Rotator cuff tears

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Abstract

Rotator cuff tears are most commonly the result of age-related degenerative pathology, although they can result from trauma at any age. Patients with rotator cuff tears (RCT) often present with pain, loss of strength and decreased range of motion, which significantly affects their function and quality of life. Some uncertainty persists about optimal treatment strategies and the timing of repairs for some RCTs. The incidence of rotator cuff repairs has rapidly increased in recent years, and studies show that the vast majority of patients go on to experience significant improvements in pain and function, although re-tear is a recognized complication. Novel strategies using grafts to augment repairs are being employed in the hope of reducing these re-tear rates. Augments are expensive and currently not fully supported by high-quality evidence demonstrating their efficacy. The management of larger, potentially irreparable cuff tears is challenging. It remains unclear whether surgery offers effective long-term results for most patients with large and massive cuff tears, as rotator cuff repairs for this cohort are associated with high failure rates.

Keywords double row repair; patch; rotator cuff; superior capsular reconstruction; tendon

Incidence

The ball and socket shoulder joint affords a huge degree of mobility, by partially sacrificing stability. Stability is partly offered by the four muscles that make up the rotator cuff, supraspinatus, infraspinatus, subscapularis and teres minor. The first surgical repair of a rotator cuff tear (RCT) is often accredited to Codman, who was thought to have performed the procedure in 1909, and his classic observations were described in 1934. Rotator cuff degeneration and tears pose a significant management challenge and impose a huge functional, economic and social burden. The prevalence of clinically relevant shoulder problems in the UK has been estimated at 14% in the general population, with higher estimations in several other countries such as Japan, where the prevalence is estimated at 20.7%. Some 1–2% of British adults consult their general practitioner (GP) annually with a new onset of shoulder pain, and it has been suggested that 40–50% of all patients in the UK consult their GP for shoulder pain at some stage.[1] Shoulder problems are the third commonest cause of musculoskeletal problems following spine and knee problems, and accounted for 2.4% of all GP consultations in the UK.[2] Rotator cuff tears are one of the commonest causes of shoulder pain and dysfunction. Although some cases of shoulder pain do improve with treatment and rest, 40% of patients are estimated to still have persistent or recurrent symptoms a year after presenting to their GP. Whilst there are many causes of shoulder pain, such as impingement, frozen shoulder, calcific tendonitis and osteoarthritis, this study will only focus on rotator cuff tears.

Census information from the USA predicts that 17 million people potentially face the risk of disability from rotator cuff failure.[3] Rotator cuff pathology accounts for 30–70% of shoulder problems, a significant proportion of this is attributable to RCTs. Cadaveric studies have estimated that the prevalence of rotator cuff tears in adults is 5–30% and MRI and ultrasound studies have estimated similar prevalence rates of 15–23% for asymptomatic shoulders in the general populations, with the prevalence increasing with age. For example, the rate of tears in symptomatic shoulders has been estimated as 20–30% at age 60–70 and up to 62% over 80 years.

As RCTs increase with age, particularly over the age of 50 years, an ageing population means the socioeconomic burden of this problem will only increase. The incidence of RCTs is increased by the presence of co-morbidities such as rheumatoid arthritis. Up to 50% of patients with rheumatoid arthritis over the age of 50 years are estimated to have at least one RCT. Tears can impair the ability to work or perform household tasks and result in increased time off from work. The intra-articular synovial environment of the damaged tendon often precludes normal healing and surgical repair may be required.

Over 9000 rotator cuff repairs are undertaken annually in the UK and more than 400,000 surgical repairs are performed annually in the USA for rotator cuff pathologies.[4] This procedure places a significant financial burden on the NHS as each operation reportedly costs over £6600 and total costs exceed £60 million in England. It is difficult to accurately quantify the full economic cost of rotator cuff disease to the NHS in the UK. The annual financial burden of rotator cuff surgeries in the USA has been estimated to be $3 billion, including treatment, evaluation and management.[4]

Types of rotator cuff tear

A continuing spectrum of disease has been proposed for rotator cuff tendons, ranging from tendinopathy to full thickness massive tears. Importantly, preoperative tear size seems ineffective as a sole indicator to predict clinical and functional recovery following surgery.

Full thickness rotator cuff tears

RCTs refer to full thickness disruption of at least one of the four muscles that form the rotator cuff and help to stabilize the

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shoulder. A classification system of full thickness tears based upon tear size has been proposed by Post et al., who classified tears as small (<1 cm), medium (<3 cm), large (<5 cm) and massive (>5 cm) according to the greatest diameter of the tear. Rotator cuff tears are proposed to be part of a spectrum or cascade of disease ranging from reversible tendonitis irreversible tendonitis, partial tears, small through to massive full thickness tears and finally cuff tear arthropathy. Each of these stages can involve calcific tendonitis, but this is often transient and is outside of the scope of this article.

Partial thickness rotator cuff tears

Any tears that involve rotator cuff fibre disruption but do not extend all the way through the cuff are termed partial thickness tears (PTTs). Ellman has proposed a classification system for this group of tears which describes three features: location (articular, bursal or interstitial), tear area (measured in mm^2) and grade (grade 1 < 3 mm, grade 2 is 3–6 mm, and grade 3 is >6 mm deep).

Cuff tear arthropathy

Neer coined this term in 1983 to describe severe rotator cuff tendon insufficiency associated with arthritic changes of the glenohumeral joint, typically with superior migration of the humeral head.

Biomechanics of rotator cuff tears

A number of dynamic and passive stabilizers help to stabilize the inherently unstable glenohumeral joint. The four rotator cuff muscles (supraspinatus, subscapularis, infraspinatus and teres minor) contribute to stability through force couples in both the coronal and transverse planes. Supraspinatus and deltoid equally contribute to abduction, wherein the resultant joint reaction force compresses the humeral head against the glenoid in the coronal plane and aids concentric rotation of the humeral head.

Supraspinatus tears results in loss of key humeral head depression forces in the coronal plane. However, if the subscapularis tendons anteriorly and the posterior cuff tendons (infraspinatus and teres minor) remain intact, then the anterior-posterior transverse force couples are balanced around the glenohumeral joint. This acts as the primary mechanism to resist superior head migration and is referred to as a 'balanced tear'.

Burkhart popularized the concept of the rotator crescent and cable, explaining how the location of tears is a key determinant of likelihood of propagation and whether shoulder biomechanics are significantly altered. The rotator crescent describes a thin, crescent-shaped sheet of rotator cuff, which comprises of the relatively avascular distal portions of the supraspinatus and infraspinatus insertions. This crescent is proximally and medially bounded by a semi-circular thickening of the glenohumeral capsule referred to as the rotator cable. This cable–crescent configuration spans between the tubercle insertions of supraspinatus and infraspinatus tendons, anchoring the tendons to the tubercles and acting like a suspension bridge. The rotator cable is believed to provide stress-shielding of the rotator crescent by transferring stress when loaded. Thus, tears disrupting this suspensory mechanism are believed to be more likely to be symptomatic and result in greater weakness, particularly as it can disrupt nearby structures such as the long head of biceps and the coracohumeral ligament.

Aetiology of rotator cuff failure

Tendinopathy describes tendon injury that results in pain or tearing of the RCT, whereas tendonitis describes tendon inflammation, and microtearing of the tendon is termed tendinosis and is a histological diagnosis.

RCTs can be classified according to their presumed aetiology, as being either acutely traumatic or chronic degenerative; extrinsic or intrinsic; mechanical or biochemical; or based upon anatomy as articular, bursal or interstitial. Acutely traumatic tears are presumed to occur in tendons without evidence of pathological abnormalities, wherein excessive force exceeds the tendons capacity to withstand and it tears. There is widespread literature about rotator cuff tendon failure associated with sport, which is assumed to be due to overuse in activities such as swimming, throwing and overhead activities etc. Chronic degenerative tendons are more commonly seen and are assumed to occur as a result of a combination of the changes of normal ageing in conjunction with some abnormal active pathological processes.

A more common classification for the pathogenesis of rotator cuff disease involves extrinsic (extratendinous) factors such as impingement beneath the coracoacromial arch, and intrinsic (intratendinous) degeneration, which may or may not be related to ischaemia and usually worsens with age.

Biomechanical changes in rotator cuff tears

Mechanical factors have been shown to contribute to tendon tears, such as excessive loading, which can damage the tendon body and result in inflammation of the tendon sheath. A large Japanese cross-sectional study identified a history of trauma as a risk factor for RCTs.[5] The tensile strength of tendons is a function of thickness and collagen content. Once the tendon is stretched beyond its elastic threshold, then failure may ensue with subsequent inflammation of the tendon sheath and/or tendon degeneration. Tendon damage can also occur when microtraumatic forces are applied within the tendon's physiological threshold but the normal reparative mechanisms are overwhelmed. Overuse has been suggested as a contributing factor to the development of RCTs, as tears are often more symptomatic in the dominant rather than the non-dominant arm. Overuse cannot be used as a sole explanatory factor, as most tears (more than 70% estimated by one study) occur in sedentary individuals who only do light work.

Supraspinatus is the most frequently torn rotator cuff muscle. Extrinsic causes of rotator cuff tendon failure were proposed by Neer in 1972, who suggested that more than 95% of rotator cuff tears occur when mechanical contact occurs between the rotator cuff tendons and the overlying coracoacromial arch. Neer described three progressive stages of impingement: (1) oedema and haemorrhage, (2) fibrosis and tendonitis and (3) rotator cuff and biceps tendon tears with acromial bony spurs. Two-dimensional finite element models have suggested that subacromial impingement generates high stress concentrations in and around the critical zone. A correlation between RCTs and associated impingement due to the acromial shape has been proposed by Bigliani et al.[6] He suggested that tears are contributed to by 'spurs' at the antero-inferior margin of the acromion, particularly a hook-shaped acromion rather than a flat shape, as well as changes in the coracoid and coracoacromial ligament. The supraspinatus outlet can be reduced by thickening of the

coracoacromial ligament. It has been proposed that different glenoid and humeral versions can act via by both an extrinsic mechanism, by narrowing the subacromial space, and contribute to intrinsic degeneration by increasing the shear stress on the rotator cuff.

Extrinsic causes cannot explain all rotator cuff tendinopathies and tears. It has been proposed that rather than being a primary cause for rotator cuff tendon failure, impingement occurs secondary to intrinsic rotator cuff tendon failure, which impairs tendon function and results in superior migration of the humeral head, narrowing of the subacromial space and impingement against the acromion and coracoacromial ligament. The relationship between RCTs and impingement was also studied by Ozaki et al. who examined 200 cadaveric shoulders and found pathological changes in the acromial undersurface with bursal tears rather than articular tears.[7] They therefore hypothesized that articular tears occurred due to a degenerative process.

The effects of RCTs on muscle moment arms have been simulated using finite element modelling. It has been proposed that torn tendons are associated with reduced moment arms when compared to intact tendons, which reduces the effectiveness of muscles. An MRI study found that the glenoid version of rotator cuff tears was significantly different compared to controls, with retroversion being predictive of anterior cuff tears and anteversion being predictive of posterior cuff tears.[8] Full thickness RCTs have been shown to have an association with increased glenoid inclination angle.

RCTs are prone to heal with the formation of scar tissue at the tendon-bone interface, thus this regenerated tissue is usually weaker and more prone to fail. Tissue near the tendon-bone attachment, or enthesis, has been shown to have a low degree of fibre alignment. Itoi et al. suggested that supraspinatus tears are associated with decreased strength.[9] Following artificially created tears in human cadaveric supraspinatus tendons, Reilly et al. demonstrated alterations in strain.[10] A small study looking at 20 patients with partial and full thickness supraspinatus tears compared to their normal contralateral shoulder, found that after intrarticular or intrabursal local anaesthetic blocks, there was a significant increase in strength in abduction and external rotation. It may be argued that the decreased strength in torn tendons is due to pain rather than an inability to transmit mechanical load. Following RCTs, it was found that the remaining intact tendons had decreased mechanical properties with a decreased modulus but increased cross-sectional areas.[11] A study looking at the morphology of RCTs found that full thickness tendon tears had a significantly decreased muscles fibre length and increased functional tendon length (extramuscular tendon length plus tear length) when compared to PTTs and normal tendons.[12]

There is some evidence to suggest that different sized RCTs may have different mechanical properties. Rokito et al. found a trend between tear size and the recovery of shoulder strength after repair, with large and massive tears being weaker[13] and taking longer to regain strength compared to small and massive tears.

Intrinsic changes of rotator cuff failure

Codman proposed in 1934 that intrinsic mechanisms related to inherent tissue changes within the tendon played a significant role in the aetiology of rotator cuff tendon tears. The biology of tendons is important, and this theory is supported by the fact that the incidence of tears increases with age, and poorer outcomes are seen following smoking. Nobuhara et al. found that 97% of 189 repairs performed for massive tears were in patients aged 45 years and above, and 81% of patient tendon biopsies showed degeneration.[14] Degenerative changes are likely to play a key role, as a large cadaveric study found these changes in 97% of spontaneously ruptured tendon tears ranging from the Achilles to biceps brachii, but also in 33% of normal tendons.[15] As degeneration was found to increase with age, distinguishing pathological degeneration from normal physiological age-related degeneration is complex, but the levels of degeneration in normal and ruptured tendons have been shown to be different, even in advanced age groups. Degenerative changes of the rotator cuff tendon are likely to affect mechanical properties, as the increasing degeneration has been found to result in decreasing ultimate tensile stress. The role of biological factors in the aetiology of RCT is further supported by evidence that some patients have a genetic predisposition to the development of RCT pathologies and to earlier disease progression.

Alterations in the biochemical environment of torn tendons have been demonstrated. Factors implicated in the pathophysiology of RCTs include apoptosis genes, cytokines, oxygen free radicals and growth factors such as transforming growth factor-β and matrix metalloproteinases.

Changes in cellularity

There is increasing evidence to support the idea of a progressive multistage disease process where small tears have different characteristics to larger tears. Recent evidence suggests that both the cellular and extracellular matrix composition of the torn tendon edge change significantly as the tear size increases. Matthews et al. reported that, compared to normal tendons, small RCTs had an increase in the numbers of inflammatory cells and fibroblasts, which then decreased as tear sized increased.[16] Rats subjected to overuse activities were found to have hyperplasia and hypertrophy of fibroblasts.[17] Cells within the compressive regions of rotator cuff tendons have been identified which synthesize aggrecan and type II collagen, and as such are interpreted as signs of chondrogenesis. Chondroplasia has been proposed as an adaptation to hypoxia secondary to compressive loading or overuse. The exact mechanism is unknown, but a higher incidence of chondroplasia has been detected in the articular side of rotator cuff tendon tears, in areas of few fibroblasts and larger (medium to massive) tears.

Metabolic changes

It has been suggested that tendon tears occur when the healing mechanisms within tendons are overwhelmed. In addition, tissue metabolism, and therefore possibly the viability of tendons, is believed to diminish as tear size increases. It is known that tenocytes from diseased or overused RCTs demonstrate features of programmed cell death or apoptosis. Multi-stage disease models have been proposed. The apoptotic process is believed to progress as tear size increases and may be induced by hypoxia. Torn rotator cuffs express increased levels of the DNA (deoxyribonucleic acid) repair enzyme Apr/Ref-1, which is thought to signify attempted repair of hypoxia induced DNA or mitochondrial damage.

Extracellular matrix changes

Extracellular matrix changes in torn rotator cuff tendons have been demonstrated previously, including increased collagen III, reduced collagen I, altered metalloproteinases and an accumulation of glycosaminoglycans and lipids. Interestingly the total collagen content has not been found to change with age but does decrease with degeneration. Torn tendons have been shown to have altered chemical compositions with varying levels of proteoglycans, glycoproteins, cytokines, oxygen free radicals, atrophy and chondrocyte markers. Remodelling of the tendon extracellular matrix has been demonstrated in response to load, ageing and injury. Altered levels of matrix metalloproteinases (MMPs) and tissue inhibitors of matrix metalloproteinases (TIMPs) have been demonstrated in torn tendons.

Supraspinatus tendinopathy has been shown to result in MMP-3 (stromelysin-1) mRNA expression, and both a decrease and increase in MMP-2 (gelatinase A). Inhibition of matrix metalloproteinases has been shown to improve histological healing, but interestingly there was no difference in mechanical properties.[18] Tear propagation may be encouraged by the presence of fewer cells at the edges of tears, which have an altered metabolism and are less able to synthesize and degrade the extracellular matrix to repair the structural damage.

Supraspinatus tendons usually have higher rates of collagen turnover than other tendons, such as biceps tendons, so any disruption in the turnover is likely to affect the collagen and proteoglycan content. Increased amyloid deposition has been reported in chronic RCTs compared to acute tears, although this may be confounded by the reported correlation with increasing age. Mucoid and lipoid degeneration have been reported in tendons, with accumulation of mucoid patches and vacuoles, and lipid accumulation respectively. Muscle atrophy and fatty infiltration into the muscle have also been demonstrated.

However, neither the extrinsic nor intrinsic theories of aetiology can fully explain RCTs. Belief in the extrinsic theory alone has been largely superseded as tears can occur without acromial or other mechanical stresses. It is therefore likely that RCTs are contributed to by a complex interaction of intrinsic changes resulting in extrinsic contributions. At present the aetiology and progression of RCTs are not fully understood, which may explain why there is currently no definitive or curative treatment.

Vascular changes

Codman proposed that the perfusion of rotator cuff tendons played a significant role in the development of tendon pathology. A few studies have suggested that the supraspinatus tendon has reduced vascularity close to its bony insertion. An avascular zone has been reported approximately 1 cm from the supraspinatus insertion, and this area has been termed the ‘critical zone’ and represents an area of vessel anastomoses. It has been observed that tendon degeneration and rupture often occur at watershed areas, and thus reduced vascularity has been proposed as an important predisposing factor. The supraspinatus tendon most commonly ruptures at or near its insertion into the greater tuberosity, at the so-called critical zone. This under-perfused area was found to be more evident on the articular side of the tendon. The area of avascularity has been proposed to increase with age. A review of randomized clinical trials comparing the use of topical nitroglycerin (GTN) patches to rehabilitation for the treatment of chronic supraspinatus tendinopathy found improved outcomes in terms of patient-reported pain, increased tendon force measures, functional measures and patient outcome.[19]

Imaging of tears

Plain radiographs of the shoulder are a useful first-line investigation, as they can help to identify bony abnormalities, arthritic and calcific changes as well as tumours. X-rays have limited diagnostic accuracy for soft tissue conditions such as RCTs. Ultrasound is a cheap and rapid modality, which is increasingly performed in outpatient clinics by either radiologists or trained surgeons as part of a ‘one stop’ clinic. Due to its high accuracy, ultrasound is useful in the detection of many soft tissue pathologies of the shoulder. CT scans have limitations in the detection of soft tissue lesions, particularly PTTs, resulting in less frequent employment of this technique. High accuracy in the detection of RCTs has been demonstrated with CT arthrography (CTA). MRI is considered by many as the gold standard investigation of choice for rotator cuff lesions, and some studies have suggested that MRI is a more accurate diagnostic tool than ultrasound. However, a meta-analysis of studies revealed that MRI and ultrasound have comparable sensitivity and specificity for diagnosing both full and partial thickness RCTs.[20] The same analysis found that MRI arthrography (MRA) had greater sensitivity and specificity for the detection of rotator cuff tears when compared to ultrasound and MRI. MRA is particularly accurate for diagnosing certain conditions such as PTTs. The disadvantages of MRA are the additional time, the expertise required, cost and the need for an intra-articular injection with its associated risk of joint infection.

Goutallier demonstrated that fatty degeneration of rotator cuff muscles, particularly infraspinatus or the subscapularis muscles, is associated with less favourable outcomes after repair.[21] The classification was initially developed using CT scan results, although it is also applied to MRI scans. This classification assumes the muscle disuse due to a tear of the associated tendon results in fatty infiltration of the muscle belly.

Management of tears

Uncertainty about optimal management strategies and timings persist. Four of the top ten priorities related to shoulder problems identified by the James Lind Alliance were related to RCT management. This priority setting partnership involved patients, carers and clinicians. Two of the most specific questions regarding RCTs were the need for more information about whether (a) it is possible to predict who will do well with surgery to help patients decide about having surgery and (b) whether surgery or physiotherapy was the best treatment. The management of RCTs can be broadly divided into three categories: non-operative treatment, primary repairs and augmented repairs.

Conservative treatment

Regardless of the size of the tear, some cases can be effectively treated with a trial of a combination of advice, exercises, simple analgesics and anti-inflammatory medication as some patients are able to ‘compensate’ with good active shoulder function, despite an RCT. Most physiotherapy exercises are aimed at strengthening rotator cuff muscles and retraining deltoid. A trial comparing a

single session of best practice physiotherapy to progressive physiotherapy found no significant difference at 1 year.[22] More severe or persistent cases may require physiotherapy or glucocorticoid injections (with or without local anaesthetic). These interventions have variable outcomes in the literature ranging from excellent to no better than placebo. Evidence for long-lasting benefit from these treatments is weak and repeated use of injections may be harmful.

The evidence for the effectiveness of common conservative treatment strategies, such as oral non-steroidal anti-inflammatory drugs (NSAIDs) and glucocorticoid injections, is weak and addresses the pain and inflammation rather than reversing the underlying pathophysiology. A Cochrane review of steroid injections found that subacromial glucocorticoid injections showed short term benefits over a placebo in some trials, particularly for improving abduction.[23] Glucocorticoid injections were not found to have any significant advantages over oral NSAIDs. A randomized clinical trial found that subacromial steroid injections for rotator cuff disease did not provide significant symptomatic relief at 1 year.[22] Repetitive injections may be inappropriate as there is an association with cell death, inhibition of tenocyte migration and proliferation of synovial fibroblast proliferation as well as slowing of healing, which may impair repair. It has been suggested that corticosteroid injections may be detrimental to tendon strength, as they damage collagen ultra-structure, causing increased collagen disorientation and weaken collagen fibres, which ultimately increases the likelihood of rupturing. There have been reported cases of tendon rupture, subacromial joint infections and deleterious effects on glenohumeral cartilage following glucocorticoid injections.

An alternative approach to glenohumeral or subacromial steroid and local anaesthetic injections are treatments that target the suprascapular nerve, with the aim of denervation and ultimately reducing pain. Treatments can either be suprascapular injections, thermal or radiofrequency ablation.

Surgical management of tears

The intra-articular environment of the damaged tendon often precludes normal healing. If conservative strategies fail after a 3–6-month period then surgery may be considered to restore stability and improve pain and function. Indications for surgery usually involve the sudden onset of severe symptoms and pathology or a combination of failed conservative care, persistent or worsening pain and functional disruption. The first rotator cuff repair was performed in 1909. Notwithstanding developments in techniques and repair materials, current surgical treatment strategies are often unsuccessful in the long term. Despite high failure rates following surgery, the number of rotator cuff repairs undertaken globally has rapidly increased. Currently a variety of surgical techniques are undertaken to manage RCTs such as arthroscopic repair, mini-open repair, open repair (all of which can be done with or without a subacromial decompression) or less commonly with a biceps tenodesis, debridement, tendon transfer or synthetic grafts. Different repair techniques include a single row medially, a single row laterally, a double row of suture anchors and a double-row transosseous equivalent technique. For both partial thickness and massive tears debridement is sometimes performed. The type of surgery employed will depend not only upon the diagnosis but also upon the patient profile. Rotator cuff repairs are not without risks and have a number of known complications such as recurrence or failure to heal, infection, stiffness, persistent pain and weakness, neurovascular damage and chondrolysis.

Whilst repair of RCTs is usually advocated for painful tears with functional impairment, some uncertainty exists as to exactly when to operate and what features should guide this decision.

Many symptomatic tears respond to non-surgical management and some patients with tears demonstrate marked improvement following subacromial decompression and rotator cuff debridement without cuff repair. A randomized clinical trial of patients with rotator cuff disease found that patients who underwent both non-operative and operative treatment had significantly improved pain and function. However, patients who underwent surgery had a significantly greater improvement compared to non-operative management.[24] The 10-year data from a Canadian randomized trial demonstrated significant improvement in clinical outcomes and function.[25] A randomized clinical trial of open versus arthroscopic repair (UKUFF trial), concluded that there was no significant difference in outcomes when comparing open and arthroscopic rotator cuff repairs. Rotator cuff repairs are reported to be a high-value and cost-effective treatment, which provides a sustainable and notable positive improvement. A further argument in support of repairing tears is that without surgical intervention there is a significant likelihood of tear progression and subacromial decompression alone does not reduce this risk.

Of concern is that a large proportion of current surgical treatment strategies of suture repair strategies, including tendon grafts or synthetic grafts, are often unsuccessful with high reported re-rupture or failure to heal rates of between 13 and 94%. The UKUFF trial revealed that 40% of repairs fail within 12 months irrespective of surgical technique. However even patients with a retear significantly improved compared to baseline levels. In cases of a massive tear where a complete repair is not possible, various strategies including tendon transfers and incorporation of the biceps tendon have been employed. In the absence of any controlled data it is unclear how effective these approaches are.

Re-rupture has serious clinical consequences and has been shown to correlate with poorer outcomes. Larger tears and increasing age are associated with a higher incidence of rerupture, irrespective of the surgical technique employed. Other factors which have been suggested to influence surgical outcomes include fatty infiltration of the cuff, and co-morbidities such as smoking and time elapsed before surgery. RCTs associated with systemic diseases such as rheumatoid arthritis are an additional surgical challenge.

What is surprising is that despite failure of the surgical repair, most patients report improved pain levels and satisfaction postoperatively. However, patients whose repairs remain intact do relatively better in terms of function and strength but differences in terms of pain scores may be less apparent compared to the reruptured group.

Management of partial thickness rotator cuff tears

Management of PTTs is a controversial subject without widespread consensus, despite their prevalence. Classically patients were treated conservatively, and if this failed there was a progression to arthroscopic debridement with or without

subacromial decompression. There is some uncertainty regarding the risk of progression to full thickness tears. Cordasco et al.[26] demonstrated that lesions involving less than 50% of the thickness of the cuff did progress in the long term. If symptoms fail to settle with conservative measurements then subacromial decompression may be beneficial. Some favourable results have been reported following debridement of PTT in association with subacromial decompression as well as following open repair of PTTs involving >50% of the tendon but most studies lack controls. PTTs can be repaired in-situ or completed to a full thickness tear prior to undertaking a repair. Repair of partial thickness are also associated with high failure rates.

Management of full thickness tears

Rotator cuff repairs can be repaired utilizing single or double row techniques aimed at restoring the rotator cuff footprint without excessive strangulation or undue tension. A single row of anchors uses sutures passed through the rotator cuff to compress the tendon onto the tuberosity with low tension. This method is relatively simple, quick, cheap, has a lower risk of damaging tendon margins, shorter learning curve and is easier to revise. Double row repairs utilize medial and lateral row anchors, which can include a suture bridge technique (transosseous equivalent). Medial row suture anchors are placed adjacent to articular margin while lateral row suture anchors are typically placed along the lateral side of the greater tuberosity. Double row repairs are believed to be more effective at increasing the tendon–bone insertion contact area, reconstituting a more anatomic rotator cuff footprint configuration to allow for cuff healing. Although biomechanical and in-vivo studies have demonstrated improved mechanical properties for double row compared to single row techniques, this has not reliably translated to superior clinical outcomes or lower rerupture rates. One randomized controlled trial comparing 10-year follow-up data for single versus double row repairs did not find a consistent difference in outcomes, however double row repairs were less likely to see a functional decline between 2 years and 10 years.[25] Repairs can be aided by techniques such as margin convergence or interval slides, which may help to aid mobilization and reduce the tension on the repaired tendon.

Management of massive, potentially irreparable rotator cuff tears

Massive RCTs are often technically challenging, as traditional suture repair strategies cannot achieve adequate re-approximation of the tendons without undue tension. The term typically refers to tears greater than 5 cm, which often include both supraspinatus and infraspinatus. Tears are deemed irreparable when it is impossible to connect the tendon back to its insertion footprint without an unacceptable degree of tension, or poor tendon quality which prevents purchase of a suture.

Due to clinical ambiguity, there is widespread variation in the management of large and massive rotator cuff tears. Many surgeons would undertake some form of attempted surgical repair for large to massive tears, with most opting for a direct repair. A smaller but rapidly rising group of surgeons are utilizing patches to augment surgical repairs of large and massive tears. A survey of surgeon members of the British Elbow and Shoulder Society revealed that 58% had used a patch to augment rotator cuff repairs.[27] About 28–40% and 19–59% of surgeons stated they would use patches for large or massive tears respectively. In the presence of widespread fatty infiltration or atrophy, most surgeons would probably choose non-operative treatment with focused physiotherapy.

Debridement or partial repair: debridement and subacromial decompression have been reported to improve pain, function and patient satisfaction. Alternatively, a partial repair (predominantly of subscapularis and the posterior cuff muscles) may be undertaken to try to restore the anterior-posterior force couples. Simply attempting a primary repair may be effective, even if the repair fails. Some uncertainty exists in the literature regarding the need for a structurally intact repair to achieve significant clinical improvement. Despite studies suggesting poorer outcomes following failed rather than intact repairs, the UKUFF trial demonstrated that patients with failed repairs significantly improved compared to their baseline level.

Graft augmentation: augmentation grafts aim to enhance the biological milieu for rotator cuff healing. Augmented repairs can broadly be considered together despite variation in types of augmentation grafts currently utilized and fixation modalities. Four broad groups exist:

1. Xenograft (decellularized animal extracellular matrix)
2. Allografts (decellularized human cadaveric tissue)
3. Synthetic (bio-inert polymers)
4. Autografts (patient's own harvested tendons).

Grafts are either secured over the repair (onlay) or fill the tendon defect (bridging). A systematic review and meta-analysis reported that several studies showed decreased failure rates and small improvements in shoulder function and pain, but there was a paucity of rigorous evaluation of effectiveness or safety.[28]

Superior capsular reconstruction: one increasingly popular bridging technique is superior capsular reconstruction, (SCR). Early biomechanical work by Mihata et al. demonstrated that reconstruction of the capsule with fascia lata grafts was found to significantly reduce superior translation of the humeral head and purportedly acts as a passive constraint. The technique has largely evolved to utilize a dermal decellularized allograft, eliminating the potential donor site morbidity associated with the fascia lata graft harvest techniques. Ideally this technique is best suited to cases with an intact infraspinatus and subscapularis, without arthritis. While reported to provide at least short-term pain relief and improved strength and function, this is a technically challenging procedure with a learning curve and there is limited evidence about the long-term outcomes. UK National Institute for Health and Care (NICE) guidelines found limited quality and quantity of evidence to support SCR augmentations for massive RCTs. Despite the lack of any level 1 studies evaluating its efficacy or the fact that most are only level 4 studies, the number of procedures undertaken is rapidly expanding at a somewhat concerning rate. This relatively new technique has sparked a great interest and resulted in a 300% increase in publications between 2016 and 2018. Most of these studies are low quality and small series with a median number of 19 patients.

Reverse shoulder arthroplasty: reverse shoulder arthroplasty is another option for management of massive tears, particularly

when associated with cuff tear arthropathy, particularly for elderly, low-demand patients. The biomechanical principles of a reverse shoulder arthroplasty rely upon the deltoid and do not require a functioning rotator cuff, especially supraspinatus, although better function and internal rotation is achieved if subscapularis remains intact. Most outcomes reported include small cohorts with inconsistent results, varying patient satisfaction levels and relatively high complication rates.

Tendon transfers: management of massive cuff tears in younger patients is especially challenging. One approach is to consider tendon transfers to try to restore force couple balance around the shoulders. For posterior-superior RCTs, latissimus dorsi transfers have traditionally been the transfer of choice since described by Gerber. The aim is to provide an interior force to counterbalance the deltoid and intact subscapularis, while acting as an external rotator and humeral head depressor. However, a lower trapezius transfer is more anatomic and is being increasingly utilized when indicated. Options for anterior-superior RCTs include pectoralis major, with some latissimus dorsi transfers also undertaken for subscapularis deficiency. Tendon transfers are reported to provide good pain relief and reasonably good function, although outcomes are reliant upon careful donor tendon selection and careful application of tendon transfer principles.

Subacromial balloon spacers: implantable biodegradable subacromial balloon spacers were introduced in 2012 with a view to acting as a humeral head depressor. This is often used as a bridging technique for patients with irreparable cuff tears where delayed reverse shoulder replacement is being considered, although the utilization of this technique remains relatively limited to date. In some cases balloon spacers have been inserted in conjunction with a SCR. NICE guidelines found that there was limited evidence for its use and as a consequence, two randomized clinical *trials* are being undertaken to compare its efficacy to either debridement or partial repairs for the management of irreparable cuff tears. The results of these trials should guide future utilization of this technology.

Summary

RCTs are a common presentation with increasing age, which can result in significant pain and dysfunction. The number of repairs undertaken is rapidly increasing. There is good evidence that rotator cuff surgery usually improves outcomes, although it is associated with small but significant risks. The underlying degenerative and pathological tendon can make achieving a successful repair more challenging. A number of strategies are available for managing RCTs, with decisions predominantly informed by tear size, quality and patient age. Novel strategies such as grafts to augment repairs are being employed in the hope of reducing high retear rates and are awaiting high-quality evidence demonstrating efficacy. It is important to carefully examine the tear morphology and patient variables when assessing RCT management and repair potential to allow patients, clinicians and stakeholders to help optimize patient outcomes. ◆

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