Rotator Cuff Tendinopathy: Navigating the Diagnosis–Management Conundrum

Rotator cuff (RC) tendinopathy refers to pain and weakness, most commonly experienced with movements of shoulder external rotation and elevation, as a consequence of excessive load on RC tissues. Excessive load is a relative term and will vary within and between individuals as a consequence of changes in activity levels. Epidemiological data are difficult to determine due to a poor level of association between structural failure and symptoms, and uncertainty as to the cause and location of the symptoms. Rotator cuff tendinopathy is commonly referred to as subacromial impingement syndrome. However, the belief that acromial irritation is the primary cause of symptoms may be erroneous.

The anterior (subscapularis) and posterior (ie, attaching to the posterior surface of the scapula; supraspinatus, infraspinatus, and teres minor) RC muscles, respectively, provide internal and external rotation torque at the shoulder. The RC muscles also provide functional shoulder joint stability, with anterior and posterior RC muscles being recruited at significantly different activity levels, depending on the movement performed: shoulder flexion (greater posterior RC muscle activation) or shoulder extension (greater anterior RC muscle activation). This suggests that counterbalancing humeral head translation resulting from shoulder flexor, extensor, and abductor muscle activity is an important function of the RC.

The aim of this commentary is to present information related to the function of the RC, to discuss uncertainties related to pathoetiology and assessment, and to present strategies for management.
based in part on a specific approach to shoulder symptom modification procedures (SSMPs) outlined herein. Emerging research implicating the potential for central sensitization and cortical involvement is also discussed.

Pathoetiology

Neer²⁶,⁶⁷ argued that 95% of RC pathology was caused by irritation from the overlying acromion, calling the condition subacromial impingement syndrome and recommending acromioplasty in the event of failure of nonsurgical care. Support for this pathoetiological model of RC tendinopathy is equivocal,⁶⁴ and recommendations to avoid the use of the term subacromial impingement syndrome have been made.⁶⁵,⁹⁰ The definitive cause of RC tendinopathy remains uncertain, as does the reason for the pain experienced by people with this common condition.

A poor level of association exists between symptoms related to RC tendinopathy and structural failure observed on imaging (ultrasound, magnetic resonance imaging [MRI]) or intraoperatively,⁶⁷,⁶⁸ and uncertainty persists regarding the role of inflammation in the tendon and associated bursae.²³,⁸²,⁹⁵,¹¹³ Higher concentrations of inflammatory substances have been reported in the subacromial bursal tissue in people diagnosed with RC tendinopathy. However, this finding is not consistent,⁹⁹,¹²⁰ and a definitive understanding of the relationship between bursal and tendon symptoms with respect to both causation and association remains elusive.⁵⁵

There is poor understanding of the source of the pain in RC tendinopathy, as subacromial bursectomy has been shown to be as effective as the combination of subacromial bursectomy and acromioplasty.⁶⁹ Although this finding challenges the benefit of acromioplasty, this study, like other surgical studies, did not control for placebo⁸⁴,¹⁰⁶ and the substantial relative rest following surgery.⁹⁸,⁷³ Therefore, there is no certainty that any derived benefit was due to either surgical procedure.⁶⁴ Proposed mechanisms of RC tendinopathy include intrinsic, extrinsic, or combined mechanisms.⁶⁵,¹⁰⁴ Extrinsic or external mechanisms potentially involve attrition of the RC tendons from contact with structures such as the humeral head below and the coracoacromial arch above,⁷⁷ possibly due to poor function of the musculature responsible for controlling the position of the humeral head secondary to weakness, fatigue, pain-related inhibition, and structural incompetence. Diagnostic ultrasound is both a reliable and valid method to measure the subacromial space and the acromiohumeral distance.⁷⁶,⁷⁷ Approximately 45% of people diagnosed with RC tendinopathy have a reduction in the subacromial space during elevation of the arm, which rehabilitation has the potential to normalize.⁹⁰ Studies of the effect of RC muscle fatigue suggest that the size of the subacromial space is reduced following fatigue⁷⁴ and that recovery to normal is delayed in those with RC tendinopathy.⁷⁵ In addition, electromyography studies in people with RC tendinopathy have reported reduced RC muscle activation,³¹,⁸⁵ as well as delayed onset of activation in muscles controlling position and movement of the scapula.²⁹

The evidence for the acromion being the principal cause of external irritation on the RC tendons has been challenged.⁶⁸ Variations described in acromial shape⁸ may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹ Deviations in posture from an idealized⁴ four may not be morphological but may instead develop over time,¹⁰,¹¹⁶ or be a secondary consequence of RC failure.¹⁸,⁸⁹

Cortical Changes

Intrinsic mechanisms relate to factors that directly influence tendon health and quality, including aging,¹²⁰ genetics,¹⁰⁹ vascular changes,⁴⁶ and altered loading.⁶⁶ Excessive tissue load remains the most substantial causative factor in the development of RC tendinopathy, as reflected by the fact that RC tendinopathy occurs more frequently in the dominant limb¹¹⁷ and in occupations⁷⁷ and sports with high rates of upper-limb loading.¹⁰³ Underloading may also disrupt tendon homeostasis,²³ potentially resulting in a temporally earlier point of failure, when the tendon is subject to load. Lifestyle factors such as obesity, metabolic syndrome, and smoking may increase the risk and detrimentally impact recovery of RC tendinopathy.⁵⁴,⁹⁴

There is clear potential for interaction between intrinsic and extrinsic mechanisms. McCreesh et al⁷¹ have demonstrated that RC muscle fatigue leads to short-term decrease in acromiohumeral distance and swelling of the supraspinatus tendon in people with RC tendinopathy. The enlarged tendon occupies more subacromial space, a phenomenon represented by the subacromial occupation ratio (acromiohumeral distance-supraspinatus thickness), increasing the potential for compression⁷⁹ and the possible development of secondary acromial osteophytes.⁶⁰,⁶¹ This swelling,⁷⁵ combined with loss of humeral head control (superior migration), may lead to symptoms clinically associated with subacromial impingement. As such, it would be appropriate to direct treatment to restore local homeostasis by reducing pain, improving the tendon’s capacity to sustain loading, and re-establishing humeral head control before considering surgical subacromial decompression, even in the presence of RC tendon tears and acromial osteophytes.⁵⁰,⁶⁵,⁶⁴,⁶⁸

Central Sensitization and Cortical Changes

The cause of local pain in tendinopathy remains elusive, and frequently the level of pain experienced varies substantially
among individuals. Central sensitization could contribute to explain this disparity, and several studies have investigated its role in individuals with RC tendinopathy. Gwilym et al demonstrated that a significant proportion of individuals with RC tendinopathy have pain radiating down the arm and hyperalgesia to punctate (pinprick) stimuli of the skin. Furthermore, the presence of either hyperalgesia or referred pain preoperatively is associated with a worse outcome from subacromial decompression 3 months after surgery. Two other studies compared pain thresholds between individuals with and without unilateral RC tendinopathy and found hypersensitivity at local and remote sites bilaterally in the symptomatic population, suggesting central sensitization. These findings suggest that central sensitization is present in a proportion of people diagnosed with RC tendinopathy, and that the pain experienced may not always relate to local pathology.

Another potential influence in the development or maintenance of pain is the presence of central motor alterations. Ngomo et al have shown that individuals with RC tendinopathy demonstrate decreased corticospinal excitability of the infraspinatus muscle on the affected side compared to their unaffected side. Furthermore, this interhemispheric asymmetry is associated with the duration of pain, suggesting that the corticospinal excitability may decrease over time in the affected shoulder. Corticospinal hyperexcitability at rest and hypoexcitability during voluntary activation have also been reported for the deltoid muscles in individuals with chronic full-thickness tears of the RC. These altered muscle cortical representations show adaptive changes in the central nervous system associated with RC tendinopathy and may contribute to the neuromuscular deficits associated with this disorder.

**Assessment**

Assessment involves a number of sequential and interrelated stages. Following the patient interview and careful screening for health-related systemic conditions, further information is gained from the completion of pain, quality-of-life, and disability questionnaires and measurements of impairments (active and passive ranges of movement, shoulder capsule extensibility, and muscle function: strength and endurance).

As is easily understood from observing sporting activities such as the tennis serve and pitching in baseball, as well as function during many vocational activities, energy to complete many of them is transferred from the lower limbs, through the trunk, to the shoulder. Pain, weakness, and restricted range of movement in the lower limbs or trunk are examples of deficits distal to the shoulder that have the potential to detrimentally impact shoulder function. Reduced energy transfer to the shoulder could result in higher requirements at the shoulder itself, potentially leading to early fatigue of the RC muscles and a lower threshold at which tissue failure and/or shoulder symptoms may occur. Although the validity of the whole-body screening that is relevant to every sport and vocation remains in its scientific infancy, the assessment of the influence of pain, restricted movement, instability, and weakness in the rest of the body should be considered as an integral part of shoulder assessment.

Assessment of impairment is typically followed by special orthopaedic tests designed to assess the structural integrity of the RC. Reproduction of pain and identifying weakness during these procedures are considered clinically diagnostic. However, multiple narrative and systematic reviews have concluded that the capability of these tests to assess and implicate the RC as the source of symptoms cannot be achieved with the certainty and confidence required to meaningfully inform clinical decision making. Tests have been described to individually assess the 4 RC muscles and their related tendons. A fundamental requirement for a clinical procedure to implicate a structure would be the ability of the clinical test to assess that structure in isolation; the morphology of the RC muscle group, however, does not allow for this. Clark and Harryman reported that the infraspinatus and supraspinatus fuse near their insertions and cannot be separated, and that the teres minor and infraspinatus merge inseparably just proximal to the musculotendinous junction. The RC tendons are also confluent with the capsule of the shoulder and the coracohumeral and glenohumeral ligaments. Interweaving of the RC with the glenohumeral joint ligamentous and capsular tissues negates the possibility of isolated testing of individual structures. The inability to test shoulder muscles in isolation has been demonstrated in an intramuscular electromyography investigation comparing the full- and empty-can tests, which are commonly used to test for supraspinatus pathology. During the full-can test, 8 other shoulder muscles were found to be equally activated relative to the supraspinatus, and during the empty-can test, 9 other shoulder muscles were equally activated, a finding that challenges the validity and clinical utility of these tests.

Bursae function to reduce friction between moving structures, and up to 12 bursae have been identified throughout the shoulder region. Bursae receive a rich sensory innervation from mechanoreceptors and nociceptors and have the potential to substantially contribute to shoulder pain. Substance P is one of many substances identified in the shoulder bursae that may stimulate free nerve endings and result in shoulder pain, with higher concentrations of substance P in the subacromial bursa being associated with higher levels of shoulder pain. All shoulder special tests stretch and compress multiple structures, including the bursae, and, as such, it is unlikely that orthopaedic special tests can be used to isolate a single structure.

Diagnostic ultrasound, MRI, and surgery have been used as reference standards to validate clinical orthopaedic tests. An essential criterion for validity is...
that structural failure, seen on imaging, is present in those with symptoms, and not present in those without. Of concern, the authors of several studies have reported the presence of substantial shoulder tissue structural abnormality in people without shoulder symptoms.25-29 In an MRI investigation, Frost et al30 reported that 55% of people diagnosed with subacromial impingement syndrome had evidence of supraspinatus tendon pathology, compared with 52% in people without symptoms, with the incidence increasing equally in both groups with advancing age. Asymptomatic partial- and full-thickness RC tears have been reported in 50% of people in their seventh decade and in 80% of people over 80 years of age.81 In a separate study using MRI, a very high incidence of RC pathology (79% for the pitching shoulder and 86% for the catching shoulder) was reported in asymptomatic professional baseball pitchers.82 In a recent study, 96% of men without shoulder symptoms were reported to have some form of structural abnormality identified on ultrasound, including subacromial bursal thickening, supraspinatus tendinosis, and supraspinatus tears.83 It is apparent that the presence of structural tissue failure in large numbers of people without symptoms challenges the validity of imaging to identify the source of symptoms. This also includes intrasurgical observation of tissue failure, which has been considered by some to be the gold standard comparator to determine the validity of clinical tests.79 These data highlight the fact that many people undergo surgery on shoulder tissue(s) that potentially may not be the cause of their symptoms.

A consequence of the difficulty in deriving a definitive structural diagnosis from clinical tests and imaging procedures has prompted some individuals to advocate treatment-direction (also known as treatment classification) tests to guide patient management.115 One method, the SSMP, is clinically meaningful, and the SSMP relies on the patient to make that determination. Patients report what is important to them, such as improvement in movement/function, less pain, reduced paresthesia, or a greater feeling of stability. If an individual expresses that any alteration in symptoms is definitely what alteration in symptoms are altered. The first stage of the SSMP is to identify relevant (typically 1-3) aggravating movements, activities, or postures that reproduce symptoms. Then, as detailed in the APPENDIX, a systematic and step-wise algorithm is applied to the aggravating movements, activities, or postures to determine if the symptoms are altered and to what extent. It is difficult to state definitely what alteration in symptoms is clinically meaningful, and the SSMP aims to guide the thorax, and hold this position while repeating the movement of shoulder abduction through that painful arc. For simple activities, thoracic extension is achieved by asking the patient to place a finger on the sternum and use athletic tape is used in an attempt to hold the thoracic spine into extension.

If the thoracic maneuver reduces the symptoms by 100%, then the assessment is complete and treatment is initiated with a combination of postural aware-
reposition the scapula prior to the initiation or facilitate scapular movement, but to described. The aim of the scapular procedures can be performed the movement is relatively simple, these procedures are assessed. Again, if symptom changes secondary to scapular procedures may help to resolve thoracic and scapular procedures, humeral head procedures aim to assess symptoms potently related to a lack of anterior or posterior shoulder stability. As with the thoracic and scapular procedures, humeral head procedures that meaningfully improve symptoms are used to guide treatment. In many instances, combinations of thoracic, scapular, and humeral head procedures may help to resolve symptoms.

FIGURE 2. Symptomatic shoulder flexion is first performed in the patient's natural posture. Then, symptomatic shoulder flexion is repeated after the scapula is passively placed in 1 of 3 movement planes (elevation/depression, protraction/retraction, anterior/posterior tilt) or combinations of movement planes. The scapula is allowed to move and return to its "new" starting position. Scapular movement is not facilitated or restricted.

FIGURE 3. With more demanding movements, such as throwing and swimming, assessment of the influence of scapular position on the painful movement is achieved using taping or specially designed neoprene belts. In this instance, throwing is repeated after the scapula is taped into elevation.

FIGURE 4. Humeral head procedures aim to assess the effect of changing the relationship between the humeral head and glenoid fossa. In this instance, the shoulder is positioned in a short lever just below the point of symptoms. The individual is requested to gently push the elbow to the ground, 3 isometric contractions of 5 to 6 seconds in duration are performed, and then the arm is gently passively lowered and the symptomatic movement is retested.

If the thoracic procedures do not or only partially alleviate symptoms, then symptom changes secondary to scapular procedures are assessed. Again, if the movement is relatively simple, these scapular procedures can be performed manually (FIGURE 2). If the activity is more demanding, where manual stabilization would not be possible, then athletic tape may be used with the goal to change the scapular position (FIGURE 3). At present, the most effective approach to determine how best to assess the influence of the scapula on presenting symptoms is not known, with facilitation, taping, and repositioning techniques having been described. The aim of the scapular procedures used in the SSMP is not to assist or facilitate scapular movement, but to reposition the scapula prior to the initiation of movement. This is achieved by the therapist gently placing the scapula into a new starting position in 1 of 3 movement planes and combinations of these planes, and allowing the scapula to move from this new starting position without assistance. By doing this, the clinician can learn if 1 position or a combination of positions reduces symptoms. For example, positioning the scapula in a position of elevation and posterior tilt and allowing the scapula to then move actively from this position may be beneficial for one individual; for another, it may be beneficial to start from a more retracted position, and for another to start from a more depressed and posteriorly tilted position. In others, scapular reposition does not change symptoms and would then not be included in management.

Consistent improvement in symptoms that occur as a result of adjusting the starting scapular posture informs management of the condition, and treatment consisting of exercise and manual therapy can be initiated. The aim of this treatment is to change the motor control of the scapula consistent with the assessment findings during the provocative movement to reduce or alleviate symptoms.

If the scapular procedures do not fully alleviate symptoms, the clinician progresses to assess the effects of the humeral head procedures. The purpose of the procedures is to positively influence the patient's symptoms by applying techniques that aim to depress, elevate, or anteriorly or posteriorly glide the humeral head. Two examples are detailed in FIGURES 4 and 5. FIGURE 6 shows other procedures used to assess symptoms potentially related to a lack of anterior or posterior shoulder stability. As with the thoracic and scapular procedures, humeral head procedures that meaningfully improve symptoms are used to guide treatment. In many instances, combinations of thoracic, scapular, and humeral head procedures may help to resolve symptoms (FIGURE 7).

If the first 3 stages of the SSMP do not completely alleviate or reduce symptoms, then the final stage of the SSMP (neurmodulation) involves assessing the influence of manual procedures, such as pressure-based procedures (aimed at the soft tissues and joints), that may modulate shoulder symptoms and are routinely performed throughout the cervical, thoracic, and shoulder regions.
The SSMP is designed to influence symptoms felt in the shoulder from a multitude of potential sources and for a multitude of potential reasons, some of which may be due to RC tendinopathy. If a component or combination of components of the SSMP reduces or alleviates symptoms, then the technique found to be beneficial (ie, reduce symptoms) during the assessment process informs clinical management and forms part of the treatment. The SSMP cannot determine if the changes in symptoms produced when assessing the thoracic, scapular, or humeral head positions are the primary cause of the patient’s presenting symptoms. The SSMP may also modify symptoms that are attributable to RC tendinopathy (and from other sources as well), but we currently are unable to determine the exact mechanism or source of symptoms. Potential mechanisms whereby the SSMP may reduce symptoms include alterations in scapular position, changes in length-tension relationships of RC muscle and tendons, increasing space under the acromion, stabilizing or optimizing the position of the humeral head or the scapula, or neuromodulation of the sensation of pain.

The reliability, validity, and prognostic value of procedures such as those used with the SSMP need to be determined. At this time, their purpose is to bridge the gap in knowledge in current clinical practice imposed by the uncertainty of the source of symptoms until robust imaging and clinical diagnostic methods of identifying the location and cause of symptoms become available.

If clinical evidence suggests that RC pathology is present, such as a history of increased loading and the presence of pain and weakness principally identified during shoulder external rotation and elevation (suggestive of involvement of the supraspinatus, infraspinatus, and teres minor), and the SSMP does not fully alleviate the symptoms, a graduated RC rehabilitation program is added to the components of the SSMP that were found to be beneficial.

Rotator Cuff Treatment/Management

The primary intervention for treating RC tendinopathy is active exercise therapy. Current research evidence provides considerable confidence for people diagnosed with RC tendinopathy to expect an equivalent outcome to surgical intervention with a well-structured and graduated exercise program, with the additional generalized benefits of exercise, less sick leave, a faster return to work, and reduced health care expenses. Surgery does not confer additional benefit at 1-, 2-, or 5-year follow-up for the treatment of RC tendinopathy, and a structured exercise program significantly reduces the need for surgery. Also, surgery (acromioplasty or acromioplasty and RC repair) is not associated with an improved outcome over exercise alone for atraumatic partial-thickness tears (less than 75% thickness tear of the supraspinatus) or atraumatic full-thickness RC tears. All these studies used graduated exercise designed to target the RC musculature, with the number of formal treatment sessions ranging from 6 to 19. Some studies also included motor control exercises, scapular exercises, and shoulder stretches as part of the plan of care.

Although active exercise is the most valuable type of treatment for RC tendinopathy, many exercise strategies have been proposed, and uncertainty as to the most effective exercises persists. Despite varied rationales being proposed to justify these different exercise strategies, a number of common guiding principles emerge to direct the implementation of exercise therapy to address pain, weakness, and loss of normal function associated with this condition.

Shoulder range of motion and muscle function may improve when pain is reduced, and therefore strategies to reduce pain are a common feature of exercise programs for the treatment of RC tendinopathy. These strategies commonly include relative rest, which consists of advice/strategies to reduce/modify affected-limb activities to avoid...
pain exacerbation. It is conceivable that the average of 6 to 12 weeks of relative rest following subacromial decompression surgery for RC tendinopathy and the subsequent graduated rehabilitation may be the mechanism of benefit from surgery, and not the operative procedure itself.

The restoration of normal shoulder movement patterns is another common primary aim of RC tendinopathy exercise programs, whether they are designed to address degenerated tendons, altered scapular kinematics, or abnormal neuromuscular control. Performing the exercise therapy in a controlled and graduated manner is emphasized, whether the aim is to exercise an underloaded RC tendon to improve motor control by gradually increasing the complexity of the exercises, or to achieve conscious scapular control before progressing to scapular- and RC-strengthening exercises.

As with other musculoskeletal conditions, individuals diagnosed with RC tendinopathy can be clinically classified into irritable and nonirritable presentations. It is important to implement a structured exercise program appropriate for the presenting clinical symptoms. Guiding principles for the spectrum of RC tendinopathies are relative rest, modification of painful activities, an exercise strategy that does not exacerbate pain over time, controlled reloading, gradual progression from simple to complex shoulder movements, and, ideally, prevention of future recurrence.

Pain reduction is a priority in managing irritable RC tendinopathy. People with irritable RC tendinopathy commonly report combinations of constant pain, night pain, and persistent pain following minimal activity that continues for a protracted period of time. It is important to advise people with irritable RC tendinopathy to restrict activities of the affected limb to avoid exacerbation of their symptoms (relative rest). In addition, any exercise program should be carefully planned and delivered so as not to exacerbate symptoms. This may include devising exercises that support the arm, are performed slowly, and are also typically performed through a reduced shoulder range of motion. The reasons for the irritability are uncertain, but may suggest local bursal involvement. With an unexplained high level of pain, especially at rest, sinister pathologies must be excluded.

There is some evidence to suggest that sustained isometric contractions performed in the direction of the pain and weakness may help control pain. If a combination of relative rest, isometric exercises, and carefully graduated rehabilitation is not helpful in reducing symptoms, then injection therapy with the goal to control pain and reduce potential inflammation may be considered. In the United Kingdom, physical therapists have been performing joint and soft tissue injections to support clinical practice since the 1990s, and, more recently, physical therapists have started to perform ultrasound-guided procedures. Ultrasound-guided injections can target the subacromial bursa, and evidence (albeit equivocal) suggests that an intrabursal injection may lead to a more effective clinical outcome.
addition, both glucocorticoids and analgesics have been shown to reduce the tenocyte proliferation,65,66,68 that may be a feature of irritable RC tendinopathy.66

As corticosteroids have been associated with reduced RC tissue strength in rats and potential tendon apoptosis,23 and as research studies have not demonstrated differences between corticosteroid and analgesic subacromial injections,2,3,33 it may be clinically more appropriate, when considering injection therapy, to initially try subacromial analgesic injections followed by graduated rehabilitation.

Once the irritability has settled, or if the initial presentation was one of low irritability of the condition, then the graduated rehabilitation program progresses. Shoulder rotation exercises are commonly employed to treat RC tendinopathy. Although these are frequently performed with the arm by the side, evidence derived from electromyographic studies indicates that the RC muscles can be recruited in a more specific manner when rotation is performed with the arm in 90° of abduction.9,27 If exercise in an elevated position
is not initially possible due to symptom exacerbation, then an aim of management is to progress to these ranges. As the RC functions to counterbalance potential destabilizing humeral head translation forces generated by muscles producing shoulder abduction, flexion, and extension, exercises incorporating these shoulder movements will preferentially target

### TABLE 2

**Detailed Components for a Rotator Cuff Tendinopathy Exercise Program**

<table>
<thead>
<tr>
<th>Irritable RC Tendinopathy</th>
<th>Mechanical Nonirritable RC Tendinopathy</th>
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<tr>
<td><strong>Treatment Program</strong></td>
<td><strong>Exercise Options</strong></td>
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<tr>
<td>Graduated shoulder flexion</td>
<td>Flexion in low (&lt;60°) ROM</td>
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<td>program</td>
<td>Flexion with short lever to end ROM</td>
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<td>Full ROM flexion with long lever</td>
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<td>Shoulder ER program</td>
<td>ER with arm by side, in sitting and in sidelying</td>
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<td>ER in supported abduction/flexion, in sitting (full ROM), in prone (inner ER ROM), and in supine (outer ER ROM)</td>
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<td>ER in unsupported abduction/flexion, in standing, in prone, and in supine</td>
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<td>Full rotation ROM in unsupported abduction/flexion in prone/supine</td>
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Table continues on page 932.
Detailed Components for a Rotator Cuff Tendinopathy Exercise Program (Continued)

**TABLE 1**

| Phase 1. Supine, facilitated shoulder flexion to 90°, followed by incremental increases in shoulder flexion and extension (unloaded and loaded) to 120°. | Phase 2. 45° supported arm, inclinor assisted shoulder flexion to 90°, followed by incremental increases in shoulder flexion and extension (unloaded and loaded) to 120°. | Phase 3. Calf-dragging, inclinor assisted shoulder flexion to 90°, followed by incremental increases in shoulder flexion and extension (unloaded and loaded) to 120°. |

**Abbreviations:** ER, external rotation; RC, rotator cuff; ROM, range of movement.

**TABLE 2**

<table>
<thead>
<tr>
<th>Components of the Program (Continued)</th>
<th>Exercise Description</th>
<th>Notes</th>
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<tbody>
<tr>
<td>RC muscle contraction, but also to gradually increase the load and complexity of the exercise to facilitate progression and aid the patient in achieving the goals of the rehabilitation program.</td>
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REFERENCES


35. Frost P, Andersen JH, Lundorf E. Is supraspinatus pathology as defined by magnetic reso-


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### APPENDIX

**SHOULDER SYMPTOM MODIFICATION PROCEDURE V4, IN WHICH POSITIONING OF THE THORAX, SCAPULA, AND HUMERAL HEAD IS SYSTEMATICALLY TESTED SEQUENTIALLY**

**Shoulder Symptom Modification Procedure (SSMP) v4**

[www.LondonShoulderClinic.com](http://www.LondonShoulderClinic.com)

<table>
<thead>
<tr>
<th>Patient's name:</th>
<th>D.o.B:</th>
<th>Date:</th>
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#### 1. Thoracic kyphosis

<table>
<thead>
<tr>
<th>Position</th>
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<tbody>
<tr>
<td>Thoracic extension</td>
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<tr>
<td>Thoracic flexion</td>
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<tr>
<td>Taping</td>
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<tr>
<td>Other</td>
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#### 2a. Scapular position

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<td>Elevation</td>
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<tr>
<td>Depression</td>
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<tr>
<td>Protraction</td>
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<tr>
<td>Retraction</td>
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<tr>
<td>Anterior tilt</td>
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<td>Posterior tilt</td>
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<tr>
<td>Combination</td>
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#### 2b. Winging scapula

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#### 3. Humeral head procedures

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<td>Depression [flexion] - standing / sitting</td>
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<td>Depression /abduction - standing / sitting</td>
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<td>Depression [abduction] - supine</td>
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<td>Depression [abduction] - axillary</td>
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<td>Assisted elevation-flexion</td>
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#### 4. Symptom neuromodulation

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#### Clinical reasoning and management plan

**Abbreviations:** P = pain, W = week, S = slight, SH = shoulder, +ve = positive, -ve = negative / absent, pl = patient, I = increase, D = decrease