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The role of scapular dyskinesis on rotator cuff tears: a narrative review of the current knowledge

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- Scapular dyskinesis can be present in healthy individuals as in patients with shoulder pathology.
- Altered patterns of scapular kinematics can cause or exacerbate rotator cuff tear pathology. However, more research is needed.
- Regardless of the cause or the consequence of rotator cuff tear, scapular dyskinesis impairs shoulder function, worsens the symptoms, and compromises the success of clinical intervention.
- The available literature suggests physical therapy as the first treatment for degenerative cuff tears, and scapular dyskinesis should be addressed if present. Non-responsive cases or traumatic tears may require surgery.
- Postsurgical physical therapy protocols after rotator cuff repair must consider scapular dyskinesia to improve the outcomes.

Introduction

Scapular dyskinesis (SD) is described as any alteration on scapular position or motion, and can impair shoulder stability and function.[1,2] SD can be found in healthy individuals,[2] but statics revealed a higher prevalence in overhead (61%) than in non-overhead athletes (33%).[3] Increased SD prevalence is also found in elderly individuals, in whom overuse injuries and degenerative pathology of the shoulder are more frequent. Rotator cuff (RC) disorders are a subject of great relevance in clinical practice as they are a relevant cause of shoulder pain and loss of function that is potentially treatable.[4] In spite of being both frequent, there is a lack of studies on the association between rotator cuff tears (RCT) and SD. It is still unclear whether SD is

the cause, consequence, or a compensatory mechanism of the RC lesion.

To review the current knowledge of SD, plus the clinical and treatment implications on RCT, we performed an electronic database search using PubMed, Web of Science, and Scopus of studies published in the last five years, using various combinations of the search terms: "shoulder", "kinematics", "scapular dyskinesis", "rotator cuff tear", "treatment" and "outcome". Studies that were more relevant to the subject were retrieved and their bibliographies searched by hand. The search was limited to articles in English.

Scapular dyskinesis

Normal scapular motion

The normal three-dimensional (3D) kinematic pattern of the scapula (relative to the thorax) during arm elevation is upward rotation, posterior tilting, and external rotation.[5] The coordination of shoulder joints depends on the pattern of muscular activation. The scapula must be dynamically stabilized in a retracted position during movement of the arm to an optimal activation of the periscapular muscles, and the main scapular stabilizers are the upper and lower portions of the trapezius and serratus anterior muscles.[6]

Definition

SD is defined by the altered rest position and dysfunctional motion of the scapula. It is best seen as an impairment of optimal shoulder function, rather than pathology, as it can be present in asymptomatic individuals.[2]

Aetiology

The aetiology of SD can be neurological, such as cervical radiculopathy, long thoracic palsy, which can lead to serratus anterior weakness, or spinal accessory nerve palsy, which impairs trapezius muscle function.[7,8] There are also

musculoskeletal aetiologies, such as tightness of the pectoralis minor and biceps short head, posterior shoulder inflexibility, periscapular muscle lesions, muscular activation alterations and strength imbalances, clavicle fracture, and acromioclavicular and glenohumeral joint instability. Posture abnormalities, such as thoracic kyphosis, can also be related to SD.[7,8]

Clinical evaluation and diagnosis

SD is a clinical diagnosis. An adequate physical examination must be performed to assess all possible contributors to dyskinesis.[9] However, currently, there is no standardized method to diagnose SD.[10] Clinical examination is preferred to static imaging techniques to diagnose SD, due to its important dynamic component.[8] In the presence of suggestive symptoms, computed tomography or magnetic resonance imaging scans can help find an aetiological diagnosis of SD.[8]

There is a four-type classification for SD, based on the aspect of the medial border of the scapula with the arm at rest or during arm motion in forward flexion.[1] In type IV, both position and scapular motion are normal and symmetrical, while the other three types represent dyskinetic patterns.[1,2] Type I is characterized by the prominence of the inferomedial angle of the scapula due to abnormal posterior tilt. In type II, an entire medial border is prominent due to excessive external rotation, and type III shows prominence of the superomedial border, with upward migration of the scapula.[1,2] These patterns represent the net effect of different aetiologies and often require the countervailing mechanisms of the surrounding musculature to achieve the desired function, predisposing them to injury.[11] Type III is often associated with subacromial impingement and injuries to cuff tendons.[2] More than one type can coexist, making clinical evaluation challenging. The yes/no method is used as a simple screening tool with a sensitivity of 74–78% and categorizes the three abnormal patterns into the 'yes' category, and type IV into the 'no' category.[12,13]

The four-type classification, the yes/no method, or other visual assessment criteria have low interrater reliability.[10] Therefore, several dynamic corrective manoeuvres, such as the Scapular Assistance Test (SAT) and Scapular Retraction Test (SRT), have been introduced to evaluate the effect of the correction of dyskinesis on shoulder symptoms during arm elevation.[10] In the SAT, the clinician manually stabilizes the scapula and assists its upward rotation. The scapular contribution to impingement and RC strength is assessed, as it mimics the coupling activity of the serratus anterior and lower trapezius muscles. If this manoeuvre leads to improved range of motion or pain reduction, it is considered positive, and the patient can benefit from the rehabilitation of the scapular stabilizing muscles.[6,10] The SRT requires manual stabilization of the scapula in a retracted position on the thorax during arm elevation. This manoeuvre will grant a stable origin of the RC. SRT is considered positive if the pain decreases or the strength improves and may point out internal impingement, which can occur in association with a labral lesion.[6,10]

The Shoulder Symptom Modification Procedure (SSMP), described by Lewis,[14] can help to assess shoulder pathology. The SSMP can also guide clinical decisions as it determines whether the patient will respond to a rehabilitative programme. It consists of four techniques: thoracic kyphosis reduction, scapular positioning, humeral head positioning, plus pain and symptom neuromodulation. They are sequentially applied while the patient performs the activity or movement that most closely reproduces their symptoms, to identify one or a set of techniques that reduce symptoms by decreasing pain/symptoms and/or increasing movement and function.[14]

The evaluation of SD using objective quantification methods seems promising, although the current clinical use is limited. Analysis of the scapulothoracic motion can be carried out by tracking systems with reflectors or electromagnetic sensors attached to the skin or fixed to the scapula with pins.[15]

The skin motion artifact must be considered when interpreting the data. The gold standard for research purposes is the use of electromagnetic sensors fixed to the scapula and humerus with percutaneous pins. This method overcomes skin motion interference, but it is not adequate for the clinical setting as an invasive technique.[15] As a non-invasive, inexpensive, and clinically suitable alternative, Hsu et al suggest using the Kinect motion capture system to measure shoulder motion.[15]

The quantification of dyskinesis seems to have prognostic value. Cutti et al pointed out the limitation of the application of the Constant–Murley score (CMS) to assess shoulder function in the presence of SD and suggested a modified version, namely the Scapula-Weighted CMS (SW-CMS).[16] To avoid misclassification of those patients, the SW-CMS weights the points attributed to the humerothoracic elevation, according to the affected-to-contralateral side difference in scapula–humeral coordination.

Implications for shoulder pathology

The scapula is a critical connecting element of the kinetic chain that allows the correct transfer of strength from the body core to the arm.[1] Even if initially asymptomatic, SD considerably raises the risk of developing shoulder pain.[17] The altered scapular motion contributes to the decrease of the subacromial space, which may precipitate the most frequent shoulder pathologies, such as RCT. It also increases the strain on the anterior glenohumeral ligaments and decreases RC strength.[7,8]

The concept of 'shoulder at risk', introduced by Burkhart et al,[18] refers to the increased predisposition to injury and impaired optimal in individuals with high functional demand and shoulder structural and kinetic chain alterations.[9,18]

SD, when symptomatic, clinically manifests as SICK syndrome, characterized by **s**capular malposition, **i**nferior medial border prominence, **c**oracoid pain and malposition, and dys**k**inesis of scapular motion.[2]

Overhead athletes displayed increased scapular upward movement during the throwing motion, which is believed to be an adaptive change to preserve subacromial space and thereby avoid RC compression.[19] Although the compensatory mechanisms keep the correct function of the shoulder in certain cases, when they are no longer effective, they can contribute to an exacerbation of the shoulder's previous condition and symptoms.[20] The disabled throwing shoulder results from the successive kinetic chain alterations that occur in response to the high demands of throwing or hitting, which can impair optimal shoulder function. SD is present in 94% of athletes with a disabled throwing shoulder and is associated with 67–100% of all shoulder injuries.[9] Therefore, a clinical evaluation of the scapula should be routinely carried out for all athletes with shoulder symptoms because early diagnosis and adequate treatment of SD help prevent shoulder lesions.[7]

Another adaptative process that can occur in throwing shoulders and which must be considered is glenohumeral internal rotation deficit (GIRD). However, little evidence relating to a clear association between GIRD and scapular dysfunction is available.[21]

Scapular dyskinesis in patients with rotator cuff tears

RCT is age-related, with a prevalence of 22% at the age of 65 years, increasing to more than 62% in the population older than 80 years, resulting from normal progressive tendon degeneration.[22] RC lesions are also a common cause of pain and dysfunction in athletes across all sports, being particularly frequent in overhead and contact athletes.[23]

Two main factors may explain the negative impact of SD and RCT on shoulder function: pain avoidance and structural damage.[24] The scapula's kinematics and muscular activation pattern changes can result from the acquired adaptive, or compensatory mechanisms in the context of an RC injury and associated pain.[16]

Patients with RCT and/or SD frequently claim shoulder pain and inability to lift or perform overhead work. Shoulder pain may influence shoulder function. In this context, pain models are considered valuable tools to clarify the effect of pain on global shoulder function.[25] The mechanisms responsible for scapular alterations that coexist in RCT remain to be clarified.

Scapular kinematics changes

Previous studies on RC tendinopathy characterized SD as protraction, excessive internal rotation, anterior tilting, and increased or decreased upward rotation of the scapula (Table 1).[5,19,26,27] Insufficient scapular upward rotation and posterior tilt limit glenohumeral joint internal rotation and contribute to impingement and RC tendinopathy.[5,19,26]

In a case-control study, Kijima et al compared 3D scapular and glenohumeral kinematics during scapular-plane abduction among patients with symptomatic or asymptomatic RCT and healthy shoulders.[28] The study showed a significantly lower posterior tilt of the scapula in the symptomatic RCT than in healthy shoulders and less humeral external rotation than healthy shoulders and asymptomatic tears (Table 1). The absence of kinematic changes among the asymptomatic and healthy shoulders compared to symptomatic ones suggests a correlation between the development of symptoms and kinematic alterations.[28]

In a systematic review, Keshavarz et al asserted that patients with full-thickness RCT had greater scapular than humeral elevation during arm elevation compared to tendinopathy or healthy groups.[29] No differences were reported for posterior tilt and protraction (Table 1). Those patients also had a lower range in flexion and less power in abduction and external rotation of the shoulder. The authors concluded that the loss of RC muscle function contributes more to SD than the associated pain.[29] Robert-Lachaine et al reported an increased scapulothoracic joint contribution on arm elevation, with an increased scapular upward rotation and posterior tilt, to compensate for the glenohumeral movement lost in RC pathology.[24] Kolk et al demonstrated that patients after a repaired RCT presented a normalization of scapulothoracic rotations toward the motion patterns found in the contralateral asymptomatic shoulder, which reinforces the deviation from normal kinematics as an adaptation in the context of the RCT suffered.[30]

Muscular activation pattern changes

Previous studies on patients with pain associated with shoulder impingement and overhead athletes with SD or RC tendinopathy have reported strength deficits and reduced electromyography (EMG) activity of the serratus anterior and delayed activity onset and activation of middle and lower trapezius relative to the upper trapezius (Table 1). Those force coupling alterations were associated with the loss of posterior tilt and upward rotation of

Table 1. Summary of the main findings

Topic	Study	Type of study (level of evidence, if available)	Study population	Methods	Results	Conclusion
Scapular kinematics	Leong et al,[5] 2017	Observational, case-control study	43 male volleyball players (17 asymptomatic and 26 with rotator cuff (RC) tendinopathy) between 18–35 years of age (mean age = 22.9 ± 3.5 years).	Vicon v-370 3-D motion analysis system and acromial marker cluster method to detect scapular motion. RC tendinopathy diagnosed by clinical tests and ultrasound (US) imaging.	Athletes with RC tendinopathy with less scapular upward rotation in the early phase of shoulder abduction from 0° to 30° vs. asymptomatic athletes (6.6 ± 2.3 vs. 8.2 ± 1.1°, p = 0.021).	Rotator cuff tendinopathy alters scapular kinematics.
Scapular kinematics	Fu et al,[19] 2020	Systematic review	9 articles included a total of 332 athletes between 18–32 years of age (mean age = 23.41 ± 2.62 years). 4 studies compared dominant (throwing) vs. non-dominant shoulders of overhead athletes; 3 studies compared overhead sports athletes vs. non-athlete controls; 4 studies compared athletes with vs. without RC tendinopathy.	PRISMA guidelines; Published articles about scapular kinematics (SK) in overhead athletes with and without RC tendinopathy. RC tendinopathy diagnosed by clinical tests and/or conventional imaging; Motion analyser or similar kinematic methods to calculate SK.	Increased scapular anterior tilting and internal rotation in the dominant shoulders vs. non-dominant shoulders of overhead athletes; Increased scapular upward rotation during arm elevation in athletes vs. non-athletes; No consensual SK pattern in athletes with RC tendinopathy when compared with healthy controls.	Scapular kinematics is changed in overhead athletes.
Scapular kinematics	Keshavarz et al,[29] 2017	Systematic review	20 articles included, of which 2 systematic reviews on RC tears (RCT) (a total of 116 subjects between 30–74 years of age (mean age = 48± 8.69 years)).	PRISMA guidelines; Published articles about SK in patients with shoulder musculoskeletal disorders (SMD). SMD confirmed by clinical examinations; Motion analyser or similar kinematic methods to study SK.	Patients with RCT had scapula moving more than the humerus during arm elevation vs. healthy or patients with tendinopathy; Scapular elevation during arm elevation in patients with RCT vs. healthy and patients with tendinosis. No differences were reported for posterior tilt and protraction.	The RC pathology affects the shoulder rhythm during arm elevation in scapular and sagittal planes. Increased scapular upward rotation and posterior tilt compensate glenohumeral movements in RCT. No clear causal relationship between scapular alteration and a specific pathology.
Scapular kinematics	Kijima et al,[28] 2015	Observational, case-control study (Basic science study, kinesiology)	7 healthy subjects between 55–65 years of age (mean age = 62 years), 5 symptomatic RCT between 66–74 years of age (mean age = 70 years), and 7 asymptomatic RCT between 62–72 years of age (mean age = 67 years).	RCT were confirmed by magnetic resonance imaging (MRI); 3D/2D model-image registration techniques: shoulder computed tomography derived 3D bone models and fluoroscopic images of scapular-plane abduction.	Less posterior tilt of the scapula in the symptomatic RCT vs. healthy shoulders (3.1° ± 1.8° vs. 10.4° ± 0.8°, p = 0.049); Less humeral external rotation relative to the scapula during activity in the symptomatic RCT vs. healthy shoulders and asymptomatic RCT (p = 0.006 and p = 0.028, respectively); No differences between the asymptomatic RCT and healthy shoulders.	Possible association between kinematic changes and the development of symptoms in RCT.
Muscular activation pattern	Leong et al,[5] 2017	Observational, case-control study	43 male volleyball players (17 asymptomatic and 26 with RC tendinopathy) between 18–35 years of age (mean age = 22.9 ± 3.5 years).	Electromyography to access the activity onset of the upper (UT), middle (MT), lower trapezius (LT), and serratus anterior (SA), during arm abduction.	Delayed activity onset of LT relative to UT (14.1 ± 31.4 ms vs. 74.4 ± 45.1 ms, p < 0.001) and SA (44.9 ± 26.0 ms vs. 23.0 ± 25.2 ms, p < 0.001) in the tendinopathy vs. the asymptomatic group. Patients with RC tendinopathy with a tight EMG activation in time (particularly UT, MT, and LT), which did not happen in the asymptomatic group.	The control of the scapular upward rotation is related to the activity onset of the scapular muscles in athletes. The idea of the muscular activation adaptability in RC tendinopathy deserves further investigation.
Functional limitation	Robert-Lachaine et al,[24] 2016	Observational, cohort study (Basic science study, kinesiology)	14 patients (mean age = 56.4 ± 6.3 years) with RCT in need of surgery and 14 healthy individuals (mean age = 25.2 ± 4.1 years). 2 categories of patients considered: category A for a maximal arm elevation of 85° and category B for a maximal arm elevation of 40°.	RCT diagnosed by shoulder US or MRI; Optoelectronic system to measure the scapulohumeral rhythm; DASH [1] and WORC [2] questionnaires to evaluate subjective shoulder symptoms.	Category A patients showed inferior scapulohumeral rhythm (p = 0.032); Category B patients' scapulohumeral rhythm increased more during arm elevation vs. the healthy individuals (p = 0.044).	Pathologies affecting the RC perturb shoulder joint coordination; A decrease in glenohumeral motion is compensated by an increase of the scapulothoracic joint contribution.
Functional limitation	Hsu et al,[15] 2018	Retrospective cohort study (Level III prognostic study)	12 healthy subjects between 26–62 years of age (mean age = 34.6 years), and 352 patients with shoulder pathology before elective surgery.	The Kinect system to assess active scapulothoracic (ST) and humerothoracic (HT) abduction; Simple shoulder test (SST) for patient self-assessment of shoulder function.	Limitation of ST abduction in patients with glenohumeral pathology unable to perform standardized shoulder functions (12 ± 10° or 17% of the active HT abduction) vs. healthy shoulders (26 ± 7° or 19% of the active HT abduction); Patients with self-assessed loss of shoulder function with decreased ST and HT abduction (p < 0.001).	ST motion is an important component of active shoulder motion and function in both healthy and pathological shoulders.

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Topic	Study	Type of study (level of evidence, if available)	Study population	Methods	Results	Conclusion
Functional limitation	Baumer et al,[34] 2016	Controlled laboratory study	25 patients between 48–79 years of age (mean age = 60.2 ± 8.4 years) with symptomatic RCT, and 25 asymptomatic control subjects between 51–74 years of age (mean age = 59.0 ± 5.5 years).	RCT documented by shoulder US or MRI; Shoulder motion measured by a biplane radiography imaging system; Strength assessed by a Biodex dynamometer; Patient-reported outcomes assessed by WORC questionnaire and visual analogue scale (VAS) pain score.	Patients with symptomatic RCT with worse pain/function scores, less range of motion (ROM), lower abduction, external and internal rotation, strength (p < 0.01), less scapulothoracic posterior tilt (p = 0.05), and lower glenohumeral joint elevation (p < 0.01) vs. control subjects.	Symptomatic full-thickness RCT impairs shoulder function.
Retear risk	Reuther et al,[42] 2015	Randomized controlled trial (Basic science, in vivo animal study)	A rat model of scapular dyskinesis (SD) was used. 70 adult male Sprague-Dawley rats (400–450 g) were randomized into 2 groups: nerve transection of the accessory and long-thoracic nerves or sham nerve transection (control).	All rats underwent unilateral detachment and repair of the supraspinatus tendon and were sacrificed at 2, 4, and 8 weeks after surgery. Elastic and viscoelastic mechanical properties of the supraspinatus tendon determined by uniaxial tensile testing; cellular and organizational changes by histologic analysis and the distribution of extracellular matrix proteins by immunohistochemical techniques.	SD altered joint function and compromised supraspinatus tendon properties: it diminished mechanical properties, altered histology, and decreased tendon organization.	SD alters the shoulder loading environment (type of loading more than its amount/ magnitude) and increases the risk of re-rupture of the healing supraspinatus tendon following repair. The functional consequences associated with SD may compromise supraspinatus tendon healing following repair by diminishing some tendon mechanical properties.
Conservative treatment	Baumer et al,[34] 2016	Controlled laboratory study	25 patients between 48–79 years of age (mean age = 60.2 ± 8.4 years) with symptomatic RCT, and 25 asymptomatic control subjects between 51–74 years of age (mean age = 59.0 ± 5.5 years).	RCT documented by shoulder US or MRI; Shoulder motion measured by a biplane radiography imaging system; Strength assessed by a Biodex dynamometer; Patient-reported outcomes assessed by WORC questionnaire and VAS pain score. A standardized physical therapy (PT) protocol was prescribed for patients with symptomatic RCT. Patients' data acquired before and after 8 weeks of PT and acquired at 1 time point for the control subjects.	PT improved pain/function scores (p < 0.01), increased ROM (p < 0.02), increased scapulothoracic posterior tilt (p = 0.05), increased glenohumeral joint elevation (p = 0.01), and decreased acromiohumeral distance (p = 0.02).	PT has a positive effect on clinical outcomes in patients with RC pathology.
Conservative treatment	Giuseppe et al,[8] 2020	Systematic review	127 articles included.	PRISMA guidelines; Published articles about SD, its causes and effects, clinical examination, and treatments.	Specific exercises for scapular rehabilitation include exercises to optimize scapular kinematics: flexibility exercises to decrease traction and neutralize scapular positions, and scapular stabilization exercises, based on stretching and strengthening scapular stabilizers, to improve muscle strength and proprioception.	A large variety of SD alterations can be managed by an individualized rehabilitation protocol based on clinical and isokinetic tests. SD conservative treatment aims to restore scapular kinematics, reinforce the scapular muscles and guarantee the optimal length–tension relationship of RC muscles.
Surgical treatment	Ueda et al,[27] 2019	Observational, case-control study	14 healthy controls (mean age = 24.7 ± 4.5 years) and 16 patients with RCT surgically treated: 10 patients with small RCT (mean age = 62.7 ± 7.7 years) and 6 with massive* RCT (mean age = 64.5 ± 9.5 years). *Massive tear was defined as 'the conditions that L × H is > 5.6 cm² (where L is the length of the tear region at the attachment site of the tendon and H is the depth to the tendon end; >= 2 tears are present and the diameter of exposed humeral head is > 3 cm or the circumstance of the ruptured region is > 9 cm)'.	All RCT treated by McLaughlin procedure. Fluoroscopic imaging to analyse scapular motion. RCT patients' motion analyses were performed in the preoperational stage, and at 2 and 5 months after surgery.	Before surgery: Both rotator cuff groups with greater scapular upward rotation vs. healthy controls (p < 0.01); No significant difference was observed between the 2 RCT groups (p = 0.17). At 2 months after surgery: significant greater scapular upward rotation only in patients with small RCT vs. healthy controls at arm elevations of 90° (40.7° ± 5.5° vs. 34.2° ± 4.0 °, p = 0.02); but for both RCT groups (small, massive RCT) vs. healthy controls for arm elevations of 120° (61.2°, 61.3° vs. 48.2°, p < 0.01). At 5 months after surgery: Significant differences still existed in healthy controls vs. both rotator cuff groups.	Tear size affects scapular motion: massive RCT are associated with greater scapular upward rotation. The rehabilitation programme should consider the tear size.

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					For the patients with small tears, but not for massive RCT, the scapular upward rotation decreased over time (2–5 months) at arm elevations of 120° (from 61.2° to 52.4°, p = 0.01), but did not return to the same level of the healthy controls (48.2°).	
Surgical treatment	Kolk et al,[30] 2016	Observational case series (Level IV)	26 patients with RCT proposed to surgical treatment, between 46–73 years of age (mean age = 60 years); The asymptomatic contralateral shoulder was used as the control.	RCT were confirmed by magnetic resonance imaging or computed tomography; Shoulder kinematics was analysed before and 1 year after RC repair by 3D electro-magnetic motion analysis (Flock of Birds).	Surgery improved mean arm abduction by 20° (118° ± 37.3° vs. 138° ± 20.0°, p = 0.025) and forward flexion by 13° (127° ± 31.4° vs. 140° ± 15.6°, p = 0.044) and decreased mean scapular protraction and lateral rotation during abduction by 3° (95% CI, 0.0°–5.2°; p = 0.046) and 4° (95% CI, 1.6°–8.4°; p = 0.042), respectively. Glenohumeral elevation increased by 5° (95% CI, 0.6°–9.7°; p = 0.028) at 80° to 90° of abduction, after RC repair. Humeral ROM increased when scapular lateral rotation decreased, and posterior tilt increased. No differences in scapular motion before vs. after surgery, in the asymptomatic contralateral shoulders.	Scapular kinematics normalize after RC repair toward the scapular motion patterns observed in the asymptomatic contralateral shoulder. Changes in scapular kinematics after surgery are associated with an increased overall ROM, suggesting the restored function of shoulder muscles.

1- The Disabilities of the Arm, Shoulder and Hand (DASH) score

2- The Western Ontario Rotator Cuff (WORC) index

the scapula.[5,7,19] SD can be associated with the inhibitory effect that RCT-related pain has on the individual muscle activation capacity, which disrupts normal activation patterns. Therefore, dyskinesia may also arise in RCT due to movement-related pain and avoidance.[7] It is also acceptable that SD can appear like an adaptation mechanism after RCT, acting as a compensatory strategy to optimize the elevation of the injured arm, with a weakened or absent RC activation.[7] Studies on electromyographic activity of the scapular muscles during shoulder abduction showed an early muscular activation in athletes without RC tendinopathy and deficits in those with RC tendinopathy (Table 1).[5] The early muscular activation may arise from the need to stabilize the scapula and retain a stable base for the scapulohumeral muscles during arm movements in overhead athletes during the increased demands of the sport. All muscles became simultaneously activated on EMG to stabilize the scapula in patients with tendinopathy, but not in asymptomatic ones.[5]

Treatment of patients with rotator cuff tear and scapular dyskinesis

RCT include a wide range of lesions. The therapeutic approach for RCT must consider the pathogenesis, tear morphology, clinical symptoms, patient's functional demands, and presence of concomitant pathology.[23,31]

Conservative treatment

The current evidence recommends initial physical therapy for symptomatic degenerative RCT.[31] However, specific exercises for the ageing population are not yet established, and it is controversial whether they should promote the loading or unloading of the RC muscles. For athletes with RCT, the non-surgical therapeutic approach is based primarily on the interruption of all throwing activities. Therapy with non-steroidal anti-inflammatory drugs and a rehabilitation programme can also be considered.[23,31,32] After the reduction of shoulder symptoms, more sport-specific training programmes can be implemented.[23]

In a recent review, Weiss et al contended that the RC injury rehabilitation programme should be gradual, with different phases that will complement each other to best prepare the athlete for the return to competition.[23] This programme included early rehabilitation, an intermediate phase, and advanced/late-stage rehabilitation. It aimed to progressively reduce symptoms, restore the normal range of motion, gain flexibility, enhance proximal stability, and work on proprioception.[23] The entire kinetic chain should be strengthened, and disturbances which might be causing patient symptoms, such as soft tissue tightness, postural abnormalities, or SD, should be addressed.[31]

Regarding SD, the best current treatment is also the conservative one: physical therapy to correct positional and muscular force abnormalities, with specific exercises

for scapular stabilization and muscular strengthening to optimize scapular kinematics (Table 1).[11,17]

Scapular posture abnormalities can be addressed with exercises that increase muscle flexibility.[8] Shoulder strengthening exercises change the activation patterns of the scapulothoracic muscles, decrease pain and improve shoulder function. Initial treatment should focus on the proximal kinetic chain, with exercises for core musculature stabilization, and then distally, on the strengthening of scapular stabilizers. [11,23] Stretching and strengthening exercises improve proprioception and contribute to scapular stabilization.[8,11,33] Baumer et al demonstrated that physical therapy has a beneficial impact on clinical outcomes in patients with chronic RCT, by reducing pain, increasing the shoulder range of motion, and improving functional scores, but it had a subtle effect on glenohumeral and scapulothoracic motion patterns (Table 1).[34]

Turner suggested that treating SD in RCT might be as effective as surgery, by reporting a case of one non-throwing athlete with a traumatic RCT whose treatment protocol addressing scapular dysfunction alleviated pain and dysfunction and led to a faster return to the full level of sport, when compared to surgical interventions.[17] A meta-analysis by Saito et al showed that patients receiving scapular-focused interventions significantly improved pain (MD [95% CI] = −0.88 [−1.19 to −0.58]) and shoulder function (MD [95% CI] = −11.31 [−17.20 to −5.41]), compared to the control group. However, they did not consider those improvements clinically relevant.[26]

The appropriate timing to initiate rehabilitation is yet to be defined. Tear severity can be considered a determining factor: individualized early rehabilitation protocols may be the best approach to promote an early functional shoulder for small to medium tears, but delayed therapy is preferred for large tears in the postoperative period, to provide adequate time for tendon healing.[35]

Surgical treatment

Surgical reconstruction of RCT can effectively restore the biomechanical stability of the shoulder, normalizing scapular kinematics (Table 1).[27,30] Early surgical repair can be the therapy of choice for large traumatic RCT, particularly in young or high functional demand patients, due to the increased risk of tear progression.[36] Current clinical practice guidelines support surgery for persistent shoulder pain and dysfunction. Arthroscopic repair is generally preferred to the open surgery, but the evidence does not support a specific surgical technique over another. It depends on several factors, including surgeon experience and preference, tear and patient-specific factors.[23]

According to the population studied, the outcome differs: results are more satisfactory in the general population than in high-level athletes.[23] For elite overhead athletes, particularly those without full-thickness tears, outcomes

following surgery are not predictable.[32] The high failure of recovering and returning to the same performance level discourages surgery: more than 75% return to sport, but less than half achieve their pre-injury level.[32]

Several predictors of worse postoperative outcomes have already been identified: older age, lower baseline functional scores, increased tear size, preoperative fatty infiltration, tendon retraction, shoulder stiffness, muscle atrophy, among others.[37]

Most cuff retears are well tolerated by the patient, not requiring further intervention.[38] Desmoineaux highlights that the work-up of a failed cuff repair must slightly differ from the initial one and address all the possible diagnostic and technical failures during the first intervention, as well postsurgical conditioners, such as an inadequate rehabilitation.[38] Concomitant abnormalities, such as the pathology of the long head of the biceps or the acromioclavicular joint, should be diagnosed and treated correctly.[23,38,39]

Some patient-related factors (smoking habits, existing comorbidities, poor motivation, and adherence) are important and cannot be controlled by clinicians.[38] Those can be significant contributors to high retear rates.

The reported retear risk following RCT surgical reparation varied from 10.3% to 94%.[35,40,41] This disparity present across studies probably results from a non-uniformity regarding the retear definition, tear sizes considered, surgical and postoperative imaging techniques, and many other criteria, and reflects the multifactorial character of retearing.[35,40,41]

To study the effect of SD on supraspinatus tendon healing following repair, Reuther et al used a rat model. The evaluation focused on shoulder function, passive joint mechanics, and tendon properties. It concluded that, besides altering joint function, SD compromises supraspinatus tendon properties. SD diminishes its mechanical properties, alters histology, and decreases tendon organization (Table 1). SD overloads, compresses, and submits tendon to shear stress, affecting optimal tendon-to-bone healing.[42]

While low activity and loading promote healing, excessive or abnormal joint loading increase the risk of re-rupture.[42,43] Further research is required to identify the modifiable factors that compromise tendon healing and contribute to repair failure. SD could be one of them.

Final considerations

This review gathers relevant studies and their findings on SD and RCT. The current work identified some existing evidence gaps, which should be addressed in future studies.

The literature search was not conducted systematically; therefore, other pertinent studies may have been inadvertently omitted. The lack of studies acknowledging the simultaneous presence of RCT and SD limited this review.

More quality evidence is further needed to clarify abnormal scapular kinematics in RCT, identify the changes in scapular muscle activation patterns, and establish rehabilitation strategies to prevent and manage RC disorders when SD is clinically diagnosed.

At the time of writing, there is a predominance of reports of athletes with RCT treated by surgical intervention and postsurgical rehabilitation, clearly not applicable to the general population. This review also found conflicting data regarding the recommended conservative approach. The best conservative treatment for RCT remains unclear: randomized controlled trials and detailed exercise protocols are scarce.

Efforts should be made to study modifying factors of scapular kinematics and how they can be addressed pre, peri or postoperatively, to improve postoperative outcomes and prognosis of patients with rotator cuff tears (either diagnosed or not previously diagnosed with scapular dyskinesis).

Conclusions

SD may be the cause, the result, or the compensating mechanism of RC lesions. The clinician must consider early recognition and treatment of SD in patients with RCT to accomplish the best clinical outcomes. While conservative treatment may be sufficient for certain patients with SD and RCT, others necessarily require surgery followed by proper rehabilitative strategies.

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ICMJE CONFLICT OF INTEREST STATEMENT
The authors declare no conflict of interest relevant to this work.

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