature it is clear that alterations in dynamic control of the glenohumeral and scapula-thoracic joints are important factors in shoulder pathology (Ludewig and Cook 2000; Morrissey 2005; Alexander 2007; Ogston & Ludewig 2007). Although these studies demonstrated clear differences in movement patterns for symptomatic shoulders, they do not describe test manoeuvres that could be used specifically to detect the abnormalities in the clinical environment, therefore neglecting a significant component of assessment. This chapter details the assessment of UCM at the shoulder region and describes retraining strategies.
The shoulder girdle
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The site and direction of UCM at the shoulder girdle can be observed at the scapula in terms of downward rotation, forward tilt, winging (internal rotation), elevation, retraction and protraction (abduction) and the glenohumeral joint in terms of anterior, inferior, posterior translation and medial rotation (Table 8.1).

<table>
<thead>
<tr>
<th>SITE</th>
<th>SCAPULA</th>
<th>GLENOHUMERAL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Downward rotation</td>
<td>Anterior translation</td>
</tr>
<tr>
<td></td>
<td>Forward tilt</td>
<td>Inferior translation</td>
</tr>
<tr>
<td></td>
<td>Winging</td>
<td>Posterior translation</td>
</tr>
<tr>
<td></td>
<td>Elevation</td>
<td>Medial rotation</td>
</tr>
<tr>
<td></td>
<td>Retraction</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Protraction</td>
<td></td>
</tr>
</tbody>
</table>

**Diagnosis of the site and direction of UCM at the shoulder girdle**

The diagnosis of site and direction of UCM at the shoulder girdle can be observed at the scapula in terms of downward rotation, forward tilt, winging (internal rotation), elevation, retraction and protraction (abduction) and the glenohumeral joint in terms of anterior, inferior, posterior translation and medial rotation (Table 8.1).

**Linking the site of UCM to symptom presentation**

A diagnosis of UCM requires evaluation of its clinical priority. This is based on the relationship between the UCM and the presenting symptoms. The therapist should look for a link between the direction of UCM and the direction of symptom provocation: a) Does the site of UCM relate to the site or joint that the patient complains of as the source of symptoms? b) Does the direction of movement or load testing relate to the direction or position of provocation of symptoms? This identifies the clinical priorities.

The site and direction of UCM at the scapula and the glenohumeral joint can be linked to different clinical presentations and postures and activities that provoke or produce symptoms (Table 8.2).

In a shoulder with signs and symptoms of impingement and instability, control of movement needs to be effective to manage symptoms and dysfunction. These mechanisms are highlighted in Table 8.3 and the assessment of these mechanisms is an important aspect of a comprehensive shoulder girdle assessment.

The site and direction of uncontrolled movement at the shoulder girdle can be linked to different clinical presentations of shoulder impingement syndrome and glenohumeral instability. Table 8.4 illustrates the clinical guidelines for impingement and instability.

**Identifying site and direction of UCM at the scapulothoracic and glenohumeral joints**

The key principles for assessment and classification of UCM are described in Chapter 3. All dissociation tests are performed with the scapula and glenohumeral in the neutral training region. This can be passively positioned by the therapist prior to each test. The subject needs to clearly understand the test and appropriate facilitation strategies employed, including cognitive awareness, tactile, visual and technological feedback, and proprioceptive input.

**Scapula and glenohumeral joint neutral training region**

The natural resting position of the shoulder girdle is often displaced from an optimal training position. If there is poor antigravity postural control the scapula often rests in a downwardly rotated or forward tilted position. If there is a restriction (e.g. a stiffer pectoralis minor), the scapula will rest in relatively more forward tilt than is ideal. The therapist should passively position the shoulder girdle into its ‘neutral training region’ and then palpate reference landmarks to ensure optimal neutral alignment for testing and retraining movement control.

As a useful guide to repositioning the scapula in the neutral training region, the therapist stands to the side of the patient’s shoulder and places the pisiform and ulnar border of one hand on the medial side of the patient’s inferior scapular angle. The therapist then places the ulnar border of the other hand on the patient’s coracoid with the hollow of the palm over the humeral head (Figure 8.1). With the fingers of both hands pointing to the ceiling, the therapist lifts both their elbows so that both forearms are in line. The therapist then gently ‘squeezes’ both hands...
### Table 8.2 The link between the site and direction of UCM at the shoulder and different clinical presentations

<table>
<thead>
<tr>
<th>SITE AND DIRECTION OF UCM</th>
<th>CLINICAL EXAMPLES OF SYMPTOMS PRESENTATIONS</th>
<th>PROVOCATIVE MOVEMENTS, POSTURES AND ACTIVITIES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SCAPULA</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Downward rotation</td>
<td>• Symptoms of subacromial or coracoacromial impingement; that is, pain at the point of the shoulder, in the region of the coracoid and anterior and lateral deltoid region</td>
<td>Symptoms provoked by arm movements and postures into elevation above 60° (if especially sustained or loaded); for example, lifting, reaching forwards, reaching overhead, pushing or pulling with the arm above shoulder height, sustained static postures with the scapula dropped.</td>
</tr>
<tr>
<td>• Forward tilt</td>
<td>• ± Referral from myofascial, articular and neural structures</td>
<td></td>
</tr>
<tr>
<td>• Winging</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Elevation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Retraction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Protraction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Can present as:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• uncontrolled movement of the scapular into any of these directions resulting in an increased inferior–anterior orientation of the glenoid (IAG) (± hypermobile range)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>GLENOHUMERAL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medical rotation</td>
<td>• Symptoms of coracoacromial impingement; that is, pain at the point of the shoulder, in the region of the coracoid and anterior and lateral deltoid region</td>
<td>Symptoms provoked by arm movements and postures into forward elevation above 60° (if especially sustained or loaded); for example, lifting, reaching forwards, reaching overhead, pushing or pulling with the arm above shoulder height, sustained static postures with the scapula dropped.</td>
</tr>
<tr>
<td>Can present as:</td>
<td>• ± Referral from myofascial, articular and neural structures</td>
<td></td>
</tr>
<tr>
<td>• uncontrolled range of the humerus into medial rotation (± hypermobile medial rotation range)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>GLENOHUMERAL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Anterior translation</td>
<td>• Symptoms of glenohumeral instability; that is, pain in the anterior and posterior shoulder, at the point of the shoulder and deep axillary pain</td>
<td>Symptoms provoked by arm movements and postures into end range positions (especially if end-range rotation is combined); for example, lifting, reaching forwards, reaching overhead, pushing or pulling with the arm above shoulder height, sustained static postures with the scapula dropped.</td>
</tr>
<tr>
<td>• Inferior translation</td>
<td>• ± Referral from myofascial, articular and neural structures</td>
<td></td>
</tr>
<tr>
<td>• Posterior translation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Can present as:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• uncontrolled translation of the humeral head into any of the above directions (anterior is most common) (± hypermobile translation)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 8.3 Normal mechanisms to minimise impingement and instability during arm elevation

<table>
<thead>
<tr>
<th>IMPLICATION</th>
<th>INSTABILITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Upward rotation of glenoid</td>
<td>• Passive capsular and ligamentous restraints</td>
</tr>
<tr>
<td>• Glenohumeral lateral rotation timing</td>
<td>• Dynamic (active) control of translation</td>
</tr>
<tr>
<td>• Inferior humeral head glide</td>
<td>• Ideal length and recruitment of glenohumeral rotator muscles</td>
</tr>
<tr>
<td></td>
<td>• A stable scapula to provide a biomechanically sound platform for glenohumeral movement</td>
</tr>
</tbody>
</table>
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Together so that the acromion rises up, the humeral head moves backwards and the inferior scapular angle moves laterally around the chest wall (Figure 8.2). While the therapist passively supports the shoulder here, the person is asked to relax the shoulder and then is asked to use ‘minimal’ effort to actively maintain this position (Figure 8.3). With the scapula being actively maintained in this position, the therapist should palpate a series of landmarks (Box 8.1) and make any minor adjustments required.

A useful guide is to passively position the shoulder and, using visual and palpation feedback, the person feels the neutral position as a mid-position between elevation and depression, forward and backward tilt, upward and downward rotation and protraction and retraction. They are then instructed to move away from neutral and actively return to neutral using this feedback to ensure accurate repositioning.

Inferior anterior glenoid (IAG)

A common dysfunction pattern seen with loss of scapula neutral is the orientation of the glenoid in an inferior anterior direction, termed the inferior anterior glenoid (IAG) (Figure 8.4). This can be corrected by rotation of the scapula in the coronal plane – observed by the acromion moving superiorly while the inferior angle moves laterally (upward rotation of the scapula in the sagittal plane). The scapula also moves upward and backward (posterior or backward tilt) (Mottram et al 2009b) away from the IAG position.

Table 8.4 Clinical guidelines for impingement and instability

<table>
<thead>
<tr>
<th>IMPINGEMENT</th>
<th>INSTABILITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Palpable tenderness ++</td>
<td>• Full or hypermobile range</td>
</tr>
<tr>
<td>• Mid-range arc or catch of pain</td>
<td>• Pain (if any) at the limits of range (often only at stress points)</td>
</tr>
<tr>
<td>• Pain on static isolated muscle loading</td>
<td>• Symptoms of instability, subluxation, dislocation, clicking, dysfunction and disability (loss of performance)</td>
</tr>
<tr>
<td>• Associated weakness/inhibition</td>
<td>• Resisted rotation often pain-free</td>
</tr>
<tr>
<td>• Positive impingement tests</td>
<td>• Good strength (mid-range)</td>
</tr>
<tr>
<td>• Positive manual therapy stress tests to implicate pain-sensitive compression of subacromial or coracoacromial structures</td>
<td>• Positive instability tests</td>
</tr>
<tr>
<td>• Movement dysfunction indicates impingement; that is, positive kinetic medial rotation test (scapula)</td>
<td>• Positive manual therapy stress tests to implicate pain-sensitive capsular strain and ligamentous laxity</td>
</tr>
<tr>
<td></td>
<td>• Movement dysfunction indicates instability; that is, positive kinetic medial rotation test (glenohumeral)</td>
</tr>
</tbody>
</table>

Figure 8.1 Therapist hand position for positioning the scapula ‘neutral’ orientation
Box 8.1 Palpation reference guidelines for a neutral shoulder girdle

Palpation guidelines for shoulder girdle neutral

- Superior-medial corner of scapula is level with T2.
- Medial edge of the spine of the scapula is level with T3.
- Spine of the scapula projects to T4.
- Inferior scapular angle level with T7.
- Acromion should be higher than the superior-medial scapular corner, with the spine of the scapula angled upwards (i.e. no downward rotation).
- Plane of the spine of the scapula is orientated between 15 and 30° forward of the coronal plane.
- Acromions are level or horizontal.
- Coracoids are symmetrical.
- Clavicles are symmetrical and inclined slightly upwards.

- Inferior scapular angle is in contact with ribcage (i.e. no forward tilt/pseudo-winging').
- Medial border of scapula is in contact with ribcage (i.e. no winging).
- Medial border of spine of the scapula is approximately 5–6 cm lateral from the vertebral spinous processes.
- No more than 1/3 of the humeral head should protrude forward of the acromion.
- Scapula must be positioned in neutral alignment prior to assessing humeral rotation and the humerus must be positioned in neutral alignment to assess forearm position.
- Elbow olecranon faces posteriorly and the elbow cubital fossa faces anteriorly (differentiate from forearm pronation).
Segmental translatatory and global range specific UCM

When direction-specific UCM is observed at the shoulder, it can present in two ways. The UCM can present as either a segmental translatatory UCM (primarily of the humeral head) or a global range UCM (of either the scapula or the gleno-humeral joint).

Segmental translatatory UCM

This is a segmental UCM in which the humeral head appears to ‘glide forwards’ into excessive anterior, inferior or posterior translatory displacement associated with medial rotation, lateral rotation, flexion, abduction or extension motion testing. Uncontrolled humeral head anterior translation can be identified in motion testing. In medial rotation, lateral rotation and extension movements of the shoulder, palpation of the anterior prominence of the humeral head is used to identify excessive anterior translation. The ability to maintain the neutral axis and prevent excessive forward glide of the anterior prominence of the humeral head during active medial rotation, lateral rotation and extension is evaluated.

Global range-specific UCM

A global range-specific UCM demonstrates UCM (± hypermobile range) of the scapula or the gleno-humeral joint. This is observed as either excessive...
or dominant scapular or glenohumeral motion at the initiation of the movement or hypermobile range of motion to complete the movement.

A global range specific scapular UCM can be identified in motion testing. In any functional movement of the arm, observe or palpate for uncontrolled scapular:

- downward rotation
- forward tilt
- winging
- elevation
- protraction/abduction
- retraction/adduction.

A global range-specific glenohumeral UCM can be identified in motion testing. In any functional movement of the arm, observe or palpate for uncontrolled glenohumeral medial rotation.

The following section will demonstrate the specific procedures for testing for UCM in the shoulder girdle.

### SHOULDER GIRDLE TESTS FOR UCM

### Shoulders medial rotation control

#### OBSERVATION AND ANALYSIS OF SHOULDER MEDIAL ROTATION

**Description of ideal pattern**

While supine or sitting, with the shoulder in 90° of abduction (scapular plane), there is 60° of medial rotation of the humerus without significant scapula-thoracic movement or glenohumeral anterior translation.

The therapist passively stabilises the scapula and glenohumeral joint and assesses the passive range of glenohumeral rotation without compensation (Figure 8.5).

**Movement faults associated with glenohumeral medial rotation**

**Relative stiffness (restrictions)**

- *Restrictions – reduced glenohumeral medial rotation with the arm abducted.* When the scapula and humeral head are passively supported to control scapula-thoracic

movement or glenohumeral anterior translation, a significant loss of glenohumeral medial rotation is often observed. Ideal passive range of medial rotation is 60°. Loss of medial rotation range may be due to several reasons:

- **Capsular restriction.** Capsular shortening may contribute to a loss of medial rotation, though this is not the most common cause. If capsular shortening is present then there is usually a significant loss of lateral rotation first observed as less than 90° lateral rotation in abduction.

- **Myofascial restriction.** Over-activity, dominance and relative stiffness of the glenohumeral lateral rotator muscles: a common presentation-related to the loss of medial rotation is over-activity and shortening of the lateral rotator muscles (infraspinatus and teres minor).

Assessment of both the contractile and connective tissue shortening needs to be made and appropriate soft tissue work applied.

- **Co-contraction rigidity.** Occasionally, active medial rotation range at the glenohumeral joint may be limited by co-contraction rigidity. In the attempt to medially rotate, all glenohumeral muscles co-contract excessively and seem to ‘splint’ the shoulder from achieving full rotation. This is often a guarding response associated with instability or acute pathology or a protective ‘spasm’ in an acute inflammatory episode.
Relative flexibility (potential UCM)

- **UCM – compensatory strategies associated with restriction of glenohumeral joint medial rotation.** A variety of compensation strategies for restrictions can be employed to maintain functional range of motion. If glenohumeral medial rotation is restricted, compensatory movement can be made at both the scapula and the humeral head (Sahrmann 2002; Morrissey 2005).
  - Uncontrolled scapula forward tilt, downward rotation or elevation. The scapula may forward tilt, downwardly rotate or elevate to compensate for the loss of medial rotation. The accuracy of this palpation has been validated with three-dimensional ultrasound and motion analysis measures (Morrissey et al 2008). A positive test (scapula movement) has been linked with risk of impingement and symptoms (Morrissey 2005). The test is useful for diagnosis, especially for impingement, particularly when used with other impingement tests (Morrissey 2005).
  - Uncontrolled glenohumeral translation control. Excessive anterior translation of the humeral head compensates for a lack of glenohumeral medial rotation. A positive test (glenohumeral movement) has been linked with instability symptoms and risk (Morrissey 2005).

**Indications to test for shoulder medial rotation UCM**

Observe or palpate for:
1. hypermobile medial rotation range
2. discrepancies of shoulder medial rotation range in different positions of arm elevation
3. excessive initiation of scapular compensation during shoulder medial rotation
4. excessive glenohumeral translation during medial rotation
5. symptoms (pain, discomfort, strain) associated with shoulder medial rotation movements.

The person complains of rotation-related symptoms in the shoulder. During shoulder medial rotation load or movements, the scapula or glenohumeral joint has greater ‘give’ or compensation relative to the trunk or arm. The dysfunction is confirmed with motor control tests of shoulder medial rotation dissociation.
**Test of shoulder medial rotation control**

**T60 KINETIC MEDIAL ROTATION TEST (KMRT) (tests for scapula and glenohumeral UCM)**

This dissociation test assesses the ability to actively dissociate and control scapula movement and glenohumeral translation during glenohumeral medial rotation.

**Test procedure**

Start supine and with the humerus in 90° abduction (hand to the ceiling), and the humerus supported in the plane of the scapula. The therapist palpates the coracoid and humeral head during the procedure (Figure 8.6). The accuracy of this palpation has been measured (Morrissey et al. 2008). Medial rotation of the humerus should occur without compensation at the scapula or glenohumeral joint. The scapula should not move into forward tilt, downward rotation or elevation and the humeral head should not translate anteriorly (Morrissey 2005). There should be 60° of active medial rotation (Figure 8.7). An alternative test position is in standing with or without wall support of the scapula.

**Rating and diagnosis of shoulder girdle UCM**

(T60.1 and T60.2)

These UCMs have been linked to pathology. The scapula may forward tilt, downwardly rotate or elevate to compensate for the loss of medial rotation. A positive test (scapula UCM) has been linked with risk of impingement and symptoms (Morrissey 2005). The test is useful for diagnosis, especially for impingement, particularly when used with other impingement tests (Morrissey 2005). Uncontrolled anterior translation of the humeral head compensates for a lack of glenohumeral medial rotation. A positive test (glenohumeral UCM) has been linked with instability symptoms and risk (Morrissey 2005) (T60.3).

**Correction**

With visual, auditory and kinaesthetic cues the person becomes familiar with the task of medially rotating the glenohumeral joint to 60° without scapula movement or glenohumeral translation. Some useful clinical cues are illustrated in Box 8.2.
**T60.1** Assessment and rating of low threshold recruitment efficiency of the Kinetic Medial Rotation Test

**KINETIC MEDIAL ROTATION TEST**

**ASSESSMENT**

**Control point:**
- Prevent scapula forward tilt, downward rotation and elevation
- Prevent glenohumeral anterior translation

**Movement challenge:** glenohumeral medial rotation (supine – arm 90° abduction)

**Benchmark range:** 60° glenohumeral medial rotation

<table>
<thead>
<tr>
<th>Site</th>
<th>Direction</th>
<th>(L)</th>
<th>(R)</th>
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<tbody>
<tr>
<td>Scapula</td>
<td>Forward tilt</td>
<td>☐</td>
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<tr>
<td></td>
<td>Downward rotation</td>
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<td>Elevation</td>
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<tr>
<td>Glenohumeral</td>
<td>Anterior translation</td>
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</tbody>
</table>

**RATING OF LOW THRESHOLD RECRUITMENT EFFICIENCY**

<table>
<thead>
<tr>
<th>Item</th>
<th>✓ or X</th>
<th>✓ or X</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Able to prevent ‘give’ into the test direction</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Correct dissociation pattern of movement</td>
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<tr>
<td>Prevent scapula ‘give’ into:</td>
<td></td>
<td></td>
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<tr>
<td>• forward tilt</td>
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<td>• downward rotation</td>
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<td>• elevation</td>
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<tr>
<td>Prevent glenohumeral ‘give’ into:</td>
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<td>☐</td>
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<tr>
<td>• anterior translation</td>
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<td></td>
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<tr>
<td>and move glenohumeral medial rotation</td>
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<td></td>
</tr>
<tr>
<td>• Dissociate movement through the</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>benchmark range of 60° glenohumeral</td>
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<tr>
<td>medial rotation (arm abducted 90°)</td>
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<tr>
<td><em>If there is more available range than the benchmark standard, only the benchmark range needs to be actively controlled</em></td>
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<tr>
<td>• Without holding breath (though it is acceptable to use an alternate breathing strategy)</td>
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<td>☐</td>
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<tr>
<td>• Control during eccentric phase</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>• Control during concentric phase</td>
<td>☐</td>
<td>☐</td>
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<tr>
<td>• Looks easy, and in the opinion of the assessor, is performed with confidence</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>• Feels easy, and the subject has sufficient awareness of the movement pattern that they confidently prevent ‘give’ into the test direction</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>• The pattern of dissociation is smooth during concentric and eccentric movement</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>• Does not (consistently) use end-range movement into the opposite direction to prevent the give</td>
<td>☐</td>
<td>☐</td>
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<tr>
<td>• No extra feedback needed (tactile, visual or verbal cuing)</td>
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<td>☐</td>
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<tr>
<td>• Without external support or unloading</td>
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<tr>
<td>• Relaxed natural breathing (even if not ideal – so long as natural pattern does not change)</td>
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<tr>
<td>• No fatigue</td>
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</table>

**CORRECT DISSOCIATION PATTERN**

**RECRUITMENT EFFICIENCY**

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**T60.2** Diagnosis of the site and direction of UCM from the Kinetic Medial Rotation Test

<table>
<thead>
<tr>
<th>Site</th>
<th>Direction</th>
<th>(L)</th>
<th>(R)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scapula</td>
<td>Forward tilt</td>
<td>☐</td>
<td>☐</td>
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<tr>
<td></td>
<td>Downward rotation</td>
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<td></td>
<td>Elevation</td>
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<tr>
<td>Glenohumeral</td>
<td>Anterior translation</td>
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</tbody>
</table>
Kinetic Control: The management of uncontrolled movement

<table>
<thead>
<tr>
<th>Ideal</th>
<th>60° glenohumeral medial rotation without scapula movement or humeral anterior translation. No anterior displacement of coracoid or humeral head (Figure 8.7)</th>
</tr>
</thead>
</table>
| Impingement risk | Scapular forward tilt, downward rotation or elevation occurs before 60° glenohumeral medial rotation is achieved. Note anterior displacement of coracoid with (stable) humeral head ‘tagging along’ in proportion (Figure 8.8)  
• Confirm the impingement risk with impingement tests and palpation |
| Instability risk | Humeral head anterior translation occurs before 60° glenohumeral medial rotation is achieved. Note anterior displacement of humeral head with (stable) coracoid maintaining stable position (Figure 8.9)  
• Confirm the direction(s) of instability with instability tests |
| Combined impingement and instability risk | Scapular forward tilt, downward rotation or elevation occurs before 60° glenohumeral medial rotation is achieved. However, excessive humeral head anterior translation also occurs before 60° glenohumeral medial rotation is achieved. Note anterior displacement of coracoid with the humeral head moving even further forward of the (unstable) coracoid  
• Differentiate to determine whether symptoms are primarily due to impingement or instability |

**Box 8.2 Useful clinical facilitation and retraining cues**

**Cues for facilitation and feedback to enhance teaching and retraining movement**
- Palpate the scapula or glenohumeral joint to monitor the UCM.
- Imagery of rotating the glenohumeral joint about a coronal axis (proprioceptive feedback can be given through olecranon).
- Keep the coracoids open and wide.
- Palpate acromion/coracoid.
- Visualise a string holding the acromion up.
- Unload passively.
- Tape (proprioceptive skin tension).
- Keep same distance between coracoid and ear.
- Keep shoulder blades wide.

**An alternative position for retraining the KMRT**

Lean against a wall with the wall supporting the shoulder blade position. The upper body has to turn 15–30° off the wall so that the shoulder blade and upper arm can be supported flat on the wall. Only rotate the shoulder forwards as far as the neutral scapula can be controlled (Figure 8.10). When rotation control on the wall is efficient progress to the same movement unsupported away from the wall (Figure 8.11).
Figure 8.9 Kinetic medial rotation test uncontrolled glenohumeral movement

Figure 8.10 Correction with wall support

Figure 8.11 Correction standing – unsupported
**Shoulder lateral rotation control**

**OBSERVATION AND ANALYSIS OF SHOULDER LATERAL ROTATION**

**Description of ideal pattern**

This can be observed in standing or supine. While standing with the humerus by the side in the scapular plane (elbow forward of the anterior axillary line) and the elbow flexed to 90° (hand pointing forwards, palm in) there should be approximately 60° range of functional lateral turn out of the arm – at least 45° being glenohumeral lateral rotation and about 15° coming from scapular retraction. The glenohumeral lateral rotation should be the dominant movement early in the movement with the scapula contributing later in range.

The therapist assesses the passive range of glenohumeral lateral rotation. There should be 45° of independent passive lateral rotation. It should be relatively easy to independently dissociate the 45° glenohumeral lateral rotation from the scapular movement (Figure 8.12).

**Movement faults associated with glenohumeral lateral rotation**

Scapular retraction initiates or dominates the early range of functional arm turn out. This indicates either a restriction (relative stiffness) of glenohumeral lateral rotation or compensation (relative flexibility) of scapular retraction.

**Relative stiffness (restriction)**

- *Reduced glenohumeral lateral rotation with the elbow by side.* Functional restrictions of glenohumeral lateral rotation are identified with the scapula stabilised and the arm by the side. A significant loss of glenohumeral lateral rotation with the arm by the side is frequently identified. This restriction of functional lateral rotation range may be due to several reasons:
  - *Capsular restriction.* A capsular restriction may cause a loss of lateral rotation with the arm by the side but there will be significant (if not greater) loss of lateral rotation with the arm elevated to 90°.

Examination of the shoulder ‘quadrant’ test (Maitland et al 2005) would be positive for a capsular restriction. If at 90° of arm abduction the lateral rotation range is normal then the capsule is a very unlikely source of restriction.

- *Loss of posterior translation of the humerus at limit of lateral rotation.* At the limit of active or passive glenohumeral lateral rotation the capsule tensions anteriorly and the humeral head is forced to translate posteriorly in order to achieve full range (Moseley et al 1992; Wilk et al 1997). A loss of this posterior translation of the humeral head at the limit of lateral rotation can significantly reduce the ability to achieve full active or passive lateral rotation at the shoulder when the arm is by the side. This is identified by a decreased range of joint play and a restricted end feel on posterior translation of the humeral head at end range lateral rotation. Appropriate mobilisation of this articular restriction (e.g. with glenohumeral accessory anteroposterior
### Table 8.6 Summary and rating of shoulder girdle tests

#### UCM DIAGNOSIS AND TESTING

<table>
<thead>
<tr>
<th>SITE</th>
<th>DIRECTION</th>
<th>CLINICAL PRIORITY</th>
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</thead>
<tbody>
<tr>
<td>TEST of stability control</td>
<td>RATING (√√ or √X or Xx) and rationale</td>
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<tr>
<td>Scapula</td>
<td>Downward rotation</td>
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<td>Kinetic medial rotation test</td>
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<td>Kinetic lateral rotation test</td>
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<td>Arm extension test</td>
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<td>Arm flexion test</td>
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<tr>
<td>Kinetic medial rotation test</td>
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REFERENCES


Impaired control of scapular rotation during a clinical dissociation test in people with a history of shoulder pain. 2009 3rd International Conference on Movement Dysfunction, Edinburgh, UK.


