

Masterclass

Rotator cuff related shoulder pain: Assessment, management and uncertainties



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ABSTRACT

Introduction: Rotator cuff related shoulder pain (RCRSP) is an over-arching term that encompasses a spectrum of shoulder conditions including; subacromial pain (impingement) syndrome, rotator cuff tendinopathy, and symptomatic partial and full thickness rotator cuff tears. For those diagnosed with RCRSP one aim of treatment is to achieve symptom free shoulder movement and function. Findings from published high quality research investigations suggest that a graduated and well-constructed exercise approach confers at least equivalent benefit as that derived from surgery for; subacromial pain (impingement) syndrome, rotator cuff tendinopathy, partial thickness rotator cuff (RC) tears and atraumatic full thickness rotator cuff tears. However considerable deficits in our understanding of RCRSP persist. These include; (i) cause and source of symptoms, (ii) establishing a definitive diagnosis, (iii) establishing the epidemiology of symptomatic RCRSP, (iv) knowing which tissues or systems to target intervention, and (v) which interventions are most effective.

Purpose: The aim of this masterclass is to address a number of these areas of uncertainty and it will focus on; (i) RC function, (ii) symptoms, (iii) aetiology, (iv) assessment and management, (v) imaging, and (vi) uncertainties associated with surgery.

Implications: Although people experiencing RCRSP should derive considerable confidence that exercise therapy is associated with successful outcomes that are comparable to surgery, outcomes may be incomplete and associated with persisting and recurring symptoms. This underpins the need for ongoing research to; better understand the aetiology, improve methods of assessment and management, and eventually prevent these conditions.

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1. Introduction

In most cases assessing an individual presenting with a musculoskeletal shoulder problem involves making clinical decisions in highly ambiguous situations (Lewis et al., in press). As part of the assessment process, clinicians need to determine if the symptoms are:

- (i) Referred or related to another cause (e.g. from the cervical, thoracic, abdominal regions, neural and vascular tissues)?
- (ii) Primarily related to a stiff shoulder (e.g. frozen shoulder, osteoarthritis, locked dislocation, neoplasm—such as osteosarcoma)?

- (iii) Due to shoulder instability?
- (iv) Related to the soft tissues (e.g. rotator cuff, bursa)?
- (v) Due to combinations of the above?

In addition to this, clinicians need to; exclude serious pathology, consider pain mechanisms, determine the relationship and influence of other co-morbidities, and discern the contribution from often profound, obscured and interwoven psychosocial factors. The process is complicated and becomes more so with the emergence of new research information from a multitude of specialities, which is frequently incomplete, and often contradictory.

The purpose of this Masterclass is to focus on one musculoskeletal shoulder problem, rotator cuff related shoulder pain (RCRSP), and discuss; function, pain, aetiology, imaging, surgery, assessment and management.

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2. Rotator cuff function

The rotator cuff (RC) muscles and tendons are commonly considered to be recruited synchronously and equally to dynamically stabilise the humeral head onto the glenoid fossa during shoulder movement. This precept has been challenged and laboratory data suggest that the supra- and infraspinatus are recruited preferentially during shoulder flexion and subscapularis is recruited at higher levels during extension. Higher supra- and infraspinatus activation during shoulder flexion may contribute to reducing anterior glide of the humeral head during activities involving shoulder flexion, and subscapularis activity to reducing posterior glide during extension (Wattanaprakornkul et al., 2011). This asynchronous activity may potentially be used to inform exercise prescription. In addition, in higher ranges of shoulder elevation, such as unsupported abduction, the stabilising function of the cuff may be replaced by the deltoid which may be better oriented to stabilise the humeral head onto the glenoid fossa while allowing the RC to externally and internally rotate the glenohumeral joint (Boettcher et al., 2010). In addition, the belief that supraspinatus initiates abduction appears to be incorrect (Reed et al., 2013).

Clinically, rotator cuff tests have been developed to assess each of the individual tendons by placing the shoulder into defined positions and applying a force to the arm requiring the muscle tissue to contract (Magee, 2014). However the design of the RC does not permit the assessment of an individual musculotendinous unit. The tendons of the RC fuse into one structure, with the supraspinatus and infraspinatus fusing inseparably near their insertion. The muscular portion of teres minor and infraspinatus also fuse inseparably just proximal to the musculotendinous junction. The subscapularis and supraspinatus tendons fuse to form a sheath that surrounds the biceps tendon, and the RC tendons are tightly adherent to the glenohumeral joint capsule (Clark et al., 1990; Clark and Harryman, 1992). Although the interwoven nature of RC, capsule and ligament tissue would improve resistance to failure under load, it negates the possibility of testing individual structural units. The inability to test the RC musculotendinous units in isolation is further evidenced in electromyography, which has demonstrated that during the 'full' and 'empty' can tests designed to implicate supraspinatus pathology, eight to nine other muscles are equally active (Boettcher et al., 2009).

3. Rotator cuff related shoulder pain

RCRSP refers to the clinical presentation of pain and impairment of shoulder movement and function usually experienced during shoulder elevation and external rotation. Although numerous factors including; genetics (Harvie et al., 2004), hormonal influences (Magnusson et al., 2007), lifestyle factors such as smoking (Baumgarten et al., 2010) alcohol consumption (Passaretti et al., 2015), comorbidities and level of education (Dunn et al., 2014), biochemical, patho-anatomical, peripheral and central sensitisation, sensory-motor cortex changes (Lewis et al., 2015) and a raft of psychosocial factors (Dean and Söderlund, 2015) have the potential to contribute to RCRSP, excessive and mal-adaptive load imposed on the tissues appears to be a major influence (McCreech and Lewis, 2013; Cook et al., 2015).

On-going debate persists pertaining to; (i) the cause of RCRSP, (ii) the mechanism(s) responsible for the sensation of pain, (iii) the relationship between symptoms and structural failure observed within the RC tendons, and (iv) the role and extent of inflammation (Lewis, 2009a, 2011, 2015; Seitz et al., 2011; Lewis and Ginn, 2015; Scott et al., 2015). These symptoms are commonly termed RC tendinopathy which implicates the tendon as the source of symptoms.

Although this may be the case, there is no definitive way of incriminating the tendons as the painful structures and the possibility exists that the symptoms are derived from the tendons and their related tissues. As such, and in keeping with other musculoskeletal conditions where a definitive structural diagnosis is often elusive, such as in low back pain (Waddell, 2004), deriving a definitive structural pathognomonic label maybe unachievable, and terms such as; subacromial or rotator cuff pain syndrome or rotator cuff related shoulder pain (RCRSP) may be more appropriate.

4. Aetiology

With respect to mechanism, Neer (1972, 1983) argued that 95% of all RC pathology occurred as a result of irritation onto the subacromial bursa and rotator cuff tendons from the under-surface of the over-lying anterior aspect of the acromion calling the condition; subacromial impingement syndrome. This hypothesis has also been embraced by physiotherapists (Grimsby and Gray, 1997). Once diagnosed, Neer (1983) recommended 12 months of non-surgical treatment for those aged over 40 years with persistent and disabling symptoms, and advocated that if non-surgical treatment was unsuccessful, a subacromial decompression (SAD), also known as an acromioplasty, should be considered. This operation has become one of the most commonly performed musculoskeletal surgical procedures, with a reported 746% increase in the number of SADs in England from 2001 to 2010 (Judge et al., 2014). In England and the USA there has been a concurrent increase in RC repairs. In the USA there was a 141% increase in RC repairs between 1996 and 2006 and embedded within this figure is a 600% increase in repairs performed arthroscopically (Colvin et al., 2012). The reasons for this are uncertain as arthroscopic repairs have been associated with; longer operating theatre times, a higher re-tear rate (46.4%) than open repairs (38.6%) and clinically with no significant difference in outcome (Carr et al., 2015).

The relevance of the acromion to the development of symptoms and RC tears also remains uncertain. The argument that acromial irritation leads to RC pathology is not supported by observational studies. Payne et al. (1997) reported 91% (39/43) of RC tears occurred on the inferior (articular or joint) side of the tendon with only 9% (n = 4) occurring on the bursal side (i.e. the side under the acromion). Fukuda et al. (1987) reported that 82% (n = 27) of tears were joint side or intratendinous and 28% (n = 6) were located on the bursal side. Ellman (1990) reported that from a total of 126 intra-operative investigations of the rotator cuff, 76% (n = 96) were found to have articular side tears, 14% (n = 17) bursal side, and 10% (n = 13) had both. This repeated and consistent finding that tears are predominantly located within the tendon or on the articular side does not support the acromial impingement model. Of relevance, already in 1934, Codman identified articular side tears calling them 'rim rents', stating; '...I am confident that these rim rents account for the majority of sore shoulders. It is my unproved opinion that many of these lesions never heal, although the symptoms caused by them usually disappear within a few months'. In reality, whether these tears cause pain or not, has never been established.

Observed variations in RC morphology may explain some of these findings. Hashimoto et al. (2003) reported greater fibre degeneration and disorientation in the middle and deeper fibres of the RC and argued that degeneration was the primary cause of RC tears. Nakajima et al. (1994) reported that the deeper fibres of the RC had a relatively smaller cross-sectional area than the articular side fibres and the lower fibres failed at approximately half the tensile load of those located superiorly. Bey et al. (2002) reported strain within the supraspinatus tendon that increased with increasing joint elevation between 15° and 60° abduction with no significant difference in strain between the upper and lower fibres.

As the lower fibres are relatively weaker, the suggestion is that the joint-side fibres are more vulnerable to a tensile load than the bursal-side fibres (Nakajima et al., 1994) during shoulder elevation and as such maybe more susceptible to failure and this may be independent of acromion irritation. Bigliani et al. (1986) described three distinct acromial shapes (flat, curved and hooked) attributing the differences to morphological variations and argued those with a Type III or hooked acromion would be more likely to experience subacromial pain symptoms and suffer a RC tear. Definitive causation has not been established and there appears to be a poor correlation between acromial shape and symptoms (Gill et al., 2002; Worland et al., 2003; Snow et al., 2009) further challenging the acromial model of RC tendinopathy. In addition, if the acromion is the cause for impingement and this eventuates in RC tears, a SAD should halt this process. This hypothesis has been disputed in studies that have demonstrated that a SAD provides no clinically important effect over a structured and supervised exercise programme (Ketola et al., 2013), and others reporting the development of RC tears following an acromioplasty for people diagnosed with impingement syndrome (Hyvonen et al., 1998). Additionally, contact between the acromion and RC has been investigated (Chang et al., 2006) with reconstructed magnetic resonance (MR) images challenging the construct that osseous impingement by the acromion is the primary cause of subacromial impingement syndrome and RC tears. If comparable clinical improvement can be achieved with an exercise program without performing a SAD, and if RC tears occur following an acromioplasty, the hypotheses supporting the acromial impingement model are substantively challenged. These issues have been previously discussed in detail (Lewis, 2011; Lewis et al., 2015). The importance of the acromion is further challenged. Henkus et al. (2009) randomised 57 people with subacromial impingement syndrome to acromioplasty and bursectomy or bursectomy alone. At a mean 2.5 year follow up both groups reported comparable results with no significant difference in Constant score, Simple Shoulder test, and visual analogue scores for pain and function. They argued their results supported an intrinsic degenerative model for RC pathology rather than an extrinsic irritation model. Detailed descriptions of normal tendon composition, structure and function, and changes associated with tendinopathy have been published (Cook and Purdam, 2009; Lewis, 2009a, 2010; McCreesh and Lewis, 2013; Cook et al., 2015; Scott et al., 2015; Screen, 2015).

Osteophytes have been observed on the acromial but not the coracoid side of the coracoacromial ligament (Edelson and Taitz, 1992). The development of osteophytic spurs may be a secondary phenomenon (Chambler et al., 2003a) resulting from increased tension in the coracoacromial ligament as the arm is abducted (Chambler et al., 2003b). As the coracoacromial ligament is more trapezoid in shape, the smaller area of insertion on the acromial side may account for relative increase in tension and be the mechanism stimulating osteophyte growth at this location. If this hypothesis is correct then the Type II (curved) and Type III (hooked) acromion (Bigliani et al., 1986, 1991) may not be morphological variants, but may result from strain in the coracoacromial ligament disproportionately affecting the relatively smaller area of insertion on the acromial side. Chronic strain under the coracoacromial ligament may result from increases in RC tendon volume with significant differences being demonstrated in people diagnosed with RC tendinopathy (McCreesh et al., 2014) as well as RC fatigue or failure leading to superior translation of the humeral head during arm elevation (Mura et al., 2003). The coracoacromial ligament is a potential source of symptoms for people diagnosed with RCRSP with significant increases in nociceptive nerve fibres identified in this structure in this patient group (Tamai et al., 2000). As a causal relationship between acromial shape, acromial irritation and

rotator cuff pathology has not been demonstrated, clinicians in discussion with patients should not implicate the acromion as the cause of RC tears or symptoms, and the term 'impingement' may create an incorrect perception of pathogenesis.

5. Relationship between imaging and symptoms

Clinical diagnoses are established through discussion with patients and clinical assessment procedures that may be supported by imaging studies and laboratory investigations (Hegedus et al., 2015; Hegedus and Lewis, 2015; Lewis et al., in press). The reliance on imaging may be problematic, with investigations reporting substantial numbers of people without symptoms demonstrating RC structural failure (Table 1) (Milgrom et al., 1995; Sher et al., 1995; Frost et al., 1999; Girish et al., 2011).

These findings have profound clinical implications. The sensitivity, specificity, predictive values and likelihood ratios for clinical tests are based on gold standard reference tests that have identified structural failure and these findings are then compared with the responses produced using special orthopaedic tests such as the battery of rotator cuff tests and tests for impingement. The gold standard tests are generally considered to include; US, MRI and direct observation during surgery. However, the substantial numbers of people without symptoms who demonstrate structural failure largely invalidates US, MRI and direct intra-operative observation as gold standard reference tests. In addition, these findings also invalidate the majority of special orthopaedic tests and combinations of these tests as procedures to definitively rule in the structure that is causing the patient's symptoms. If anything, these tests should be considered as symptom provocation procedures (Lewis, 2009b).

The implication of a lack of a real 'gold-standard' comparator is that many people with shoulder symptoms who are given a structural diagnosis based on clinical orthopaedic tests and/or imaging procedures will be given incorrect diagnoses. A further implication is that patients will be listed for and undergo surgical operations on tissues that are not related and not the cause of their symptoms. It is unlikely a patient would agree to start insulin injections based on considerable inaccuracies in the diagnostic tests for diabetes, yet current clinical practice suggests that this may not be the case for many orthopaedic surgical procedures and musculoskeletal treatments.

6. Additional uncertainties associated with surgery

Success rates (good to excellent results) of up to 70–90% following SAD have been reported (Ellman and Kay, 1991; Spangehl et al., 2002). Following surgery there is an extended period of reduced activity and graduated return to function that might take many months. Post-operative protocols typically dictate very slow and gentle movements in the early stages of rehabilitation, with the avoidance of active shoulder movement if the RC has been repaired. Following SAD, studies from Australia and the United Kingdom, have reported that non-manual workers take up to an average of 6 weeks and manual workers up to an average of 3 months to return to work with an avoidance of driving for up to 4 weeks (McClelland et al., 2005; Charalambous et al., 2010). A relative reduction in tissue loading (relative rest) is frequently recommended in the early stages of treating a tendinopathy (Lewis, 2010, 2014; Cook et al., 2015). The relative rest and graduated rehabilitation imposed by the surgery may be the reason for improvement in symptoms and not the surgery itself (Lewis, 2011, 2015). This hypothesis needs to be tested in an appropriately designed investigation, but until it has, surgeons need to be circumspect with their explanation of the intended benefits of SAD and RC repairs to those

Table 1
Findings from imaging studies which challenge the reliance on imaging to determine the cause of symptoms.

- In a magnetic resonance imaging (MRI) study, [Sher et al. \(1995\)](#) reported that 54% of people over the age of 60 years without shoulder symptoms demonstrated a partial (PTT) or full thickness RC tear (FTT).
- [Milgrom et al. \(1995\)](#) in an ultrasound investigation (US) reported that the incidence of RC tears increased with age and PTT and FTT were present in the dominant shoulder of 50% of people without any shoulder symptoms in their 7th decade and in 80% in those aged over 80 years.
- The baseball pitch requires considerable precision and places substantial challenges on the shoulder. From the cocking position the shoulder moves 80° in 30 msec generating peak shoulder internal rotation velocities between 6000 and 7000°/sec, and following ball release the shoulder decelerates from these speeds to cessation of movement within 50 msec ([Pappas et al., 1985](#); [Feltner, 1986](#); [Dillman et al., 1993](#); [Fleisig et al., 1999](#)). Ball speeds have been reported to reach 170 km/h. MRI findings in asymptomatic professional baseball pitchers have demonstrated; RC abnormalities in the throwing shoulder (79%) and non-throwing shoulder (86%) and labral abnormalities (79%) in both shoulders ([Miniaci et al., 2002](#)).
- In asymptomatic elite level overhead athletes (pitchers and tennis players), 40% were found to have PTT and FTT of the RC on their dominant side, and when followed up after 5 years none had reported any symptoms or had required treatment, leading the authors to conclude that MRI alone should not be used as the basis for operative intervention ([Connor et al., 2003](#)).
- [Frost et al. \(1999\)](#) reported that in an MRI investigation, an equivalent percentage of people with subacromial impingement (55%) and those without shoulder symptoms (52%) had RC pathology. The prevalence of RC pathology increased with age and the pathology observed in the MRI did not correlate with symptoms.
- [Girish et al. \(2011\)](#) using US, reported that shoulder abnormalities including; subacromial bursal thickening, supraspinatus tendinosis and tears, and glenoid labral abnormalities, were identified in 96% of men who did not have shoulder symptoms. The relationship between bursal thickening and symptoms has been challenged ([Daghir et al., 2012](#); [Couanis et al., 2015](#)), with the possible exception of a substantial single endurance event (21 km swim) ([Couanis et al., 2015](#)).

considering surgery. Two separate research investigations have suggested that the outcomes of knee surgical procedures for painful osteoarthritis of the knee and degenerative medial meniscus tears may be due to placebo ([Moseley et al., 2002](#); [Sihvonen et al., 2013](#)). This may be an additional reason for success attributed to shoulder surgery and warrants investigation in the shoulder, where one group receives sham surgery that does not directly influence the glenohumeral joint. Furthermore, patients are frequently informed that removing the irritation caused by the acromion, repairing the damaged tendon tissue, and avoiding an increase in the size of the RC tendon tear, are amongst the intended benefits of surgery. These precepts have also been challenged in investigations that suggest outcome may be related more to numbers of co-morbidities and level of education than tendon structural factors ([Dunn et al., 2014](#)).

7. Assessment and the Shoulder Symptom Modification Procedure

Assessment involves taking a detailed history and discussion with the patient, screening and on-going monitoring for potential red-flag presentations, functional/disability questionnaires, assessment of impairment that may include; range of movement, strength, posterior capsule extensibility, neural tests, pain behaviour, etc. Increasing and decreasing load on the muscle tendon-unit may also help to support clinical hypotheses. Orthopaedic tests and imaging may support the clinical examination. However, as discussed the value of orthopaedic tests may be no more than symptom reproduction and clinical reasoning to implicate the structure(s) based on these tests has been challenged ([Lewis, 2009b](#)).

Narrative and systematic reviews have challenged the extent to which a definitive structural diagnosis is achievable ([Hegedus et al., 2012](#); [Hegedus and Lewis, 2015](#)). In response to this, suggestions have been presented to use symptom modification (also known as treatment direction test/treatment classification tests/improvement test) as a method to inform clinical management ([Lewis et al., in press](#); [Lewis, 2009b](#); [Lewis et al., 2015](#)).

One such approach is the Shoulder Symptom Modification Procedure (SSMP) first described in 2009 ([Lewis, 2009b](#)). The SSMP ([Table 2](#)) systematically investigates the influence of thoracic posture, three planes of scapular posture (and combinations of scapular positioning), and humeral head position (using a battery of tests) on shoulder symptoms. SSMP assessment techniques found to reduce symptoms are then used to treat the symptoms. If the first three stages of the SSMP do not completely alleviate or reduce symptoms, then the final stage of the SSMP involves assessing the

influence of manual therapy procedures throughout the cervical, thoracic, and shoulder regions that may modulate shoulder symptoms. The process has been presented and discussed in detail elsewhere ([Lewis et al., in press](#); [Lewis, 2009b](#); [Lewis et al., 2015](#)). For the cervical and lumbar regions, there is evidence, albeit limited, that techniques found to improve symptoms within a session may be used to guide treatment selection and may contribute to predicting a between session change in symptoms ([Hahne et al., 2004](#); [Tuttle, 2005](#); [Cook et al., 2014](#)). The SSMP is supported by anecdotal evidence and as such its implementation should be considered cautiously and is only one of many assessment options available to clinicians. If proven to be of clinical value the process needs to adapt and evolve to new and emerging research. Research is needed to establish the concepts reliability and to establish the component parts (if any) that can be used to inform patient management. One important and possible hypothesis that underpins the use of the SSMP is that it may contribute to reducing the ‘threat’ associated with a symptomatic movement or activity and in turn contributes to improving function, by giving the individual the confidence to move. Manual therapy and other forms of exercise therapy may work in the same way. [Figs. 1–5](#) detail selected SSMP assessment procedures.

The SSMP may influence the symptoms associated with RCRSP by; changing the RC muscle-tendon length tension relationships, variations in relationships of anatomical structures, neuro-modulation and non-specific tissue based responses. If resolution of symptoms is not complete following the application of the SSMP, and the RC is implicated in the clinical presentation, then a structured and graduated exercise program is instigated. A model for the staged clinical management of RCRSP has been published previously ([Lewis, 2014](#)). [Fig. 6](#) is based on this model.

8. Exercise therapy

Although a structured exercise program is unequivocally the main intervention for RCRSP ([Haahr and Andersen, 2006](#); [Ketola et al., 2013](#); [Kukkonen et al., 2014](#)) consensus on dosage, frequency, method of delivery, acceptable pain tolerance, inter-exercise activity levels, and specific exercise inclusion has not been achieved. Systematic reviews investigating exercise for RCRSP have produced varied findings.

[Desmeules et al. \(2003\)](#) reported limited evidence to support the efficacy of therapeutic exercise and manual therapy. [Kromer et al. \(2009\)](#) reported equal effectiveness of physiotherapist-led exercises compared with surgery in the long term. [Kuhn \(2009\)](#) concluded that; exercise had statistically and clinically significant

Table 2
Shoulder Symptom Modification Procedure [SSMP] v5.

Shoulder Symptom Modification Procedure [SSMP] v5

Name:	DoB:	Date:			
Symptomatic movement, activity or posture					
#1:					
#2:					
Change / improvement:					
	None	Worse	Partial	Complete	
					Comment
[1] Thoracic Kyphosis					
Thoracic extension					
Thoracic flexion					
Taping					
Manual					
[2A] Scapular Position					
Elevation					
Depression					
Protraction					
Retraction					
Anterior tilt					
Posterior tilt					
Combinations:					
[2B] Winging Scapula n/a <input type="checkbox"/>					
Manual stabilisation					
Taping 1 Taping 2 Taping 3					
[3] Humeral Head Procedures					
Eccentric elevation					
Depression - flexion (sitting / standing)					
Depression - abduction (sitting / standing)					
Depression - flexion (supine)					
Depression - abduction (supine)					
Assisted elevation-flexion					
Assisted elevation-abduction					
Elevation with ER					
Elevation with IR					
AP with inclination:					
PA with inclination:					
Other:					
[4] Symptom Modulation					

Abbreviations: P = pain, W= weak, sl= slight, sh=shoulder, +ve = positive, -ve = negative / absent, pt = patient, †= increase, ‡=decrease

SSMP v5 (2016)

effects on pain reduction and improving function, but not on range of motion or strength, and manual therapy may augment the effects of exercise. In this review home exercise programs were found to be as beneficial as supervised exercises. [Braun and](#)

[Hanchard \(2010\)](#) reported limited support for manual therapy and exercise of short to medium term effectiveness, and highlighted methodological flaws and risk of bias in many of the included studies.

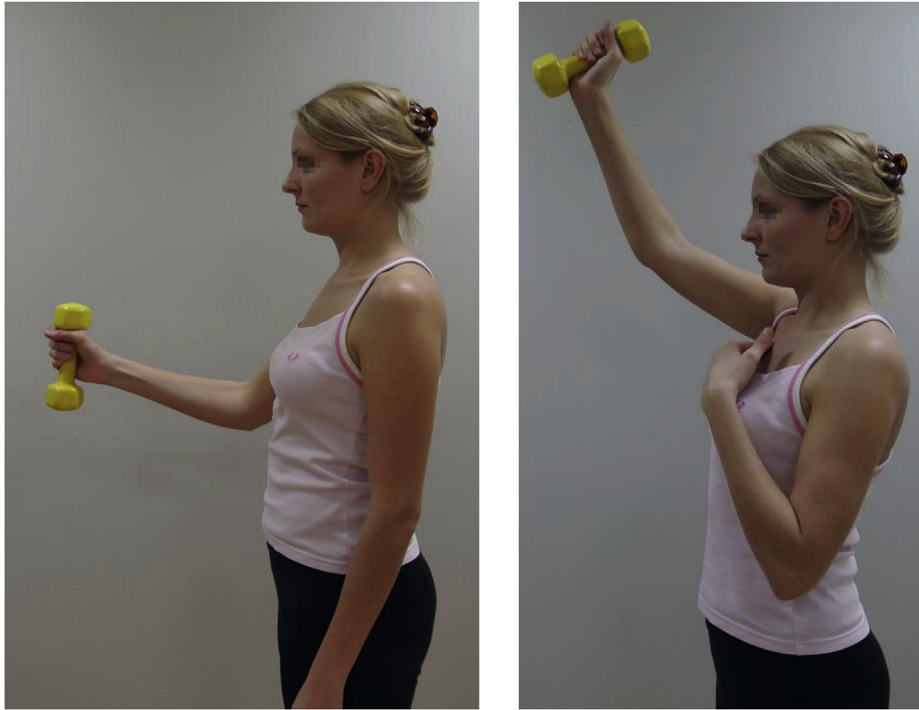


Fig. 1. SSMP (Stage 1). Example: Assessment of influence of thoracic kyphosis.

Kelly et al. (2010) reported only limited evidence was found to support the use of exercise, whereas, Hanratty et al. (2012) concluded that; strong evidence existed for exercise to decrease pain and improve function in the short-term, and moderate level evidence suggested that exercise resulted in short-term improvement in mental well-being and long-term improvement in

function. They also concluded that synthesis of definitive exercise prescription to achieve these benefits was indeterminable due to poor reporting of protocols. Abdulla et al. (2015) reported that; supervised and home based strengthening (and stretching) may lead to short term benefit in pain and disability, and that for persistent symptoms exercise leads to similar outcomes as surgery.

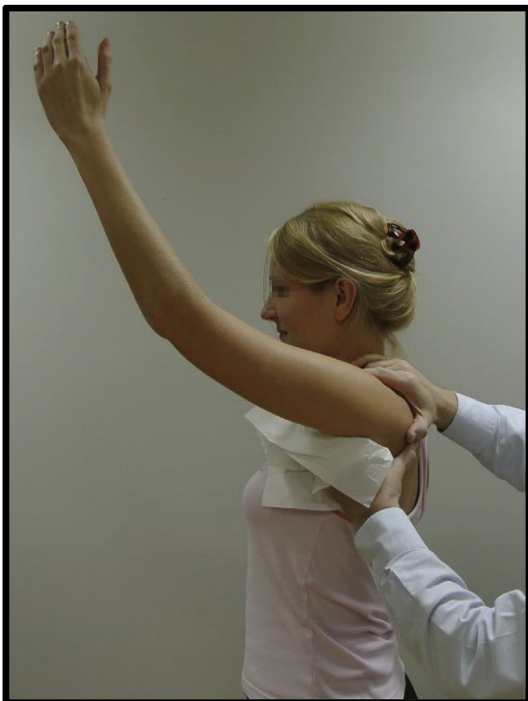


Fig. 2. SSMP (Stage 2). Example: Assessment of influence of scapular position.



Fig. 3. SSMP (Stage 3). Example: Humeral head depression – flexion. Following an isometric contraction in the direction of the solid arrow, the movement of shoulder flexion is re-assessed.



Fig. 4. SSMP (Stage 3). Example: Shoulder flexion with external rotation.

These differences in reported findings may relate to different; diagnostic categories, inclusion and exclusion criteria, quality assessment tools, and methods used within each review. Ideally treatment intervention would result in complete resolution of symptoms and in a Numbers Needed to Treat of 1 (Sackett et al., 2000; Greenhalgh et al., 2014), where everyone receiving an appropriate intervention reports a favourable outcome when compared to a control group. Unfortunately this is not the case. There are many possible reasons for this including; incomplete knowledge of the source and mechanism(s) of symptoms and inadequate and appropriate treatments to address these factors. Other factors include psychosocial influences, duration of symptoms, genetics, comorbidities, lifestyle issues and hormonal status. Inadequate and/or inappropriate provision of treatment will also influence outcome. One possible reason for suboptimal outcome is not stratifying intervention according to clinical presentation as has been suggested in other musculoskeletal conditions (O'Sullivan, 2005). As detailed in Fig. 6, one method of stratifying management for RCRSP is to sub-categorise the clinical presentation into irritable, non-irritable and advanced. Overlap in management strategies exist between these sub-categories.

9. Management of rotator cuff related shoulder pain

Common to all clinical presentations is the need to engage with the individual experiencing the symptoms, allowing the person to voice their needs and concerns. Understanding and acknowledgement of the impact of the problem demonstrates empathy. In addition, patients should be given the opportunity to discuss their understanding of the cause of the symptoms, how quickly they expect to recover, the treatments they may consider to be effective, and, their thoughts on the treatment the clinician recommends. Clinicians should provide information relating to cause, prognosis and expected outcome and avoid 'threatening' language such as 'acromion impinging and wearing away or tearing into the tendon'. Patients need to understand that the management of tendon related problems requires at least the same respect as afforded to

the management of other musculoskeletal problems, such as fractures. A person with a fracture not demonstrating clinical and radiological union involving the upper arm or forearm would be unable to perform certain activities such as; serving in tennis, hammering nails into walls and would need to make certain compromises with respect to the fracture. Frequently tendon related problems are harder to treat and follow a more protracted course than fractures and the requirement to adhere to appropriate levels of activity is necessary. Managing a tendon related problem should not be seen as only treating a local problem and attention to wider issues, including lifestyle factors, are essential (Dean and Söderlund, 2015). Providing a local shoulder based treatment in isolation maybe an ineffective management strategy in the management of RCRSP. Clinicians and patients need to appreciate that many activities involving the shoulder require that energy is transferred from the lower to upper limbs through the trunk and an inability to transfer energy from the lower limbs may result in increased demands on the shoulder (Kibler, 1995; Kibler and Chandler, 1995; Seroyer et al., 2010; Sciascia and Cromwell, 2012), potentially leading to local overload and local symptoms. This is easy to appreciate and the clinician can facilitate this by asking the patient to describe how they would throw a ball, serve in tennis or perform some other similar activity. Although currently there is no definite clinical method of identifying the impact that; movement restriction, pain, fatigue in the lower limbs and trunk has on the shoulder, clinicians should consider including exercise and appropriate management for these regions commensurate with the patient's level of function and functional requirements. Sensory-motor exercises appropriate for the patient's level of function may also enhance outcome.

9.1. Irritable RCRSP

This may occur in acute and chronic presentations and is commonly characterised by easily aggravated and prolonged (sometimes constant) shoulder pain once provoked, together with night pain. Limited research implicates subacromial bursal involvement in this presentation (Santavirta et al., 1992). As with all presentations load management (relative rest) is important and the aim is to identify a level of activity that reduces the amount of pain experienced. Training using resistance exercise bands or other equipment, performed quickly, under high loads and repetitions, commonly aggravates this clinical presentation, and one potential reason for the equivocal findings reported by the systematic reviews outlined previously, may relate to not considering the individual's response to exercise.

Anecdotal evidence suggests that isometric exercises, with the arm supported, performed in the direction of pain may help to control pain. Although there is some evidence to support this observation, (Hoeger Bement et al., 2008; Lemley et al., 2014; Rio et al., 2015), conclusive evidence for the shoulder is not available. The response to isometric exercises may be assessed across the spectrum of presentations (i.e. irritable, non-irritable and advanced). Additional anecdotal evidence suggests that the application of ice wraps may also be helpful in controlling pain. Exercise in the form of gentle loading, such as short lever flexion exercises may be well tolerated, as may shoulder flexion supported on a ball, which may be progressed as the patient is able (Fig. 7). This and other examples of motor control exercises should not increase irritability and when appropriate should incorporate lower limb weight transfer.

Pharmacological intervention may be required to control symptoms. All medicine is associated with risk, including; adverse reactions, side effects and interactions, and the risks must be considered together with the intended benefits. Non-

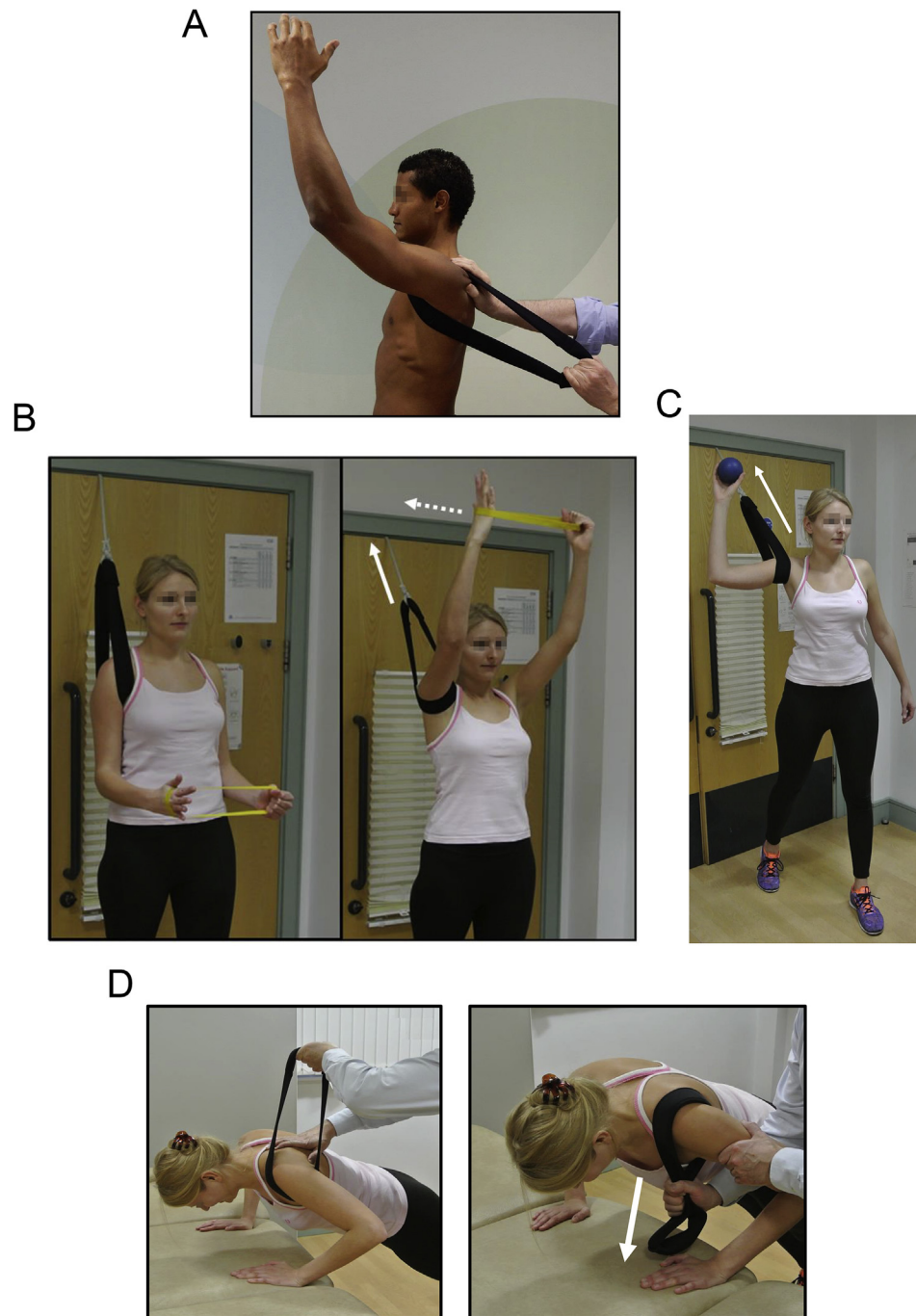


Fig. 5. SSMP (Stage 3). A: Example: Shoulder flexion with anterior-posterior stabilisation with neoprene shoulder stabilisation belt. B: Example: Shoulder flexion with external rotation (dashed arrow) and anterior-posterior-superior glide (solid arrow) with neoprene shoulder stabilisation belt. C: Example: Shoulder throwing action with neoprene shoulder stabilisation belt, providing an anterior-superior force over the anterior aspect of the shoulder. D: Example: Weight bearing assessment with anterior-posterior, and posterior-anterior pressure with neoprene shoulder stabilisation belt.

steroidal anti-inflammatories have been associated with myocardial infarction and stroke, with the risk increasing even after several weeks of use. In addition, corticosteroids have been associated with deleterious effects on rotator cuff tissue (Dean et al., 2014a, 2014b). Although there is a worrying paucity of evidence to support injection therapy for the shoulder, studies that have compared analgesic only injections to corticosteroid and analgesic injections have reported comparable outcomes up to 6 months (Akgun et al., 2004; Alvarez et al., 2005) and due to the local and systemic risks associated with corticosteroids,

analgesic only injections, although not without risk, could possibly be considered as first line injection management. One possible reason for this is that both glucocorticoids and local anaesthetics appear to be able to reduce tenocyte numbers (Scherb et al., 2009; Carofino et al., 2012). Tenocyte proliferation has been associated with tendinopathy (Scott et al., 2015; Screen, 2015) and one possibility is that injection therapy may contribute to restoration of tendon cellular homeostasis by reducing tenocyte numbers. This hypothesis requires appropriate scrutiny. Injection therapy may help reduce pain to permit

Model for the Clinical Spectrum of Rotator Cuff Related Shoulder Pain

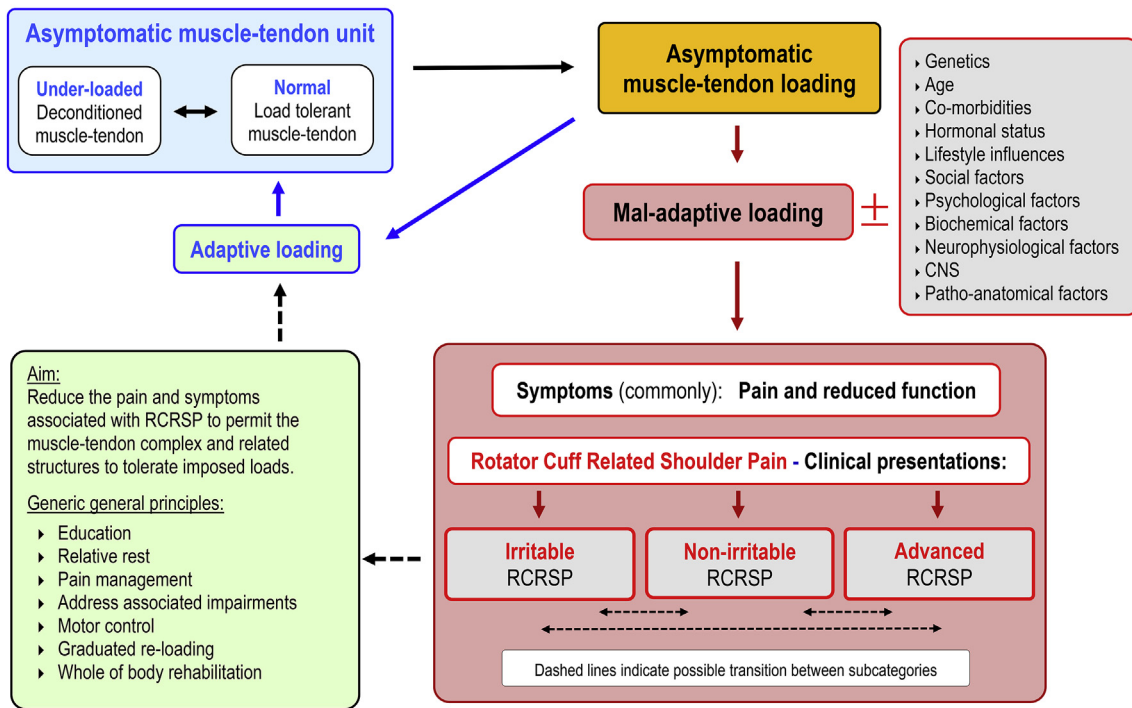


Fig. 6. Clinical model for the management of RC related shoulder pain.

exercise therapy to continue more effectively but may not confer long term benefit (Crawshaw et al., 2010).

9.2. Non-irritable RCRSP

Non irritable RCRSP is characterised by pain (that may range from mild to severe) that increases with movement and no or

minimal irritability. Pain and weakness is most commonly experienced in the direction of external rotation and elevation and the following relates to this presentation.

Non-irritable RCRSP may benefit from a graduated shoulder flexion program such as described in Fig. 7 and progresses to short lever (i.e. elbow bent) shoulder flexion initially without, then with, increasing weights and resistances. Free weights may be used and

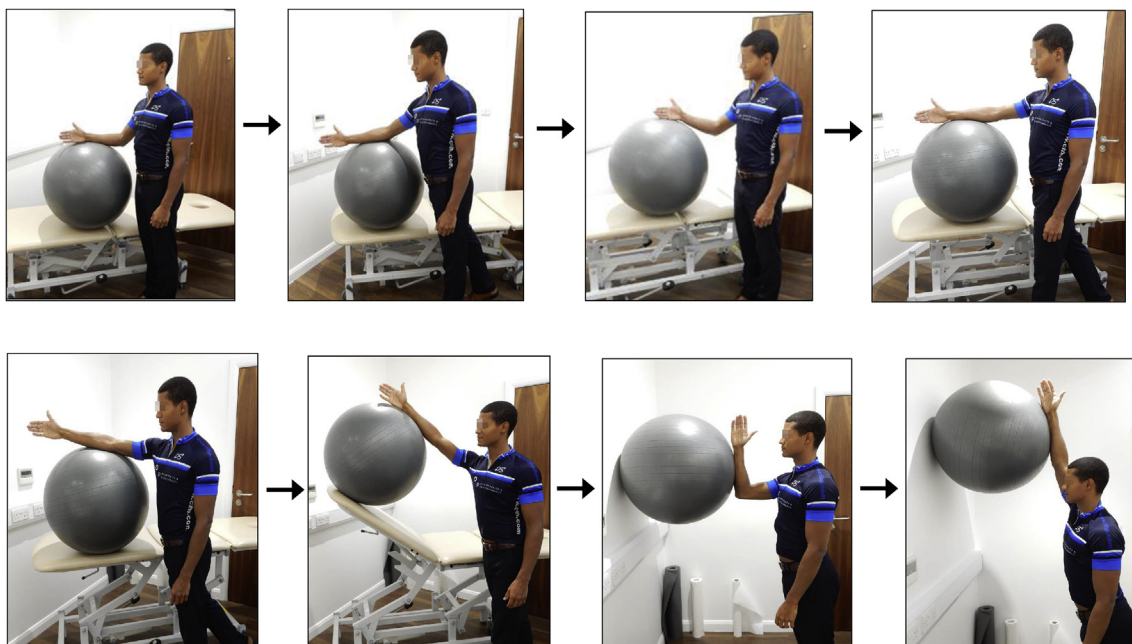


Fig. 7. Graduated shoulder flexion exercises used in motor control training and in early stages of rehabilitation.

another option is to secure resistance tubing under a door, using lower limb weight transference and flexing the shoulder within pain free ranges. As pain decreases, range may be progressed as can the resistance. One such exercise is a combination of the technique detailed in Fig. 4, with resistance bands secured under the foot or a door and attached to the hand on the symptomatic side. The effect of holding a tennis ball in the symptomatic and/or contralateral hand during the exercise can also be assessed.

In addition to a graduated and progressive program performed in the direction of shoulder flexion, a concurrent graduated shoulder external rotation program should be introduced. Entry point into the program should be commensurate with the patient's physical ability and pain response. A comprehensive approach to exercise therapy for RCRSP has recently been published (Lewis et al., 2015).

Clinicians should design treatment programs that include strengthening and endurance exercises. Strength programs are typically based on an assessment of 1 Repetition Maximum (1RM). One method of assessing this is to use a hand held dynamometer (Dollings et al., 2012). If pain precludes a definitive assessment on the symptomatic side, the asymptomatic side can provide guidance as to the expected strength on the symptomatic side. Assessment in different shoulder joint positions is often more informative than standard arm by the side tests. Guidance on strength and endurance programs have been published (McGarber et al., 2011; McArdle et al., 2014; Jull et al., 2015).

One strategy to ensure that the patient can tolerate the load imposed during the exercise program and to be able to assess the influence of change, is to only introduce one change at a time, and monitor its influence over a 24 h period. There may be some benefit in developing an exercise program that delivers training in a range of postures and arm positions (even within one training session) to introduce variability in loading. Another strategy that may be considered, especially if the response to strengthening exercises has plateaued, is to permit a degree of pain during the exercise (Holmgren et al., 2012). Pain that is tolerated by the patient in the order of 2–5 out of 10 as reported on an analogue scale of pain (where 10 is the worst imaginable pain) may be introduced to determine if this contributes to improvement. Pain should settle soon after the exercise and must not increase pain at night or pain experienced over 24 h.

9.3. Advanced RCRSP

End stage or advanced RCRSP is characterised by massive and inoperable RC tears. These tears are frequently (but not always) associated with substantial morbidity and represent a clinical challenge for physiotherapists. As these tears are generally unreparable, surgeons may recommend joint replacement surgery (Harreld et al., 2012). The findings of a randomised placebo controlled clinical trial (Ainsworth et al., 2009) suggested that an exercise program specifically designed for this clinical presentation demonstrated significant benefit at three and six months. This program may be enhanced by SSMP techniques and exercises described in the irritable and non-irritable RCRPS stages, including the exercises depicted in Fig. 7.

Although there are many reasons for suboptimal outcomes, people with RCRPS should derive considerable confidence from the research literature that they should achieve at least comparable results as they would if undergoing surgery for RCRSP (Haahr et al., 2005; Haahr and Andersen, 2006; Ketola et al., 2009, 2013), and, atraumatic partial thickness tears (Kukkonen et al., 2014). This extends to people diagnosed with atraumatic full thickness tears (Kuhn et al., 2013) as well as those diagnosed with RCRSP who have previously failed non-surgical treatment (Hallgren et al., 2014).

These studies suggest that patients with RCRSP managed with exercise therapy should expect equivalent outcomes to surgery. It is possible that the addition of whole body exercises, manual therapy, life style management, screening of potential postural factors as identified during assessment procedures such as the SSMP, and staged rehabilitation relevant to the patient's clinical presentation may further enhance outcomes. This hypothesis would require appropriate investigation. In addition, research is required to identify those that may not benefit from an exercise approach and require alternative management.

10. Conclusion

Shoulder diagnosis is fraught with difficulty and assessment techniques such as the SSMP (Lewis, 2009b) may help direct management. Although no intervention can currently guarantee complete reduction in symptoms, both physiotherapists and people with RCRSP should derive confidence that an exercise based approach produces equivalent outcomes when compared to surgery for those diagnosed with subacromial impingement syndrome/RC tendinopathy, and those with atraumatic partial and full thickness tears. There is also evidence that a graduated exercise program will benefit those who have failed an initial course of non-surgical treatment, and those with massive inoperable RC tears. Outcomes may be further enhanced if patients are categorised into clinical presentations and appropriate intervention is offered relevant to that stage. Addressing lifestyle factors may also contribute to improved outcomes as may whole of body exercise programs. The role of manual therapy is less clear and requires further research.

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