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SHOULDER

Rotator cuff tears: pathology and repair

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Abstract By virtue of its anatomy and function, the rotator cuff is vulnerable to considerable morbidity, often necessitating surgical intervention. The factors contributing to cuff disease can be divided into those extrinsic to the rotator cuff (most notably impingement) and those intrinsic to the cuff (age-related degeneration, hypovascularity and inflammation amongst others). In an era of emerging biologic interventions, our interventions are increasingly being modulated by our understanding of these core processes, many of which remain uncertain today. When we do intervene surgically, the techniques we employ are particularly challenging in the context of the tremendous pace of advancement. Several recent studies have shown that arthroscopic repair gives similar functional results to that of mini-open and open procedures, with all the benefits of minimally invasive surgery. However, the 'best' repair construct remains unknown, with wide variations in surgeon preference. Here we present a literature review encompassing recent developments in our understanding of basic science in rotator cuff disease as well as an up-to-date evidence-based comparison of different techniques available to the surgeon for cuff repair.

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Pathology of rotator cuff tearing

Since Codman in the 1930s, many theories have been proposed to explain the etiology of rotator cuff tears. Here we cover the evidence underlying these theories, divided by tradition into those 'intrinsic' and those 'extrinsic' to the cuff.

Extrinsic factors

First proposed by Neer, impingement theory is the best known extrinsic pathologic factor in cuff disease. Neer [65] felt that repetitive translation of the cuff under the acromion led to partial tears that in turn led to full-thickness tears in a method similar to that shown in Figs. 1 and 2 and that acromial morphology was fundamental to this process. Bigliani et al. [9] divided acromions into three pathological categories: Type I—flat seen in 17% of cuff tears, Type II—curved seen in 43% of cuff tears and type III—hooked seen in 39% of cuff tears. These subtypes are a predominantly congenital trait, modulated to a small extent by tractional forces [66, 90]. Wang et al. [89] have demonstrated that the success of conservative management decreases with changes in acromion morphology: Type I acromions respond in 89% of cases, Type II in 73% of cases and Type III in just 58.3% ($P < 0.008$). Where surgical intervention is indicated, subacromial decompression has been widely accepted as a highly effective procedure. Here, removal of the bursa is necessary for not just for relief of impingement but also adequate visualization of supraspinatus before repair.

Demographic variables, many poorly characterized, represent the other major group of extrinsic factors. While these factors are no doubt important, the relationship between epidemiology and cuff pathology is a challenging one due to the lack of quality data available. Mechanical overuse is one such demographic factor associated with cuff tearing. Consistent with such an association, there is more often symptomatic disease in dominant arms than in non-dominant arms [93]. However, 36% of those presenting with a full-thickness symptomatic cuff tear had a full-thickness tear of the contralateral non-dominant side. Moreover, 28% of symptomatic patients present with a full-thickness tear their non-dominant arm *only* [39]. When asked about the levels of their activity, 70% of full-thickness tears occurred in sedentary individuals who did light work only. Therefore, whilst mechanical factors are important in a subgroup of individuals, they are only one of several factors acting in concert within susceptible individuals.

Any process that impairs tissue healing will also contribute to cuff disease. For example, smokers are less likely to respond favorably to cuff repair operations, with reduced post-operative function and satisfaction relative to non-smokers [60]. In a rat animal model, Galatz et al. [32] demonstrated that a significant factor in this regard could be the deleterious effects of nicotine on tendon healing. They implanted osmotic pumps in 72 rats, delivering either nicotine or saline solution to the area around the cuff repair. The nicotine group showed increased persistence of inflammatory markers, reduced mechanical properties and reduced collagen concentrations relative to the saline-controls. Although these nicotine concentrations are supraphysiological, the effects demonstrated in this model may apply to humans in vivo.

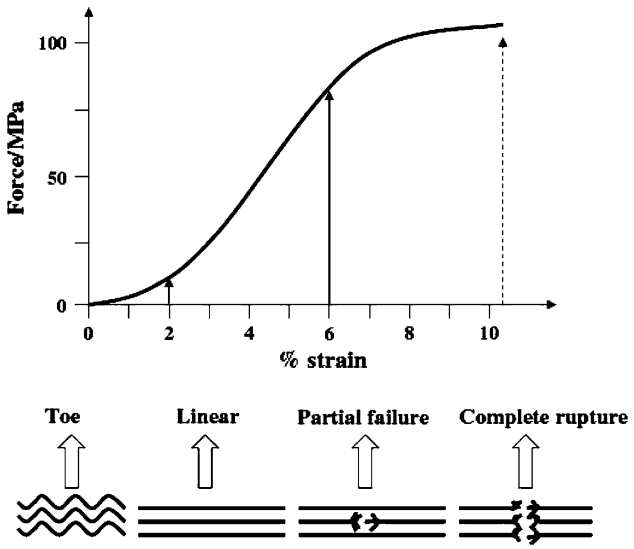

Fig. 1 Progressive tendon damage places further strain on remaining tissue predisposing to further tearing in a 'vicious circle' of disease. If tendon healing does not occur at a pace to keep up with tendon damage, the cuff will eventually tear. Figure reproduced with permission from Rees et al., Current Concepts in management of Tendon Disorders [69]

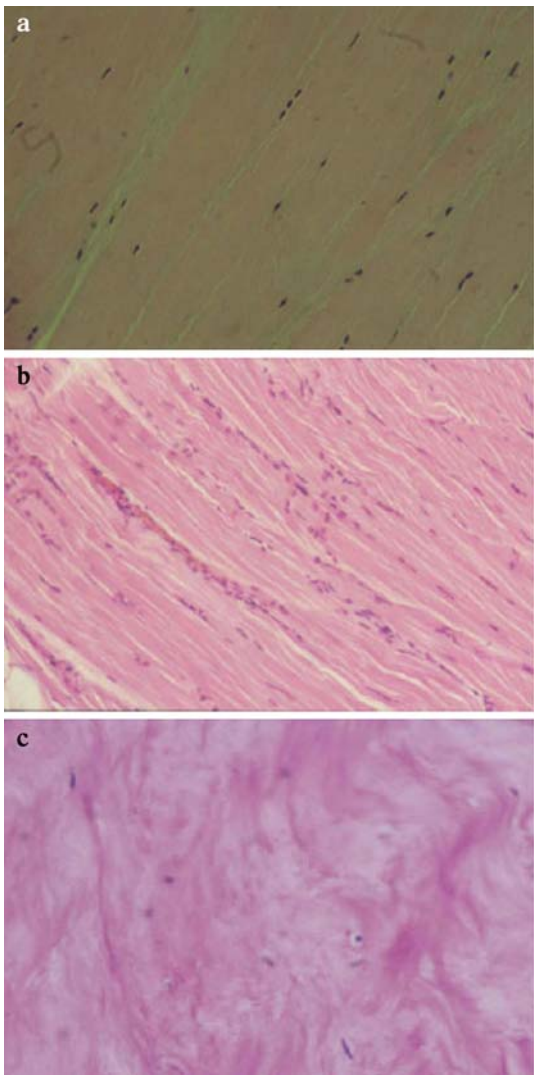

Fig. 2 **a** Normal tendon with organized, elongated tendon. **b** Moderately degenerative tissue with disorganization and increasing cellularity. **c** Highly degenerative tissue with chondroid metaplasia, little semblance of tissue architecture, *reduced* cellularity and little inflammatory response. From Maffulli N, Renström M, Leadbetter WB (ed) Tendon injuries basic science and clinical medicine. Springer, London, 2005, with kind permission of Springer Science and Business Media

Intrinsic factors

Intrinsic factors encompass the range of mechanisms that occur within the rotator cuff itself. Chief amongst these is a *degenerative-microtrauma* model that supposes age-related

tendon damage compounded by chronic microtrauma results in partial tendon tears that then develop into full rotator cuff tears. Typically after the deep fibers tear, they retract because they remain under tension, even with the arm at rest. This results in an increased load on the remaining fibers that increases the likelihood of further rupture [61]. As a result of repetitive microtrauma in the setting of a degenerative rotator cuff tendon, inflammatory mediators alter the local environment and oxidative stress induces tenocyte apoptosis causing further rotator cuff tendon degeneration.

The degeneration-microtrauma theory

Rotator cuff tendon degeneration

Epidemiological studies support a relationship between age and cuff tear prevalence. Tempelhof et al. [80] found that the frequency of cuff tears in asymptomatic volunteers increased from 13% in the youngest group (age 50–59) to 20% (age 60–69), 31% (age 70–79) and 51% in the oldest group (80–89). Hashimoto et al. [40] described seven characteristic features of such age-related degeneration in cadaveric specimens: thinning and disorientation of the collagen fibers (100%), myxoid degeneration (100%), hyaline degeneration (100%), vascular proliferation (34%), fatty infiltration (33%), chondroid metaplasia (21%) and calcification (19%). Of these, only vascular proliferation and fatty infiltration were more common on the bursal side relative to the articular side. The authors supposed that these two changes reflected reparative processes, with the remaining features representing primarily degenerative changes [43].

Of these, the modulation of collagen composition and organization is best understood. In health, the central zone of the supraspinatus tendon is primarily composed of type I collagen with smaller amounts of type III collagen, decorin, and biglycan. The fibrocartilaginous zone of the tendon insertion against the humerus is primarily composed of type II collagen, a collagen subtype associated with withstanding compressive loads. In diseased rotator cuff, there is an increase in type III collagen within the fibrocartilaginous zone, a collagen subtype associated with tendon healing. There is a concurrent decrease in type II collagen [48]. It is unclear how much of the alteration in collagen composition is a age-related degeneration, progressive injury [70], or a result of changing patterns of use [2]. These changes likely reduce the tendon's ability to withstand the compressive loads traditionally associated with type II collagen, predisposing to tears.

Muscle atrophy and fatty infiltration have also been well documented after rotator cuff tearing [36]. Degenerate, atrophied muscle fibers are infiltrated with fat and fibrous tissue as part of an attempted reparative mechanism. However, this reparative mechanism is unsatisfactory as the degree of fatty infiltration and atrophy are both inversely correlated with loss of strength [33]. Such infiltration makes a successful repair extremely challenging, but not impossible [71]. Several studies have shown a successful cuff repair can moderately improve the degree of muscle atrophy and arrest further fatty infiltration. In contrast, failed repairs and conservative management will lead to both fatty infiltration and atrophy worsening markedly over time [33, 98]. Since this process is both irreversible and functionally damaging, such evidence advocates early repair of rotator cuffs before these changes take place in symptomatic patients.

Inflammation and oxidative stress

A repetitive microtrauma model also implies the possibility of an inflammatory component—both acutely with any injury, and chronically with any reparative process. This inflammatory component has been modeled in a rat overuse shoulder system by the Soslowsky group [77] demonstrating acute increases in angiogenic and inflammatory markers associated with concomitant declines in normal collagen constituents and architecture. Human experimental models are, however, more difficult to replicate. Progressive cyclic loading of human tendons in vitro have been used to mimic tissue injury. Tsuzaki et al. [83] investigated the biochemical cascade of Il-1β on human tendon cells, on the basis of in vivo studies that suggested it may be a pro-inflammatory mediator. They found increases in mRNA levels of COX-2 mirrored with an increase in tissue concentrations of prostaglandin E2 (PGE_2). In addition, they found increased expression of matrix metalloproteases (MMP-1-3 and -13) as well as *non-lymphocyte* tissue production of the pro-inflammatory cytokine Il-1β that amplified the original exogenous cytokine delivery. While the importance of Il-1β to cuff tears remains inconclusive in vivo [35], if true, this study supposes the painful symptomatology of cuff disease is mediated via COX-2 and PGE_2 whilst the loss of tissue architecture is mediated by the range of metalloproteases released by the activated tendons.

Age-related degeneration is also influenced by the accumulation of reactive oxygen species (ROS) leading to oxidative stress and increased cell apoptosis, already implicated in damage to several other organ systems [25]. Yuan et al. [95] noted an increased proportion of apoptotic cells at the edge of a rotator cuff tear compared to controls. Possible mediators for these apoptotic pathways include matrix metalloproteinase-1 (MMP-1) within the extracellular matrix and c-Jun N-terminal protein kinase (JNK) within the intracellular environment. MMP-1 is found in

normal tendon at very low concentrations to effect the natural turnover of collagen. MMP-1 concentrations are increased in damaged tendon, likely contributing to disorganized tissue architecture, reduced collagen synthesis and weakened tendon biomechanics [34]. JNK1 is a mitogen induced protein kinase induced in tendon by interleukins and cyclic mechanical stretch [76]. When phosphorylated, the JNK-family activates a number of downstream transcription factors linked to the apoptotic pathway [29]. JNK-specific inhibitors lead to reduced MMP-1 levels and JNK2 knockout mice show reduced expression of MMP-3 and -13 with concomitant reduction in cartilage erosion, supporting the upstream role of JNK in the regulation of the extracellular matrix [38]. Antioxidants, such as the peroxidase Peroxiredoxin 5 (PRDX5), [94] reduce tendon apoptosis and result in increases in neocollagen synthesis. This implies that oxidative damage induced apoptosis may be a significant, and possibly modifiable, contributor to rotator cuff degeneration (Fig. 3).

Cuff vascularity: a theory in decline

It has been traditionally taught there exists a 'critical' or hypovascular zone 10–15 mm proximal to the insertion of the supraspinatus tendon [54]. These assertions have now become an area of controversy. Moseley and Goldie [64] examined capillary distributions in cuff specimens and concluded that no hypovascular areas existed. Brooks et al. [11] determined that both vessel diameter and number were approximately reduced by a third at 5 mm from the cuff edge compared with 30 mm, but no significantly hypovascular areas existed. Indeed, histologic and immunohistochemical and intraoperative Doppler flowmetry analysis have reported relative *hyperperfusion* at the area of the critical zone [31, 79]. The hypervascularity in such cases is thought to come from proliferation in the subsynovial layer in response to injury [86]. Fealy et al. [28] demonstrated that a robust vascular response exist post-surgery, particularly in the peritendinous region, that decreases predictably over time. Taken together, this evidence suggests hypovascularity is unlikely to be a significant contributor to cuff pathology.

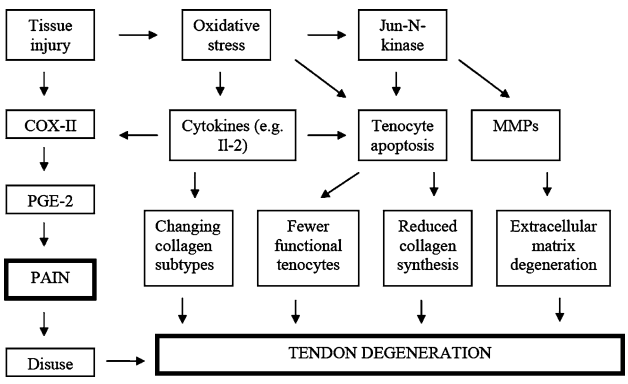

Fig. 3 The degeration-microtrauma model for cuff tearing. While tissue injury is an initiating factor, the maladaptive response to injury in susceptible individuals is likely responsible for progression into full-thickness cuff tearing

Summary

Emerging studies have elucidated the complex process of rotator cuff degeneration. Acromial morphology in particular contributes to bursal-sided cuff tears. More commonly, cuff tears are thought to begin on the articular side in the context of age-related degeneration and microtrauma. Inflammatory changes, oxidative stress, tissue remodeling and apoptosis are all important parts of this pathological process. 'Low level' mediators involved in these processes include arachadonic acid metabolites (prostaglandins, leukotrienes), MMPs, protein kinases (e.g. c-JNK), apoptotic mediators and ROS. This in turn has led to 'high-level' targets for intervention: cyclooxygenase (prostaglandins) through the use of NSAIDs, phospholipase A-2 (arachidonic acid metabolites) through corticosteroids, cytokines again through corticosteroids, and antioxidants to 'mop-up' pro-apoptotic ROS. These may be delivered systemically or locally, and in future targeted pharmacological interventions, in concert with surgical intervention, may play an increasing role in rotator cuff therapies.

Evidence-based concepts in arthroscopic rotator cuff repair

The goal of rotator cuff surgery is to optimize the connection between bone and soft tissue at the rotator cuff footprint. A recent Cochrane review showed good evidence for functional outcomes in arthroscopic repairs being equivalent to open and mini-open repairs, with potentials for earlier recovery with arthroscopic intervention [18]. The rapid growth of arthroscopy has been accompanied by equally rapid developments in suture and anchor technology. Over the last few years, a multitude of studies have investigated an array of sutures, anchors, and their respective configurations.

Imaging in rotator cuff disease

In the last decade, developments in imaging, particularly MRI, have revolutionized diagnosis and management of rotator cuff disease. Other imaging modalities can also be effective. Plain films with arthrography were the historic method of identifying complete cuff tears but are poor at

identifying partial tears and are now largely obsolete. Computed Tomography (CT) scans are excellent at diagnosing bony lesions and dislocations but also poor at diagnosing partial cuff tears and carry a high radiation dose with each scan. High resolution ultrasound (US) in experienced hands can give results equivalent to that of MRI [72] but its efficacy is limited by a marked degree of operator dependence. Modern MRI, although more expensive and time-consuming than ultrasound, is outstanding at providing high resolution, objective anatomic clarity of shoulder lesions, particularly cuff tears. The sensitivity of MRI and ultrasound for full-thickness tears is 89 and 87%, respectively, with a specificity of 93 and 96% [24]. Partial tears remain challenging to diagnose, but MR and US offer significant diagnostic improvement over CT or conventional arthrograms. Sensitivity for MR and US is 44 and 67%, respectively, with specificity of 94 and 90% [24].

Certain lesions still remain difficult to visualize with conventional MRI, most notably injury to the glenoid labrum and surrounding ligaments [26]. Where these are suspected, MRI arthrograms can provide additional information. The arthrogram effect is where a joint effusion, either pathological or iatrogenic, enhances contrast of intraarticular structures particularly on T2-weighted images. In MR arthrograms, diluted gadolinium is used to create an iatrogenic effusion to utilize this phenomenon. It is the best technique for diagnosing capsulolabral injury, and its ability to detect gadolinium in the subacromial or subdeltoid space will allow differentiation of a partial tear of the articular surface from a full-thickness tear [73]. The down-sides of MR arthrography is the additional time, expertise and money necessary for intraarticular injections and the risks that these injections carry, most notably joint infection. As 3D rendering improves, virtual MRI arthrography may provide quality of imaging similar to surgical arthroscopy [73].

Rotator cuff repair

The standard cuff repair must collectively withstand physiological loads in the post-operative period while biological healing takes place. However, it also contains several points of potential weakness. These include the stitch, the suture material, the knot, and the fixation between anchor and bone. Different studies have attempted to find the most optimal biomechanical construct to offset these potential areas of failure (Fig. 4). Factors identified with good post-operative patient outcomes include a recent tear in a younger patient (ideally less than 65 years) [88] without extensive medical co-morbidities, the absence of smoking [60], a smaller tear with minimal muscle hypotrophy or fatty infiltration [27] and an acromiohumeral distance greater than 6 mm [88].

Suture filament and material

The ideal suture must remain sufficiently strong over time so as to keep the construct stable under the burden of any physiological forces in the post-operative period. The suture should be stiff enough to resist slipping, but not so stiff as to cut through tendon or bone. In addition, the operative technique for placing the suture should be both reliable and simple to perform.

Previous studies have established braided sutures tend to be superior to monofilaments [55]. In recent years there has been a shift away from the use of simple braided polyester sutures, such as No. 2 Ethibond (Ethicon, Somerville, NJ, USA), towards hybrid sutures with a core of ultrahigh molecular weight polyethylene (UHMWPE) surrounded by braided polyester. Several studies have compared No. 2 Ethibond with one of these new hybrids: No. 2 Fiberwire (Arthrex, Naples, FL, USA). These studies [1, 19, 20, 53, 55] unanimously agree that UHMWPE sutures have an

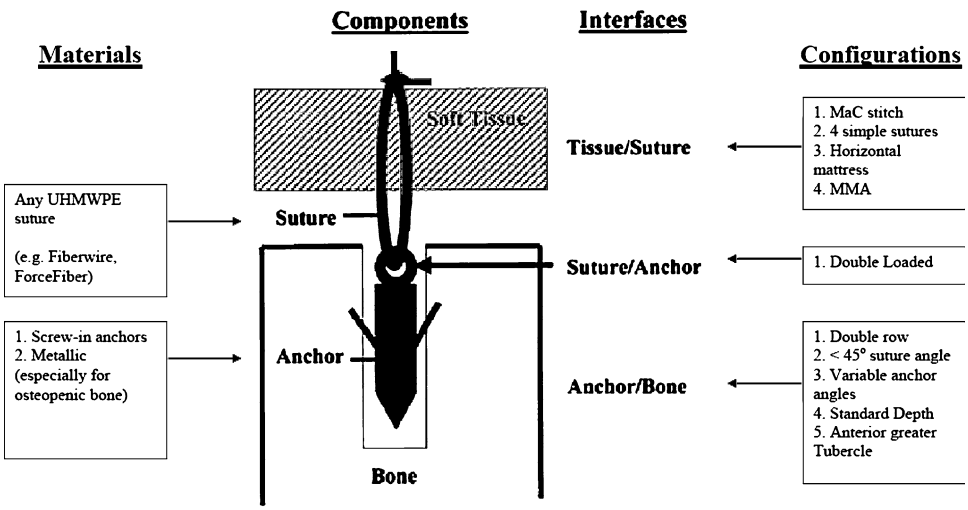

Fig. 4 The arthroscopic rotator cuff repair. Recommendations are for best biomechanical construct, not necessarily best clinical outcome. Data pooled from multiple sources [1, 19, 20, 53, 55, 81, 82]

approximately 50–80% higher tensile load than Ethibond with at least a 5× increase in cycles-to-failure [52]. De Carli et al. [19] examined failure mode and observed that Fiberwire constructs tended to fail by anchor slippage or eyelet rupture whereas Ethibond constructs failed by suture breakage. Therefore, the newer hybrids likely transfer the 'weak link' from suture breakage to other parts of the construct. However, there is a possibility that the stiffness of these sutures actually predisposes to construct failure through the suture cutting through the anchor eyelet or the tendon. Other similar UHMWPE sutures include No. 2 MagnumWire (Arthrocare, Axya), No. 2 Ultrabraid (Smith and Nephew), No. 2 Maxbraid PE (Arthrotek) and No. 2 HiFi (Linvatec), all with similar biomechanics [6].

Stitch configurations

Open rotator cuff repair often utilized a modified Mason Allen (MMA) stitch because of its biomechanical and clinical efficacy [75]. However, the MMA is difficult to perform arthroscopically, and simpler configurations with similar biomechanical properties have been sought. Again, studies have shown a variety of sutures are biomechanically suitable.

The massive cuff (MaC) stitch [57] is a combination of simple and horizontal stitches that has an ultimate tensile load (UTL) similar to that of a modified Mason–Allen (MMA) suture (233 ± 40 and 246 ± 40 N, respectively). This is attractive not only because of its relative simplicity but its fundamental structural similarity to the MMA. The MaC stitch and the MMA were superior to either a single simple (72 ± 18 N) or horizontal stitch (77 ± 15 N) alone. Koganti et al. [47] advocate the use of the locked mattress suture as an alternative to the MMA. They found the mean cycles-to-5 mm failure were significantly higher for locked mattress sutures (628) over locked inverted mattress (197), horizontal mattress (193) followed by a single simple suture (65). White et al. [91] showed the use of 4 simple stitches (155 ± 27 N) had a statistically similar UTL to the MMA stitch (140 ± 29 N), 2 mattress sutures (169 ± 56 N) and a single modified Kessler suture (161 ± 17 N).

Anchor fixation: anchor types

The purpose of the suture anchor is to fix the suture, itself connected to the rotator cuff tendon, in close proximity to bone. This therefore represents a weakness at two major points—the interface between bone and anchor, and the interface between suture and anchor. Consequently, the ideal anchor requires both an ability to withstand pullout during the physiologic loads of rehabilitation and an eyelet that protects against suture abrasion or breakage. The majority of commercially available metallic anchors have a satisfactory pullout strength, almost always higher than the tensile load of sutures they incorporate [7]. However, screw-type anchors had significantly higher failure load compared with nonscrew-type, particularly beneficial in the context of osteopenic bone [7].

Several bioabsorbable anchors are now available commercially. They have several advantages over metallic anchors: (1) no lasting foreign object in the body, (2) no imaging artifact after degradation, (3) a graduated loss of strength that slowly increases tendon-bone load thus conceivably favoring a healing process and (4) eyelet structures that have favorable characteristics with regards to suture abrasion [8]. From a biomechanical perspective, these anchors have excellent results. De Carli et al. [19] compared BioCorkscrew 5.0 and 6.5 with a metal anchor (Corkscrew). Ultimate failure loads were statistically similar for all anchors. However, the bioabsorbable anchors tended to fail by eyelet rupture whilst the metal anchors tended to fail by anchor slippage or suture breakage. Therefore, the eyelet, although preventing suture breakage, probably represents the mechanically weakest part of bioabsorbable anchors.

Potential pitfalls in bioabsorbable anchors relates to their degradation. Clearly, if strength loss occurs too quickly, the repair construct may fail. The degree of this loss in is dependant on the material used. Polyglycolic acid (PGA) polymers tend to degrade quickly over months whereas Polylactic acid (PLA) polymers tend to degrade much more slowly over years. This theory is reflected in biomechanical studies. Demirhan et al. [23] showed a pure PGA anchor retained only 75% strength at 12 weeks ($P < 0.001$). PLA anchors by contrast showed no significant change in strength over the same period both in vitro [45] and in vivo [22]. Commercially available bioabsorbable anchors are often PGA–PLA hybrids such as the Panalock (Dupuy) anchor. The manufacturer of this device claims it retains 90% strength at three months post-implantation. However, limited peer-reviewed data exists about the rates and variability in decay rates of bioabsorbable anchors in humans. Another feature of anchors, like any implant, is their capacity to induce a local foreign body reaction. Isolated case reports and series exist reflecting reactions to bioabsorbable anchors [13, 17, 44], almost never seen with metallic anchors.

Anchor fixation: configuration

Several aspects how an anchor is placed affect the repair—the angle and depth of anchor insertion, the anchor positioning on the humerus and the number of anchors used.

When inserting an anchor, the surgeon must choose both the depth and angle that is most biomechanically suitable for a repair. In a human cadaver model, Mahar et al. [58] examined failure at standard and deep depths and found

deep anchors exhibited significantly greater displacement under cyclic loading than standard-depth anchors. Consequently, the insertion of anchors at depths deeper than the manufacturer's standard cannot be recommended. With regard to angle of anchor insertion, Burkhart has advocated placing the anchor at around 45° (the 'deadman' angle) [12]. In vitro results are, however, equivocal, with little difference in pullout load at various angles between 30° and 80° [20, 51]. However, Deakin et al. [20] noted that suture angles of greater than 45° predispose to abrasion and breakage, but exclusively in metallic anchors. The bioabsorbable Biocorkscrew anchor was relatively insensitive to angle by virtue of its polyaxial eyelet. Therefore, whilst anchor insertion is relatively flexible, the suture should be inserted between 0° and 45° in metallic anchors for a more robust construct.

Another variable is whether the anchor contains a single or double loaded suture. In both theory and practice, double loading a suture anchor, doubles the number of fixation points consequently reducing the tension at each fixation point by approximately 50% [14]. This has been shown to lead to biomechanically more secure construct and is now considered standard practice [16].

Since anchors insert into bone, it seems reasonable to assume that the quality of bone will affect how well the anchor is secured. Tingart et al. [81, 82] found a positive correlation between bone mineral density (BMD) and pull-out strength in a cadaveric model. They found anchor pullout loads were 62% higher in the anterior and middle parts of the greater tuberosity compared to the posterior part, 53% higher in the proximal part of the tuberosity than the distal, and 32% higher in the lesser tuberosity compared with the greater tuberosity. These findings have since been corroborated [56]. Tingart et al. [81, 82] also showed that in the areas with high BMD, screw-in metal anchors were equal in pullout load to biodegradable hook anchors, but where BMD was low, the screw-in anchors were significantly superior. From these studies, two recommendations can be made: (1) the anterior and middle parts of the greater tuberosity give improved pullout anchor strengths, (2) osteopenic patients will benefit from the use of screw-type metal anchors and a greater number of anchors than would otherwise be used.

Another long-standing practice is the formation of a cancellous trough to aid tendon-bone healing. The rationale behind this is sound—cancellous bone is both highly vascular and more denser than cortical bone. Localized bony damage will induce an additional inflammatory response that can aid tendon-bone healing. However, this comes at the cost of marginally increasing the width of the cuff repair. Limited arthroscopic studies done so far do not show any evidence of improved outcomes with trough formation [37], however, larger studies are needed if any recommendations are to be made.

A controversial question in arthroscopic cuff surgery is the relative merits of single-row versus double-row repairs. In vitro anatomical studies strongly suggest that a double-row repair produces a significantly larger supraspinatus footprint [63, 84] and better biomechanical construct than single-row repairs. Indeed, showed gap formation during cyclic testing was significantly less for double-row repairs over single-row, and the double-row repairs had a 46% higher UTL [46]. Ma et al. [56] found the mean UTL for double-row repairs (287 ± 24 N) was higher than any of the three single-row repairs they tested [simple suture (191 N), MMA (212 N) and massive cuff (250 N) stitches, $P < 0.05$].

Whilst double-row repairs have excellent properties in vitro, Park et al. [67] demonstrated that even double-row repairs only had 50% of the contact area and 80% of the contact pressure of transosseous repairs. Hypothesizing that a larger footprint and higher pressures favored healing, they developed a 'transosseous equivalent' technique [68]. This technique used suture bridges between anchors to add mechanical support to the repair construct, with either two-bridge or a four-bridge structure. They found the four-bridge repairs had 2× the contact area and 1.4× the contact pressure of double-row repair, suggesting they may indeed be 'equivalent' in this regard with transosseous repairs, although no direct comparisons was made. The study also found failure load was significantly higher (50%) for the suture bridge repairs but gap formation during cyclic testing was unaffected. This technique clearly shows considerable promise.

Clinical studies

So far, we have discussed the in vitro biomechanical construct. Many advances in repair strengths may in fact have minimal clinical impact. A series of clinical studies have been published recently on arthroscopic rotator cuff repairs. These are summarized in Table 1. The studies have confirmed that double-row arthroscopic repairs have outcomes statistically similar to, if not better than, open and mini-open repair [3, 49]. Verma et al. found post-operative ASES scores in supraspinatus tear repairs were statistically similar between the arthroscopic repair cohort (94.6) and the mini-open cohort (95.1). MRI findings too showed similar re-tear rates between the two groups (arthroscopic: 24%, mini-open: 27%) [87].

Recent studies have also addressed the relative efficacy of single and double-row repairs. Lichtenberg et al. [50] followed 53 arthroscopic cuff tears at 24 months follow-up. They used single-row repairs with a bioabsorbable anchor and MMA stitches. They found improvements in Constant scores from 53 preoperatively to 86.1 postoperatively with a 24.5% re-tear rate on MRI at 2 years, results equivalent to

Table 1 Key clinical studies of arthroscopic rotator cuff repair with clinical and radiographic outcomes

References	Cohort size	Clinical scoring	Preop score (mean)	Postop score (mean)	Suture material	Stitch	Anchor type	Anchor configuration	Re-tear rate overall (%)	Re-tear rate small (%)	Re-tear rate medium (%)	Re-tear rate large (%)	Re-tear rate massive (%)
Galatz et al. [32]	18 (massive only)	ASES	48.3	84.6	No. 2, nonabsorbable	N/A	5 mm PLLA corkscrew (Arthrex)	Variable	94	N/A	N/A	N/A	94
Boileau et al. [10]	65	Constant	51.6	83.8	No.1 Polydioxanone (Ethicon)	Inverted horizontal mattress	Panalok RC (Mitek)	Single row	29	9	0	46	50
Verma et al. [87]	38	ASES	N/A	94.6	N/A	N/A	N/A	N/A	24	N/A	15	N/A	50
Lichtenberg et al. [50]	53	Constant	53.6	86.1	N/A	MMA	Bioabsorbable	Single row	24.5	N/A	N/A	N/A	N/A
Anderson et al. [3]	52	L'Insalata	42	93	N/A	Horizontal Mattress	Bioabsorbable (varied)	Double row	17	N/A	N/A	N/A	N/A
Huijsmans et al. [41]	242	Constant	54.9	80	No. 2 Ethibond	Mattress	Metallic	Double row	17	12	7	22	53
LaFosse et al. [49]	105	Constant + strength testing	43.2	80.1	No. 2 Ethibond	Simple/Lasso-loop	G2 Anchor (Mitek)	Double row	11	0	N/A	21	18
Sugaya et al. [78]	86	ASES	42.3	94.3	Nylon	Simple	Metallic	Double row	17	0	10	36	50

N/A implies data not specified within study

that of open procedures. Huijsmans et al. [41] followed 242 arthroscopically repaired rotator cuffs over a similar period. In contrast to Lichenberg et al., they used double-row repairs with a metallic anchor. Constant scores in their patients were similar: 54.9 pre-operatively to 80 post-operatively. Re-tear rates on ultrasound depended on tear size, with massive tears having a considerably poorer anatomic outcome. While direct comparison between these studies is difficult, functional outcomes between single-row and double-row repairs appear similar. The marginally lower scores in the Huijsmans et al. cohort may be explained by the greater proportion of massive tears in this group. Anderson et al. [3] followed 52 arthroscopic cuff tear repairs over 2 years. They used double-row repairs, horizontal mattress sutures and a range of bioabsorbable anchors. They found 17% retear rate at 2 years on MRI with impressive improvements in L'Insalata grading scores. Again, interpreted with caution, it may be that double-row repairs produce a lower re-tear rate and better anatomic results. Two studies have directly compared single-row with double-row repairs. Sugaya et al. [78] in a retrospective non-randomized study showed no apparent differences in ASES and UCLA scores at 2 year follow-up. However, post-operative MRI in the double-row repair patients showed superior structural results ($P < 0.01$) when using a subjective grading score. Franceschi et al. [30] published a randomized cohort of 60 patients with either single or double-row repair with 2 year follow-up. They too found statistically similar UCLA scores in the two groups but improved radiological results in double-row repairs. Therefore, while double-row repairs give superior anatomical and biomechanical results in vivo [3], no data currently supports better subjective outcomes than single-row repairs.

Another issue addressed in the recent studies is the prognosis, both anatomically and functionally of anatomically repaired larger cuff tears, involving multiple tendons. Huijsmans et al. [41] analyzed 32 massive cuff tears in their cohort of 242. Retear rates were high in this group: only 47% of cuffs were intact at 2 years, and 33% had already failed at 3 weeks. While there was a significant difference in improvement in Constant scores between intact (26 points) and failed (21 points) tendon ($P = 0.02$), there was still a somewhat paradoxical improvement in patients who had a failed repair. Additionally, no *subjective* index—satisfaction levels, pain scores, etc., could be correlated to repair integrity. In comparison, structural success rates for small and medium tears were excellent—93% at final follow-up. Boileau et al. [10] showed similar results. Supraspinatus-only tears had excellent structural outcomes—91% remaining intact at 2 years. However, involvement of any other tendon or of the rotator interval reduced intact repair rates to 51%. There were objective differences in constant scores between the intact (85.7) and failed (78.9) groups ($P = 0.02$). Again, there were no subjective differences between patients with radiographically intact or failed tendons with excellent satisfaction rates in both groups.

One can conclude that biomechanically stronger repairs are more likely to remain structurally intact—a logical endpoint. Both smaller cuff tears and double-row repairs have been shown to lead to better anatomical restoration. In turn, anatomical restoration has been demonstrated to correlate with post-operative strength and improvements in functional scores. However, in the direct single versus double anchor studies, double-row repairs do not lead to improvements in functional scoring. Whilst discouraging, logic dictates that it is more likely that the existing studies are underpowered and improvements in functional scores may emerge with larger studies.

It is interesting to note how patients' satisfaction and function improves even with a failed tendon repair, and that elucidating significant differences in functional scores between structurally intact and failed tendons requires large patient cohorts. The etiology of this phenomenon is likely to be multifactorial, with both non-surgical and surgical factors. Of the surgical factors, subacromial decompression, standard in may rotator cuff repairs, likely leads to improvements in pain and function, independent of the status of the damaged tendon. With regard non-surgical factors, it has been noted that patients enrolling in surgery often have a stricter, more intensive physical therapy (PT) regimen. Additionally, the aftermath of an operation can serve as a lifting of a psychological barrier that pushes patients to rehabilitate more aggressively. Both these factors may improve shoulder function. These factors are interesting to consider as we remain in our quest for better, more reliable surgical results.

Biologic augmentation: considerations in difficult cases

Several new techniques have been developed to augment the conventional rotator cuff repair. Since non-massive tears have generally good outcomes, the risk-benefit consideration of biologic therapy dissuades many surgeons from such relatively experimental interventions. Massive tears by contrast often have poorer long-term outcomes, and biologic therapies may be particularly valuable in such cases.

Several different materials have been proposed as grafts in massive tears. These include porcine small intestinal submucosa (PSIS) grafts, human/animal skin, muscle auto/allografts as well as several synthetic materials. Barber et al. [5] published a comparison of the in vitro properties of 7 different grafts. While this study represented the UTL of a repair, it clearly did not reflect any properties of the graft in augmenting healing, and thus its relevance is limited. They found that the strongest repairs were human skin (GraftJacket: 157 N, 182 N and 229 N depending on

the thickness used) followed by porcine skin (Permacol: 128 N), bovine skin (TissueMend: 76 N) and PSIS (Restore: 38 N, CuffPatch: 32 N).

PSIS has been used in dog models for the repair of ruptured Achilles tendons, with good new tendon formation and minimal residual tissue, adhesions or chronic inflammation [4]. PSIS acts as a three-dimensional scaffold attracting host cells and promoting regeneration. Several studies are now available to evaluate the role of PSIS in rotator cuff repair. Zalvaras et al. [96] found that 16 weeks following rotator cuff repair in a rat model, the rats with PSIS had UTL 78% of normal compared with 36% of normal in the unaugmented repairs ($P < 0.008$). However, dog [21] and sheep [74] animal model studies found no significant differences in UTL between PSIS and unaugmented repairs. In the landmark clinical study to date on this subject, Iannotti et al. [42] divided 30 patients evenly into PSIS-augmented and PSIS-unaugmented groups. They found that 9/15 unaugmented repairs showed healing compared with just 4/15 augmented repairs ($P = 0.11$). PENN scores were 83 postoperatively in the PSIS group compared to 91 in the control group ($P = 0.07$). Although just a single study, this along with equivocal animal models brings the future role of PSIS into doubt. Moreover, concerns remain about any xenograft, and PSIS is no exception. Zheng et al. [97] reported that the Restore acellular graft still contained porcine DNA and thus recipients were exposed to the theoretical risk of xenograft retroviruses and immunologic rejection at the graft site. Malcarney et al. [59] reported a series of 25 massive cuff tear patients treated with PSIS grafts (Restore), where 4 patients had an early (mean 13 days), non-specific inflammatory reaction at the graft site requiring a second operation for debridement and graft removal. The authors did not confirm the cause of the inflammation, although infection and graft rejection are certainly possibilities.

Other techniques proposed are still in their infancy. Gene transfection via adenovirus vectors has been successfully implemented to deliver pro-regenerative cytokines/growth factors to tendon sites in animal models [85]. Autologous tenocyte-implanted PSIS grafts were used by Chen et al. [15] to good effect in rabbit rotator cuff. This technique has particular relevance in massive tears where contracted tissue has little regenerative properties of its own. These techniques have to be first explored in animal studies before any clinical exposure can be considered.

Physical therapy

Post-operative PT is crucial to a successful patient recovery. The quality and intensity of this depends largely on the predicted strength of the cuff repair. Tears with wide margins, poor quality tissue or a previously failed repair are particularly high risk for repair failure. Initial PT should focus on passive exercises, later gradually moving to active loading with recovery taking anywhere from several months to a year depending on the fragility of the lesion [62]. A full consideration of PT is beyond the scope of this article and has been reviewed extensively elsewhere [92].

Summary

The biomechanical construct of the repair of torn rotator cuff can be broken down into three potential areas of failure: tissue—suture interface, suture—anchor interface, and the anchor—bone interface. With the development of reliable suture anchors and synthetic hybrid suture materials, the ‘weak link’ has been shifted to the tissue—suture interface. At present, the correlation between biomechanical strength and clinical failure is not yet known, and in vitro studies may not necessarily apply to conditions in vivo. Until additional studies are performed to clearly define the mechanical strength of repair that is required for biologic healing, surgeons should aim to produce the strongest possible biomechanical repair construct.

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References

1. Abbi G, Espinoza L, Odell T, Mahar A, Pedowitz R (2006) Evaluation of 5 knots and 2 suture materials for arthroscopic rotator cuff repair: very strong sutures can still slip. Arthroscopy 22:38–43
2. Amiel D, Akeson WH, Harwood FL, Mechanic GL (1980) The effect of immobilization on the types of collagen synthesized in periarticular connective tissue. Connect Tissue Res 8:27–32
3. Anderson K, Boothby M, Aschenbrener D, van Holsbeeck M (2006) Outcome and structural integrity after arthroscopic rotator cuff repair using 2 rows of fixation: minimum 2-year follow-up. Am J Sports Med 34:1899–1905
4. Badylak SF, Tullius R, Kokini K, Shelbourne KD, Klootwyk T, Voytik SL, Kraine MR, Simmons C (1995) The use of xenogeneic small intestinal submucosa as a biomaterial for Achilles tendon repair in a dog model. J Biomed Mater Res 29:977–985
5. Barber FA, Herbert MA, Coons DA (2006) Tendon augmentation grafts: biomechanical failure loads and failure patterns. Arthroscopy 22:534–538
6. Barber FA, Herbert MA, Coons DA, Boothby MH (2006) Sutures and suture anchors—update 2006. Arthroscopy 22(10):1063.e1–1063.e9
7. Barber FA, Herbert MA, Richards DP (2003) Sutures and suture anchors: update 2003. Arthroscopy 19:985–990
8. Bardana DD, Burks RT, West JR, Greis PE (2003) The effect of suture anchor design and orientation on suture abrasion: an in vitro study. Arthroscopy 19:274–281

9. Bigliani LU, Ticker JB, Flatow EL, Soslowsky LJ, Mow VC (1991) The relationship of acromial architecture to rotator cuff disease. Clin Sports Med 10:823–838
10. Boileau P, Brassart N, Watkinson DJ, Carles M, Hatzidakis AM, Krishnan SG (2005) Arthroscopic repair of full-thickness tears of the supraspinatus: does the tendon really heal? J Bone Joint Surg Am 87:1229–1240
11. Brooks CH, Revell WJ, Heatley FW (1992) A quantitative histological study of the vascularity of the rotator cuff tendon. J Bone Joint Surg Br 74:151–153
12. Burkhart SS (1995) The deadman theory of suture anchors: observations along a South Texas fence line. Arthroscopy 11:119–123
13. Burkhart SS (2000) A stepwise approach to arthroscopic rotator cuff repair based on biomechanical principles. Arthroscopy 16:82–90
14. Burkhart SS, Wirth MA, Simonich M, Salem D, Lanctot D, Athanasiou K (2000) Knot security in simple sliding knots and its relationship to rotator cuff repair: How secure must the knot be? Arthroscopy 16:202–207
15. Chen JM, Willers C, Xu J, Wang A, Zheng MH (2007) Autologous tenocyte therapy using porcine-derived bioscaffolds for massive rotator cuff defect in rabbits. Tissue Eng 13:1479–1491
16. Chhabra A, Goradia VK, Francke EI, Baer GS, Monahan T, Kline AJ, Miller MD (2005) In vitro analysis of rotator cuff repairs: a comparison of arthroscopically inserted tacks or anchors with open transosseous repairs. Arthroscopy 21:323–327
17. Chow JC, Gu Y (2004) Material reaction to suture anchor. Arthroscopy 20:314–316
18. Coghlan J, Buchbinder R, Green S, Johnston R, Bell S (2008) Surgery for rotator cuff disease. Cochrane Database Syst Rev 23(1):CD005619
19. De Carli A, Vadala A, Monaco E, Labianca L, Zanzotto E, Ferretti A (2005) Effect of cyclic loading on new polyblend suture coupled with different anchors. Am J Sports Med 33:214–219
20. Deakin M, Stubbs D, Bruce W, Goldberg J, Gillies RM, Walsh WR (2005) Suture strength and angle of load application in a suture anchor eyelet. Arthroscopy 21:1447–1451
21. Dejardin LM, Arnoczky SP, Ewers BJ, Haut RC, Clarke RB (2001) Tissue-engineered rotator cuff tendon using porcine small intestine submucosa. Am J Sports Med 29:175–184
22. Dejong ES, DeBerardino TM, Brooks DE, Judson K (2004) In vivo comparison of a metal versus a biodegradable suture anchor. Arthroscopy 20:511–516
23. Demirhan M, Kilicoglu O, Akpinar S, Akman S, Atalar AC, Goksan MA (2000) Time-dependent reduction in load to failure of wedge-type polyglyconate suture anchors. Arthroscopy 16:383–390
24. Dinnes J, Loveman E, McIntyre L, Waugh N (2003) The effectiveness of diagnostic tests for the assessment of shoulder pain due to soft tissue disorders: a systematic review. Health Technol Assess 7(29):iii, 1–166
25. Droge W (2003) Oxidative stress and aging. Adv Exp Med Biol 543:191–200
26. Ertl JP, Kovacs G, Burger RS (1998) Magnetic resonance imaging of the shoulder in the primary care setting. Med Sci Sports Exerc 30:7–11
27. Favard L, Bacle G, Berhouet J (2007) Rotator cuff repair. Joint Bone Spine 74:551–557
28. Fealy S, Adler RS, Drakos MC, Kelly AM, Allen AA, Cordasco FA, Warren RF, O'Brien SJ (2006) Patterns of vascular and anatomical response after rotator cuff repair. Am J Sports Med 34:120–127
29. Filomeni G, Aquilano K, Civitareale P, Rotilio G, Ciriolo MR (2005) Activation of c-Jun-N-terminal kinase is required for apoptosis triggered by glutathione disulfide in neuroblastoma cells. Free Radic Biol Med 39:345–354
30. Franceschi F, Ruzzini L, Longo UG, Martina FM, Zobel BB, Maffulli N, Denaro V (2007) Equivalent clinical results of arthroscopic single-row and double-row suture anchor repair for rotator cuff tears: a randomized controlled trial. Am J Sports Med 35:1254–1260
31. Fukuda H, Hamada K, Yamanaka K (1990) Pathology and pathogenesis of bursal-side rotator cuff tears viewed from en bloc histologic sections. Clin Orthop Relat Res 254:75–80
32. Galatz LM, Silva MJ, Rothermich SY, Zaegel MA, Havlioglu N, Thomopoulos S (2006) Nicotine delays tendon-to-bone healing in a rat shoulder model. J Bone Joint Surg Am 88:2027–2034
33. Gerber C, Schneeberger AG, Hoppeler H, Meyer DC (2007) Correlation of atrophy and fatty infiltration on strength and integrity of rotator cuff repairs: a study in thirteen patients. J Shoulder Elbow Surg 16:691–696
34. Gotoh M, Hamada K, Yamakawa H, Tomonaga A, Inoue A, Fukuda H (1997) Significance of granulation tissue in torn supraspinatus insertions: an immunohistochemical study with antibodies against interleukin-1 beta, cathepsin D, and matrix metalloprotease-1. J Orthop Res 15:33–39
35. Gotoh M, Hamada K, Yamakawa H, Yanagisawa K, Nakamura M, Yamazaki H, Inoue A, Fukuda H (2002) Interleukin-1-induced glenohumeral synovitis and shoulder pain in rotator cuff diseases. J Orthop Res 20:1365–1371
36. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC (1994) Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop Relat Res 304:78–83
37. Goutallier D, Postel JM, Van Driessche S, Voisin MC (2005) Histological lesions of supraspinatus tendons in full thickness tears of the rotator cuff. Rev Chir Orthop Reparatrice Appar Mot 91:109–113
38. Han Z, Boyle DL, Chang L, Bennett B, Karin M, Yang L, Manning AM, Firestein GS (2001) c-Jun N-terminal kinase is required for metalloproteinase expression and joint destruction in inflammatory arthritis. J Clin Invest 108:73–81
39. Harryman DT 2nd, Hettrich CM, Smith KL, Campbell B, Sidles JA, Matsen FA 3rd (2003) A prospective multipractice investigation of patients with full-thickness rotator cuff tears: the importance of comorbidities, practice, and other covariables on self-assessed shoulder function and health status. J Bone Joint Surg Am 85:690–696
40. Hashimoto T, Nobuhara K, Hamada T (2003) Pathologic evidence of degeneration as a primary cause of rotator cuff tear. Clin Orthop Relat Res 415:111–120
41. Huijsmans PE, Pritchard MP, Berghs BM, van Rooyen KS, Wallace AL, de Beer JF (2007) Arthroscopic rotator cuff repair with double-row fixation. J Bone Joint Surg Am 89:1248–1257
42. Iannotti JP, Codsi MJ, Kwon YW, Derwin K, Ciccone J, Brems JJ (2006) Porcine small intestine submucosa augmentation of surgical repair of chronic two-tendon rotator cuff tears. A randomized, controlled trial. J Bone Joint Surg Am 88:1238–1244
43. Kannus P, Jozsa L (1991) Histopathological changes preceding spontaneous rupture of a tendon. A controlled study of 891 patients. J Bone Joint Surg Am 73:1507–1525
44. Kelly AM, Drakos MC, Fealy S, Taylor SA, O'Brien SJ (2005) Arthroscopic release of the long head of the biceps tendon: functional outcome and clinical results. Am J Sports Med 33:208–213
45. Kilicoglu O, Demirhan M, Akman S, Atalar AC, Ozsoy S, Ince U (2003) Failure strength of bioabsorbable interference screws: effects of in vivo degradation for 12 weeks. Knee Surg Sports Traumatol Arthrosc 11:228–234

46. Kim DH, Elattrache NS, Tibone JE, Jun BJ, DeLaMora SN, Kvitne RS, Lee TQ (2006) Biomechanical comparison of a single-row versus double-row suture anchor technique for rotator cuff repair. Am J Sports Med 34:407–414
47. Koganti AK, Adamson GJ, Gregersen CS, Pink MM, Shankwiler JA (2006) Biomechanical comparison of traditional and locked suture configurations for arthroscopic repairs of the rotator cuff. Am J Sports Med 34:1832–1838
48. Kumagai J, Sarkar K, Uhthoff HK (1994) The collagen types in the attachment zone of rotator cuff tendons in the elderly: an immunohistochemical study. J Rheumatol 21:2096–2100
49. Lafosse L, Brozska R, Toussaint B, Gobezie R (2007) The outcome and structural integrity of arthroscopic rotator cuff repair with use of the double-row suture anchor technique. J Bone Joint Surg Am 89:1533–1541
50. Lichtenberg S, Liem D, Magosch P, Habermeyer P (2006) Influence of tendon healing after arthroscopic rotator cuff repair on clinical outcome using single-row Mason–Allen suture technique: a prospective, MRI controlled study. Knee Surg Sports Traumatol Arthrosc 14:1200–1206
51. Liporace FA, Bono CM, Caruso SA, Weiner B, Penny K, Feldman AJ, Grossman MG, Haher TR (2002) The mechanical effects of suture anchor insertion angle for rotator cuff repair. Orthopedics 25:399–402
52. Lo IK, Burkhart SS, Athanasiou K (2004) Abrasion resistance of two types of nonabsorbable braided suture. Arthroscopy 20:407–413
53. Lo IK, Burkhart SS, Chan KC, Athanasiou K (2004) Arthroscopic knots: determining the optimal balance of loop security and knot security. Arthroscopy 20:489–502
54. Lohr JF, Uhthoff HK (1990) The microvascular pattern of the supraspinatus tendon. Clin Orthop Relat Res 254:35–38
55. Loutzenheiser TD, Harryman DTI, Ziegler DW, Yung SW (1998) Optimizing arthroscopic knots using braided or monofilament suture. Arthroscopy 14:57–65
56. Ma CB, Comerford L, Wilson J, Puttlitz CM (2006) Biomechanical evaluation of arthroscopic rotator cuff repairs: double-row compared with single-row fixation. J Bone Joint Surg Am 88:403–410
57. Ma CB, MacGillivray JD, Clabeaux J, Lee S, Otis JC (2004) Biomechanical evaluation of arthroscopic rotator cuff stitches. J Bone Joint Surg Am 86:1211–1216
58. Mahar AT, Tucker BS, Upasani VV, Oka RS, Pedowitz RA (2005) Increasing the insertion depth of suture anchors for rotator cuff repair does not improve biomechanical stability. J Shoulder Elbow Surg 14:626–630
59. Malcarney HL, Bonar F, Murrell GA (2005) Early inflammatory reaction after rotator cuff repair with a porcine small intestine submucosal implant: a report of 4 cases. Am J Sports Med 33:907–911
60. Mallon WJ, Misamore G, Snead DS, Denton P (2004) The impact of preoperative smoking habits on the results of rotator cuff repair. J Shoulder Elbow Surg 13:129–132
61. Matsen FA 3rd (1998) The rotator cuff. In: Rockwood CA Jr, Matsen FA 3rd (eds) The shoulder, chap 15. Saunders, Philadelphia, pp 755–839
62. Matsen FA 3rd (2008) Clinical practice. Rotator-cuff failure. N Engl J Med 358:2138–2147
63. Mazzocca AD, Millett PJ, Guanche CA, Santangelo SA, Arciero RA (2005) Arthroscopic single-row versus double-row suture anchor rotator cuff repair. Am J Sports Med 33:1861–1868
64. Moseley HF, Goldie I (1963) The arterial pattern of the rotator cuff of the shoulder. J Bone Joint Surg Br 45:780–789
65. Neer CS II, Poppen NK (1987) Supraspinatus outlet. Orthop Trans 11:234
66. Nicholson GP, Goodman DA, Flatow EL, Bigliani LU (1996) The acromion: morphologic condition and age-related changes. A study of 420 scapulas. J Shoulder Elbow Surg 5:1–11
67. Park MC, Cadet ER, Levine WN, Bigliani LU, Ahmad CS (2005) Tendon-to-bone pressure distributions at a repaired rotator cuff footprint using transosseous suture and suture anchor fixation techniques. Am J Sports Med 33:1154–1159
68. Park MC, ElAttrache NS, Tibone JE, Ahmad CS, Jun BJ, Lee TQ (2007) Part I: Footprint contact characteristics for a transosseous-equivalent rotator cuff repair technique compared with a double-row repair technique. J Shoulder Elbow Surg 16:461–468
69. Rees JD, Wilson AM, Wolman RL (2006) Current concepts in the management of tendon disorders. Rheumatology (Oxford) 45:508–521
70. Riley GP, Harrall RL, Constant CR, Chard MD, Cawston TE, Hazleman BL (1994) Tendon degeneration and chronic shoulder pain: changes in the collagen composition of the human rotator cuff tendons in rotator cuff tendinitis. Ann Rheum Dis 53:359–366
71. Rokito AS, Cuomo F, Gallagher MA, Zuckerman JD (1999) Long-term functional outcome of repair of large and massive chronic tears of the rotator cuff. J Bone Joint Surg Am 81:991–997
72. Rutten MJ, Maresch BJ, Jager GJ, Blickman JG, van Holsbeeck MT (2007) Ultrasound of the rotator cuff with MRI and anatomic correlation. Eur J Radiol 62:427–436
73. Sahin G, Demirtas M (2006) An overview of MR arthrography with emphasis on the current technique and applicational hints and tips. Eur J Radiol 58:416–430
74. Schlegel TF, Hawkins RJ, Lewis CW, Motta T, Turner AS (2006) The effects of augmentation with Swine small intestine submucosa on tendon healing under tension: histologic and mechanical evaluations in sheep. Am J Sports Med 34:275–280
75. Schneeberger AG, von Roll A, Kalberer F, Jacob HA, Gerber C (2002) Mechanical strength of arthroscopic rotator cuff repair techniques: an in vitro study. J Bone Joint Surg Am 84-A:2152–2160
76. Skutek M, van Griensven M, Zeichen J, Brauer N, Bosch U (2003) Cyclic mechanical stretching of human patellar tendon fibroblasts: activation of JNK and modulation of apoptosis. Knee Surg Sports Traumatol Arthrosc 11:122–129
77. Soslowsky LJ, Thomopoulos S, Tun S, Flanagan CL, Keefer CC, Mastaw J, Carpenter JE (2000) Neer Award 1999. Overuse activity injures the supraspinatus tendon in an animal model: a histologic and biomechanical study. J Shoulder Elbow Surg 9:79–84
78. Sugaya H, Maeda K, Matsuki K, Moriishi J (2005) Functional and structural outcome after arthroscopic full-thickness rotator cuff repair: single-row versus dual-row fixation. Arthroscopy 21:1307–1316
79. Swiontkowski MF, Iannotti JP, Boulas HJ, Esterhai JL (1990) Intraoperative assessment of rotator cuff vascularity using laser Doppler flowmetry. In: Post M, Morrey BF, Hawkins RJ (eds) Surgery of the shoulder, pp 208–212, 208–212. Mosby, St. Louis
80. Tempelhof S, Rupp S, Seil R (1999) Age-related prevalence of rotator cuff tears in asymptomatic shoulders. J Shoulder Elbow Surg 8:296–299
81. Tingart MJ, Apreleva M, Lehtinen J, Zurakowski D, Warner JJ (2004) Anchor design and bone mineral density affect the pull-out strength of suture anchors in rotator cuff repair: which anchors are best to use in patients with low bone quality? Am J Sports Med 32:1466–1473
82. Tingart MJ, Apreleva M, Zurakowski D, Warner JJ (2003) Pullout strength of suture anchors used in rotator cuff repair. J Bone Joint Surg Am 85:2190–2198

83. Tsuzaki M, Guyton G, Garrett W, Archambault JM, Herzog W, Almekinders L, Bynum D, Yang X, Banes AJ (2003) IL-1 beta induces COX2, MMP-1, -3 and -13, ADAMTS-4, IL-1 beta and IL-6 in human tendon cells. J Orthop Res 21:256–264
84. Tuoheti Y, Itoi E, Yamamoto N, Seki N, Abe H, Minagawa H, Okada K, Shimada Y (2005) Contact area, contact pressure, and pressure patterns of the tendon-bone interface after rotator cuff repair. Am J Sports Med 33:1869–1874
85. Uggen JC, Dines J, Uggen CW, Mason JS, Razzano P, Dines D, Grande DA (2005) Tendon gene therapy modulates the local repair environment in the shoulder. J Am Osteopath Assoc 105:20–21
86. Uhthoff HK, Kumagi J, Sarkar K (1992) Morphologic evidence of healing in torn human rotator cuffs. J Bone Joint Surg 74B(suppl 3):293–294
87. Verma NN, Dunn W, Adler RS, Cordasco FA, Allen A, MacGillivray J, Craig E, Warren RF, Altchek DW (2006) All-arthroscopic versus mini-open rotator cuff repair: a retrospective review with minimum 2-year follow-up. Arthroscopy 22:587–594
88. Walch G, Marechal E, Maupas J, Liotard JP (1992) Surgical treatment of rotator cuff rupture. Prognostic factors. Rev Chir Orthop Reparatrice Appar Mot 78:379–388
89. Wang JC, Horner G, Brown ED, Shapiro MS (2000) The relationship between acromial morphology and conservative treatment of patients with impingement syndrome. Orthopedics 23:557–559
90. Wang JC, Shapiro MS (1997) Changes in acromial morphology with age. J Shoulder Elbow Surg 6:55–59
91. White CD, Bunker TD, Hooper RM (2006) The strength of suture configurations in arthroscopic rotator cuff repair. Arthroscopy 22:837–841
92. Williams GR Jr, Rockwood CA Jr, Bigliani LU, Iannotti JP, Stanwood W (2004) Rotator cuff tears: why do we repair them? J Bone Joint Surg Am 86:2764–2776
93. Yamaguchi K, Ditsios K, Middleton WD, Hildebolt CF, Galatz LM, Teefey SA (2006) The demographic and morphological features of rotator cuff disease. A comparison of asymptomatic and symptomatic shoulders. J Bone Joint Surg Am 88:1699–1704
94. Yuan J, Murrell GA, Trickett A, Landtmeters M, Knoops B, Wang MX (2004) Overexpression of antioxidant enzyme peroxiredoxin 5 protects human tendon cells against apoptosis and loss of cellular function during oxidative stress. Biochim Biophys Acta 1693:37–45
95. Yuan J, Murrell GA, Wei AQ, Wang MX (2002) Apoptosis in rotator cuff tendonopathy. J Orthop Res 20:1372–1379
96. Zalavras CG, Gardocki R, Huang E, Stevanovic M, Hedman T, Tibone J (2006) Reconstruction of large rotator cuff tendon defects with porcine small intestinal submucosa in an animal model. J Shoulder Elbow Surg 15:224–231
97. Zheng MH, Chen J, Kirilak Y, Willers C, Xu J, Wood D (2005) Porcine small intestine submucosa (SIS) is not an acellular collagenous matrix and contains porcine DNA: possible implications in human implantation. J Biomed Mater Res B Appl Biomater 73:61–67
98. Zingg PO, Jost B, Sukthankar A, Buhler M, Pfirrmann CW, Gerber C (2007) Clinical and structural outcomes of nonoperative management of massive rotator cuff tears. J Bone Joint Surg Am 89:1928–1934