Assessment and treatment strategies for rotator cuff tears

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Abstract

Tears of the rotator cuff are common and becoming an increasingly frequent problem. There is a vast amount of literature on the merits and limitations of the various methods of clinical and radiological assessment of rotator cuff tears. This is also the case with regard to treatment strategies. Certain popular beliefs and principles practiced widely and the basis upon which they are derived may be prone to inaccuracy. We provide an overview of the historical management of rotator cuff tears, as well as an explanation for how and why rotator cuff tears should be managed, and propose a structured methodology for their assessment and treatment.

Keywords

assessment, biomechanics, repair, review, rotator cuff, shoulder pain, tear, treatment

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Introduction

Degeneration and progressive mechanical failure of the rotator cuff (RC) is a normal age-related process in all populations.¹ Specific genotypes might be more susceptible to failure of the homeostasis of rotator cuff tendon matrix, which may lead to accelerated mechanical failure of specific zones of the rotator cuff tendons.^{2–4} This normal ageing phenomenon is not necessarily painful.^{5–7} The majority of rotator cuff tears (RCTs), particularly those in the elderly, are asymptomatic, suggesting that the shoulder has a high capacity for adaptation to changing biomechanics provided that the changes are slow.⁸

Shoulder pain is the third most common presentation of musculoskeletal pain in primary care after low back and neck pain.⁹ It is estimated that there are approximately 9 million people over the age of 60 years in the UK. Sixty percent are likely to have at least one RCT, of which half are partial-thickness and the other half full-thickness tears. Hand dominance, occupation and leisure activities appear to have a limited influence on the timing of onset and rate of progression of mature atraumatic tendon degeneration.¹⁰ By contrast, traumatic rotator cuff tendon rupture is always painful and often associated with temporary pseudoparalysis of the shoulder. Attributing factors

include mechanical incompetence (even in partial and incomplete tears), haemorrhage, oedema, micro-neural injury and central inhibition of muscle power as a result of pain. If an acute injury is superimposed on an existing RCT, then the pseudoparalysis may be profound and prolonged, such that biomechanical re-adaptation may not be achievable and the shoulder remains substantially incompetent.¹¹

In 1931, Ernest Codman described the rim rent (partial, articular surface) tear.^{12,13} This refers to the crescentic portion of the supraspinatus tendon distal to the cable, a thickening of capsular fibres running orthogonal to the main fibre direction of the tendon.^{14–16} It is now accepted that this pattern is the usual initiating pathology in the great majority of mature atraumatic tendon tears. Subsequent vascular studies^{17–21} demonstrated a loss of intrinsic capillary loops in the distal

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supraspinatus tendon in subjects older than 40 years in the rim rent zone. Intermittent position-dependent hypoperfusion within this 'critical region' was invoked as the primary causative pathology. Later still, Charles Neer introduced the concept of impingement as a result of extrinsic stenosis or compression of the space through which the supraspinatus tendon (SST) passes (the subacromial outlet), although he did not suggest that this was the initiating lesion of a RCT.²² His concept, used by others, came to be considered antecedent to the evolution of RCTs through attrition of the bursal surface of the SST, placing the emphasis on impingement as the principle lesion of rotator cuff disease. This has led to the concept of 'stages' of impingement, from roughening of the bursal surface of the tendon through progressive tearing up to a full-thickness tear. In turn, this led to the vast literature on impingement lesions and their treatment, which consumes huge surgical resources. Now, however, it is accepted that Codman's concept is the more helpful: the primary lesion of a mature atraumatic tendon degeneration is a partial articular surface or deep layer intrinsic delamination tear as a result of fibre failure.^{23,24} This is secondary to genetically programmed apoptosis of tenocytes and consequent degradation of quality of the supporting matrix, which makes the tendon susceptible to intrinsic shear failure.^{25–27} Failure of the musculotendinous structure is not confined to the distal tendon.^{28,29} Fatty degeneration of muscle (and attendant sarcopenia),^{30,31} longitudinal musculotendinous junction disinsertion (associated with loss of normal pennation angles³²) and failure of the enthesis (failure of elaboration of growth factors from and by bone in the tuberosities³³) are all relevant. These are essentially all perturbations of the biochemical processes of muscle/tendon/bone health and regeneration. Unless there is associated inflammation,³⁴ this complex process is painless until microneural and microvascular damage occurs. Hypoxia and resting tissue ischaemia modulate the classic symptoms of rest pain, use-dependent rotator cuff claudication and fatigue pain, perceived as a diffuse aching quality throughout the sclerotomy of the shoulder.

The finding of a RCT is therefore a matter of genetic programming and disturbed chemistry.

Size is not (Always) critical

Mechanical effects (intrinsic shear disruption) become relevant as the tendon fails, thus permitting the extension of the tear throughout the tendon structure in all axes. It is generally accepted that partial (i.e. incomplete thickness) tears become small full-thickness tears, which in turn become large full-thickness tears, although this is not universally the case. The challenge for diagnosis is not simply to determine the presence of

a tear but to be able to predict the progression of the tear to accurately prescribe relevant treatment. Here, the concept of the fibrous endoskeleton (FE) of the rotator cuff is helpful (Figure 1).³⁵ Muscle fibres of the supra-equatorial rotator cuff insert into the tendon structure as described by Gagey et al.;³⁵ only the most caudal muscle fibres of the subscapularis and teres minor insert directly into bone. The fibrous framework comprises the strong radial cables of the supraspinatus tendon (forming the anterior pillar or lateral pulley), the subscapularis (forming the medial pulley, with important contribution from the coracohumeral and superior glenohumeral ligaments in the lateral part of the rotator interval), the infraspinatus (contributing to a posterior pillar) and the transverse fibre bundles forming the lateral-placed so-called cable.^{36,37} Within this framework, the long head of biceps tendon (LHBT) passes to the sulcus: at the transition from intra-articular to extra-articular portions, there is a vascular watershed.³⁸ Because the intrinsic tendon vessels are accompanied by sympathetic nerve fibres,³⁹ damage to this portion of the LHBT is almost always accompanied by characteristic pain,^{40,41} often localized to the sulcus region during motion but often diffuse in the anterior compartment of the arm at rest. The close anatomical-mechanical relationship between the strong cord of the LHBT and the two pillars of the lateral aperture of the rotator interval during respective rotation of the humeral head⁴² suggests a potential feedback mechanism involving afferent information from the LHBT (under tensile load and working with the pillars) complementing that of the distal cuff tendons in determining the joint position and motion detection during the wind-up, cocking and follow-through phases of a throwing action.⁴³ The fibrous endoskeleton therefore provides a framework for muscular attachment; a linkage between the front and back of the rotator cuff, to 'co-ordinate', in a mechanical sense, the activity of the cuff across the supra-equatorial regions of the humeral head; and contributes to the effectiveness of afferent feedback from the cuff tendons for optimal cuff activation.⁴⁴ The implication of these observations is that specific, relatively well-circumscribed points of attachment of the rotator cuff tendons matter to stability of the humeral head on the glenoid rather than the entire tendon attachment. Clearly, pain is generated by a failing tendon and so treatment of the pain of a rotator cuff lesion has to reflect the potential of the entire tendon to heal. It remains the case that not all tendons or parts of all tendons need repair or reconstruction for stability (the ultimate goal of treatment other than pain relief) to be achieved.

The nomenclature of RCT size as small, medium, large and massive is popular.^{45,46} It is not particularly helpful in defining treatment strategies but is

Figure 1. (a) The normal cuff fibrous 'endoskeleton': the rotator cuff muscles act through their attachments to the fibrous endoskeleton, not only through their bony 'footprint' or attachment. a, cranial border of subscapularis tendon; b, anterior pillar or border of supraspinatus; c, posterior pillar (anterior part of infraspinatus tendon). Lhb, tendon of the long head of biceps. (b) The common 'crescent tear' or 'rim rent' of the ageing cuff. The shoulder remains stable because the fibrous endoskeleton remains attached. Pain recedes after a short period of 'pseudoparalysis' and function is restored but with decreased stamina for reaching in or forwards of the scapular plane. Surgery is not always necessary: persistent (>3/12) or changing pain, with instability suggests extension of the tear, particularly through the anterior pillar, and surgery should be considered. (c) The predominantly unstable cuff lesion(s). The fibrous endoskeleton is not attached (at one-or-more points): the cable is slack and retracts medially. The Lhb hypertrophies over time: if the tear evolves slowly, then Lhb function can substitute for the loss of the supraspinatus until the Lhb also degenerates. The rotator interval (RI) widens, allowing the humeral head to 'escape' anterosuperiorly: the Lhb dislocates (usually anteriorly) and is often the most prominent pain generator. Lhb tenotomy or tenodesis may now be useful. (d) The predominantly weak cuff lesion. The tear extends posteriorly, through the posterior pillar. The shoulder is stable during forward elevation (because the anterior pillar remains intact), although there is (occult) postero-superior subacromial 'escape' causing posterolateral acromial pain, with weak external rotation. (e) The so-called 'massive' rotator cuff tear. All supra-equatorial rotator cuff elements are absent: the shoulder is unstable, weak, painful (generated by the superior compartment, the suprascapular nerve traction lesion, the evolving Lhb lesion, and evolving joint surface roughness with synovitis). All components can be addressed arthroscopically until arthritis creates unremediable stiffness: reverse-polarity joint replacement is then indicated.

used extensively to discuss the various merits of fixation devices and techniques for rotator cuff surgery. Definition of these categories is generally on the basis of measured size, and there is indifferent correlation between size as gauged on imaging and size measured intra-operatively. This is particularly true of arthroscopic assessment when tears often appear smaller

than at open procedures. Anteroposterior dimension, the extent of mediolateral retraction, the number of tendons involved and the configuration of the tear are all qualities that have been used to define the class of tear.⁴⁷⁻⁵¹ The literature remains inconsistent.

The difficulty is that some so-called massive tears are asymptomatic and do not require surgical

treatment,^{52–54} whereas most partial-thickness (small and/or evolving) tears, particularly those involving the attachment points of the FE, are very painful indeed and do require careful analysis and treatment (which may involve surgery). ‘Small’ tears can be much more demanding for the patient than ‘massive’ tears. Pain and tear dimension are therefore not helpful discriminators when deciding options for functional restoration in a cuff-deficient shoulder.

For the RCT, ‘size’ is therefore not critical, although ‘position’ is.

The spectrum of presentation

The clinical presentation is of a spectrum of ‘weakness’ and ‘unstable motion’ of the shoulder, complicated by ‘stiffness’ and ‘roughness’, with each feature presenting with specific pain.^{55,56} Weakness occurs when either the direct muscular attachments of the anterior and posterior cuff are detached (rare), or the muscular insertion into the FE is disrupted (musculotendinous rupture/avulsion), or the FE is detached. Unstable motion of the shoulder occurs when the FE is ruptured or detached antero- or postero-superiorly. This can be compensated by the LHBT^{57–59} and, in large diameter proximal humeri, the effect of the deltoid muscle centralizing vector.⁶⁰ The large male proximal humeral geometry with an intact LHBT thus favours function in extensive RCTs (although with time can result in failure of the LHBT by rupture or displacement). Stiffness caused by synovial thickening and fibrosis, contracture of the ruptured capsule and muscular degeneration and fibrosis presents with oblige (often painful) translations. Surfaces that normally glide smoothly without restriction may be damaged and/or inflamed causing roughness. This may present with internal impingement⁶¹ (from features such as labral tears and detachments, or intra-articular buckling of the free edges of torn tendons and the obstructed biceps tendon), external impingement⁶² (e.g. superior instability of the humerus, with structural alterations of the acromion and acromioclavicular joint) or progressive degenerative or crystalline arthropathy (of the glenohumeral or acromioclavicular joints).

Following the above description, the clinical presentation can be expressed as:

$$\begin{aligned} \text{Patient - specific dysfunction} = & \\ & [\text{stiffness} + \text{weakness} + \text{failure of the fibrous} \\ & \text{endoskeleton} + \text{roughness}] - [\text{effects of the} \\ & \text{LHBT} + \text{deltoid}] \end{aligned} \quad (1)$$

Each component of the equation can be associated with pain. Therefore, the diagnosis of each pain generator in

extensive RCTs is fundamentally important in defining treatment goals and pathways. Not all shoulder pain in a patient with a RCT is of the RCT.

Weakness can only be correctly assessed if stiffness is analyzed. If the excursion of a muscle–tendon unit is limited by a checkrein (contracture), it cannot generate an appropriate force. Similarly, articular surface roughness generates pain which is inhibitory of muscle action.

Finally, the functional needs of the patient should be clearly understood. The presence of an extensive tear on an image alone is not an indication for intervention.

The rotator cuff equation

We propose the ‘rotator cuff equation’, describing a mechanistic approach to the analysis and management of RCTs:

$$\begin{aligned} \text{Diagnosis} = \text{pain} \times \{ & [\text{stiffness}^{n1} + \text{weakness}^{n2} + \text{failure} \\ & \text{of the fibrous endoskeleton}^{n3} + \text{roughness}^{n4}] \\ & - [\text{effects of LHBT}^{n5} + \text{deltoid}^{n6}] \} \end{aligned} \quad (2)$$

The factors $n1$ through $n6$ have a different patient-specific weighting, which helps guide treatment. This synthesis of symptoms and signs (the ‘syndrome’ of rotator cuff failure) helps the clinician to consider the entire patho-anatomy of RCTs and consequent treatment can be better planned.

Aids to diagnosis

Lesions of the distal tendon attachments and LHBT complex are readily accessible to ultrasound examination, with high positive and negative predictive values for full-thickness tears^{63–65} (less so for partial-thickness tears). Supplementary specific diagnostic localizing anaesthetic injections can help distinguish sources of pain, so assist the clinician to direct treatment more accurately.^{66–68} Distinguishing the sources of pain generated in the superior compartment (SST, subacromial space, acromioclavicular joint, LHBT) is certainly helped by selective, sequential localizing injections in each space. Computed tomography or magnetic resonance imaging cross-sectional scanning of the rotator cuff muscles is helpful when trying to formulate a prognosis for proposed repair.⁶⁹ If there is extensive fatty-degeneration of muscle, then the repair, even if it heals, has a high probability of failure and associated complications, particularly stiffness.^{70,71}

Cross-sectional imaging does not tell us who to operate on, when or how, although it can confirm a diagnosis, if necessary, and is useful for prognosis.

Treatment specified by patho-biomechanics

The assessment strategy described allows the clinician to separate the components of the clinical presentation, analyze the relationship between each of the components and define a patient-specific intervention. Intervention for stiffness (physiotherapy, arthroscopic release of contractures, and possible injection of hyaluronic acid analogues), roughness (arthroscopic debridement, tuberosplasty and acromioplasty) and other pain generators (subacromio-deltoid bursal injection therapy, synovectomy, acromioclavicular joint resection, LHBT tenoplasty, tenodesis and tenotomy) can all transform a decompensated shoulder into one that compensates very well for the RCT.^{72–75} These interventions are often best performed arthroscopically, and constitute a ‘conservative’ approach of RCT surgery.⁷⁶ Arthroscopic decompression or selective image-guided blockade of the suprascapular nerve and lateral pectoral nerve can be useful adjuncts in gaining pain relief, and the former can be safely repeated using pulsed-radiofrequency treatment (with variable success rates). An eccentric anterior deltoid strengthening programme can deliver a ‘stable enough, strong enough’ shoulder once a functional, painless, smooth and passive range of motion has been re-established.^{77,78}

However, RCT repair offers the best probability for pain relief in a persistently painful shoulder with failure to progress through a rehabilitation programme providing the repair does not itself generate more problems such as stiffness and roughness.⁷⁹ The detail of the repair is important: it is generally currently accepted that a double-row fixation technique onto a prepared bone surface (or ‘footprint’) provides the most durable method of re-implantation of a RCT.^{80–85} The bone must be biologically competent (i.e. it must be able to generate growth factors in a milieu of mesenchymal stem cells) and be sufficiently mechanically stable to hold the fixation devices. Whether the repair is performed arthroscopically or by open surgery is hardly relevant: the outcome (pain relief and strength) for repairs of full-thickness tears by either method is the same 6 months after surgery.^{86–90} Advocates of arthroscopic surgery rightly claim a quicker return to activities of daily function, although the failure rates of repair (overall, judged by cross-sectional imaging) are still higher in the arthroscopically treated cases. It is probably more correct to refer to failure of healing rather than failure of repair because it would appear that up to 50% of full-thickness RCTs re-tear by 1 year after surgery, representing a failure of biology rather than a failure of fixation technologies. Some of these radiological ‘failures’ are asymptomatic and are stable on testing and thus should not be classified as clinical

failures. The repaired RCT which is asymptomatic but incompletely healed behaves similarly to the incomplete RCT in a stable compensated shoulder. Partial repair is a logical intervention, especially when restoring the anchor points of the FE.^{91–93}

If weakness remains a function-limiting problem in a RCT too large or inelastic to repair, then intrinsic or extrinsic musculotendinous transfers (local rotator cuff flap or pectoralis major, latissimus dorsi and teres major flaps) are considered.^{94–99} If painful articular surface roughness persists, causing articular surface impingement, then replacement of the articular surfaces is indicated. If the shoulder is otherwise stable enough and strong enough (subscapularis-intact situation), then surface replacement arthroplasty is sufficient. If the shoulder is stable enough but weak, then surface replacement arthroplasty with extrinsic musculotendinous flaps can be valuable. If the shoulder is neither strong enough, nor stable enough, then reverse polarity or fixed-fulcrum arthroplasty can be considered.^{100,101} The better results of reverse polarity arthroplasty appear to be in patients who would have rehabilitated had there been less articular surface pain generation (i.e. the larger proximal humeral geometry, deltoid-sufficient condition).

Massive tears are generally defined as tears involving the subscapularis, as well as the two postero-superior tendons, and are characterized by painful persistent superior instability. An intact (therefore useful) LHBT should be preserved in such cases^{102–104} and, if unstable, should be stabilized in the sulcus without strangulation. The LHBT can provide a framework for the attachment of extrinsic musculotendinous transfers, or be incorporated into intra-cuff transfers for greater strength in the repair.^{105–107}

RCTs and the future

RCTs are here to stay. As we age more healthily, so the prevalence of rotator cuff disease will likely increase. It will behove us to better define the diagnosis and methods of treatments of this problem, so that personal functional independence is maintained for as long as possible. The expense of repairing and rehabilitating a RCT should be seen in the content of the social and financial burden of untreated symptomatic disease.¹⁰⁸

The efficacy of treatment is currently being examined by the UKUFF trial run by Professor Andy Carr from Oxford and involves approximately one-third of the specialist shoulder surgeons in the UK through the British Elbow and Shoulder Society. The trial aims to distinguish between rehabilitation alone, arthroscopic repair and open surgical repair in the management of well defined RCTs. The trial outcome will be important, particularly in the light of the interest of some political quarters of ‘procedures of limited value’. New

research into the biological competence of bone suggests that addressing the quality of implantation sites on the humerus (by mesenchymal stem cell seeding, or augmenting bone density with demineralized bone matrix) has a better chance of generating better surgical repair outcome than trying to further improve surgical fixation and suture technology. Epidemiological, genetic and bio-assay studies will define subgroups of patients particularly at risk of developing specific types of RCT. Patients are presenting at a younger age with partial-thickness tears of the anterior fibrous endoskeleton creating lateral instability lesions of the rotator interval. Screening shoulders in at-risk groups, possibly defined by bursal or glenohumeral fluid bio-assays, at the age of transition (good to indifferent perfusion, 40 years) might offer a way of introducing relevant preventative rehabilitation programmes, or at the least recommend optimum sports and occupational activities for concerned individuals. The future of the treatment of rotator cuff disease is in the earlier recognition and subsequent manipulation of the disease processes involved. New cross-sectional, three-dimensional molecular scanning techniques; understanding the value and control of apoptosis, restoration and maintenance of chemical homeostasis of the matrix, and anticipatory management of sarcopenia; the introduction of (preventative) percutaneous injection therapy for junctional dissociation lesions (using platelet-rich plasma, or specific transformation factors as an example); and the derivation of image and assay-guided rehabilitation programmes, as examples, may all play a role in minimizing the effect of disease in an increasingly active and economically important constituency.

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