# Massive and Irreparable Rotator Cuff Tears: Defining the Problem

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## 1.1 Introduction

Massive and irreparable rotator cuff tears remain a major challenge in shoulder surgery [1]. Due to pain, loss of range of motion, and insufficient function, these tears significantly affect the patients' quality of daily living [1]. Representing up to 40% of all rotator cuff tears, massive tears are associated with persistent defects and poorer clinical outcomes [2, 3]. Imbalance of the force couples results in unstable kinematics of the glenohumeral joint, causing the remaining shoulder function to be sustained by a significantly increased compensatory deltoid force [4, 5].

This article tries to provide a structured overview about the biological challenges and biomechanical consequences of massive irreparable rotator cuff tears, as understanding of these fac-

tors is essential to initiate a differentiated therapeutic approach. Beginning with the different existing classification systems as well as initiation and progression of massive rotator cuff tears, the authors try to outline successively the biological problems including healing potential and tissue degeneration, followed by the main biomechanical problems. These mainly comprise the effects on tractive forces, shoulder function, glenohumeral joint centering and the development of osteoarthritis. In clinical practice, all of these factors have to be considered, in order to achieve satisfactory improvement in functional outcomes.

## 1.2 Structural Problem

### 1.2.1 Classifications of Massive Rotator Cuff Tears

Massive rotator cuff tears can be characterized by size, chronicity, and location. Regarding the tear size, different definitions exist. DeOrio and Cofield [6] defined massive tears as those whose greatest diameter exceeds 5 cm. Contrarily, Gerber et al. [7] characterized massive tears as those including complete tears of at least two tendons. This definition may show a more consistent correlation to the patients' function, prognosis, and outcome [5, 7, 8]. Considering chronicity, massive tears can be classified as acute, acute-on-chronic, and chronic tears [5]. Acute tears are relatively rare, commonly occurring

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after a traumatic event in younger patients [5]. In contrast, chronic massive tears are mostly observed in the elderly [5]. Regarding location, massive tears mostly follow the two distinct patterns of anterosuperior and posterosuperior tears [5].

### 1.2.2 Tear Initiation and Progression

Previous studies have proposed that degenerative rotator cuff tears start with the supraspinatus tendon, typically initiating at the anterior part of the humeral insertion near the biceps tendon, and propagate posteriorly over time [9–12]. In contrast, more recent studies have found that full-thickness as well as partial-thickness rotator cuff tears most commonly initiate at a location approximately 10–15 mm posterior to the biceps tendon, and may even begin with the infraspinatus tendon [10, 11, 13, 14]. An explanation for this finding is based on the "rotator crescent" concept, first described by Burkhart et al. [11, 15]. The rotator crescent is a term describing the thin, crescent-shaped rotator cuff sheet, which spans from the biceps tendon to the inferior border of the infraspinatus tendon, and is bound proximally by an arch-shaped thick bundle of fibres, called the "rotator cable" [15]. The rotator cable preserves the rotator crescent from stress through a "suspension bridge" configuration [15].

As people age, relative avascularity may lead to progressive thinning of the crescent, thus increasing dependence on the rotator cable [11, 15]. The location found by Kim et al. [11] 15 mm posterior to the biceps tendon is approximately at the center of the rotator crescent [15]. However, a recent MRI study located the initial tear site 5 mm more anterior (9–10 mm posterior to the biceps tendon) than as described by Kim et al. [10, 11]. This leads to the assumption that tears might propagate in both anterior and posterior directions [10]. Given the fact that the supraspinatus footprint is much smaller than previously believed, this location may be regarded as either the junction between the supraspinatus and infraspinatus, or being purely within the infraspinatus tendon [11, 16].

As torn tendons cannot participate in load distribution, the increasing tensile load on the remaining fibres can easily lead to tear propagation, particularly if the remaining tendon is of poor quality [17].

## 1.3 Biological Problem

### 1.3.1 Healing

The tear size can directly affect the clinical outcome and tendon healing [18–20]. A series of arthroscopic rotator cuff repairs have demonstrated that postoperative healing usually occurs between 71 and 89% of cases [19, 20]. However, this rate of tendon healing may drop to 47 or 50% in the treatment of massive rotator cuff tears [19, 20]. Even though hypovascularity has been hypothesized to facilitate tear initiation and limit biological healing after repair, the complexity of the healing process has not been fully understood [21].

The cells contributing to natural tendon healing originate from loose connective tissue surrounding the tendon fascicles and tendon body [22]. In response to the injury, these cells proliferate and migrate toward the tear site where they form collagenous healing tissue [22–24]. As the endogenous healing potential of the tendon seems to be limited, biologic augmentation techniques have recently garnered more and more attention, including the application of growth factors, platelet concentrates, or mesenchymal stem cells (MSCs) [25, 26]. Despite bone marrow being the tradiitional source for MSCs for biologic augmentation of tendon injuries, recent studies have highlighted subacromial bursal tissue being a source of MSCs, demonstrating superior proliferation potential, tissue engraftment, and survival [22, 26–29].

### 1.3.2 Atrophy, Fatty Infiltration, Retraction, and Loss of Elasticity

In addition to tear propagation, the process of atrophy, fibrosis, and fatty infiltration may occur in the rotator cuff tendon, as well as in the associated muscle belly over time (Fig. 1.1) [7, 20,
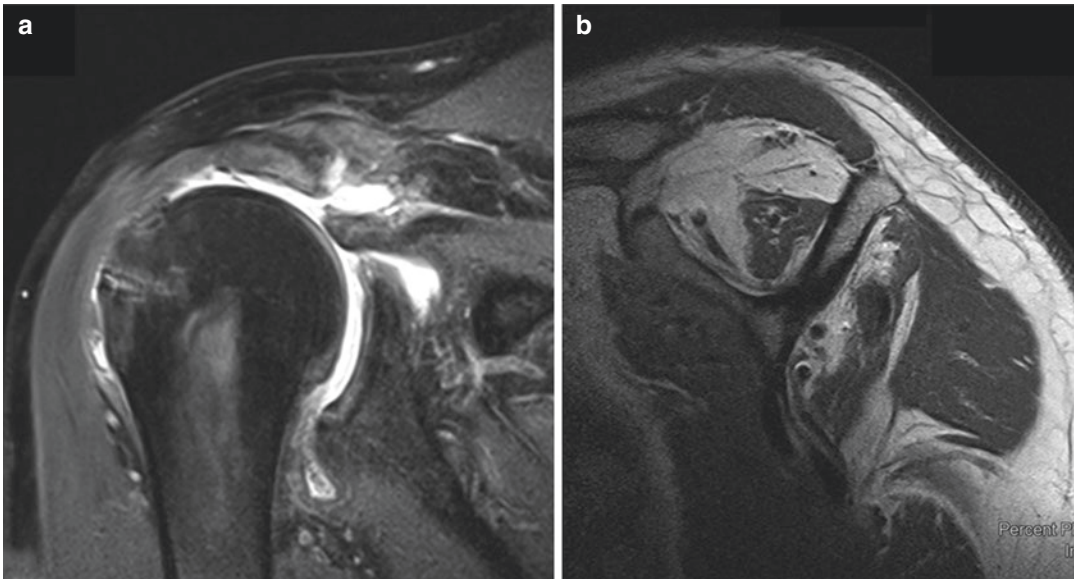
**Fig. 1.1** MRI scan demonstrating a massive re-tear of the rotator cuff tendons with retraction, atrophy, and fatty infiltration. (**a**) Coronal view and (**b**) sagittal view

30]. Moreover, these tears often cause the tissue to become less compliant and stiffer [20, 30]. Particularly, in combination with tissue atrophy or fatty infiltration, this may result in severe tendon retraction [20, 30]. A widely retracted tendon margin coupled with poor tissue quality makes surgical mobilization difficult and sometimes impossible (Fig. 1.2) [20].

Muscle atrophy and fatty infiltration have been reported to be independent factors predicting outcomes and success rate after rotator cuff repair [31]. As tears of the rotator cuff result in mechanical unloading and denervation due to suprascapular nerve injury, consistent pathological changes can occur in the muscles' myotubes [32–34]. This may lead to alterations in the central molecular pathways, which regulate muscle atrophy and hypertrophy through mechanical load signaling [32–34].

A cell subpopulation of interstitial pluripotent stem cells, named fibro-adipo-progenitor cells (FAPs) and resident in muscle tissue, has been identified to be the cellular source of fatty infiltration [35, 36]. As shown in a mouse model, FAPs proliferate and differentiate into cells primarily expressing fat genes and cellular markers of adipogenesis, after inducing cuff injury [35, 36].
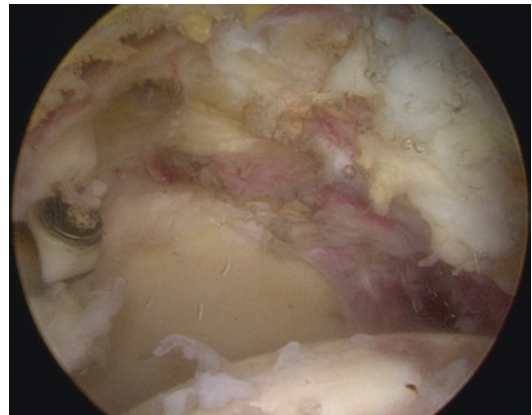


**Fig. 1.2** Arthroscopic view of a large, retracted massive rotator cuff tear with concomitant tissue degeneration

Large, retracted tears have also been shown to cause traction on the suprascapular nerve, and may contribute to the progression of atrophy and fatty infiltration of the supraspinatus and infraspinatus muscles [17]. Moreover, the tear initiation location found by Kim et al. [11] may explain why fatty degeneration of the infraspinatus is seen in some patients with a presumed isolated tear of the supraspinatus tendon, highlighting the need to assess its integrity.

## 1.4 Biomechanical Problem

### 1.4.1 Tractive Forces

Coordinated action between the rotator cuff and deltoid muscles is essential for a sufficient glenohumeral abduction motion [37]. As the anterior and middle deltoid show preferential muscle activity and loading from 30° to 90° of glenohumeral abduction, the supraspinatus is the dominant muscle during the first 30°, and therefore the main initiator of abduction [37, 38]. Rotator cuff tears may lead to kinematic alterations, potentially causing a significant change in the biomechanical synergy between deltoid and rotator cuff muscles [39]. As the cuff tear size propagates posteriorly, considerably greater amounts of force are placed upon the middle portion of the deltoid, showing a major increase between 10° and 45° of abduction [4, 40, 41]. At the same time, the mechanical advantage of the deltoid may be disrupted due to loss of balanced concavity-compression and superior translation caused by tear progression [4]. This results in greater forces required to maintain joint stability and decreased abduction capability [4, 41, 42].

A recent biomechanical study highlighted the required compensatory deltoid function to compensate for abduction motion loss in the presence of simulated rotator cuff tears [4]. Anterosuperior (combined supraspinatus and subscapularis) tears resulted in the largest loss in glenohumeral abduction motion, despite the greatest increase in deltoid force [4]. On the other hand, isolated subscapularis tears increased the anterior deltoid force, compensating for the loss of anterior joint compression without a reduction in abduction [4].

### 1.4.2 Shoulder Function and Pseudoparalysis

The rotator cuff muscles are important contributors to a smooth glenohumeral motion and sufficient joint stability [17]. Acting as force couples, they collaborate to stabilize the inherently unstable glenohumeral joint [17]. The deltoid and the inferior portion of the rotator cuff act as the coronal force couple, compressing the humeral head to the glenoid in abduction [43]. Subscapularis and infraspinatus/teres minor represent the axial force couple, providing a fulcrum for the actions of the deltoid and supraspinatus, which is essential to maintain joint stability by a compressive joint reaction force in the axial plane [17, 37, 43].

Massive rotator cuff tears may disrupt these force couples resulting in superior migration of the humeral head and dysfunction of the shoulder (Fig. 1.3) [5, 17]. The importance of the force couples was highlighted by introducing the "suspension bridge" concept [43]. Accordingly, shoulder function may be maintained in isolated supraspinatus tears due to intact force couples [43]. However, as tears propagate into the anterior or posterior cuff direction, force coupling is disturbed, resulting in unstable kinematics and loss of function [43].

In addition, instability of the glenohumeral joint results in increased internal rotation in the setting of posterosuperior tears, external rotation in anterosuperior tears, and the total rotational range of motion in all abduction angles [44]. To maintain normal kinematics in the presence of massive cuff tears, greater forces by both the deltoid and the corresponding force couple muscle



**Fig. 1.3** X-ray demonstrating the superior migration of the humeral head

are required to achieve a coordinated abduction motion [4, 40].

Due to these kinematic changes, pseudoparalysis of the shoulder may occur (Fig. 1.4). The most common definition is active elevation less than 90° with full passive elevation [1, 45–47]. Risk factors are considered to be disruption of the entire subscapularis or of the three rotator cuff muscles [45]. However, recently it has been reported that pseudoparalysis should rather be described as no active elevation with maintained passive elevation of chronic nature, usually with anterior–superior escape and being refractory following an injection [1]. This definition may be more adequate, as pseudoparalysis is often confused with pain [1]. Therefore, pain should be ruled out as a cause of apparent pseudoparalysis, since patients may benefit from a pain-relieving treatment alone [1]. Sometimes, an injection of lidocaine for pain elimination will clarify the diagnosis in the face of a massive rotator cuff tear [1].

### 1.4.3   Decentralization, Glenohumeral Joint Pressure, and Osteoarthritis

Sufficient function of the rotator cuff muscles is essential to ensure glenohumeral stability through the concavity compression principle [48, 49]. Loss of rotator cuff integrity may significantly alter the joint-reaction forces, which are required to maintain glenohumeral stability [48, 49]. Dysfunction of the infraspinatus and subscapularis may lead to superior humeral head



**Fig. 1.4** Patient with severe pseudoparalysis of the right cuff-deficient shoulder

translation and joint instability by displacing the glenoidal contact point superiorly [4, 41].

The abnormal joint loading due to rotator cuff insufficiency may cause various erosion patterns, frequently seen in type B glenoids of osteoarthritic patients [50, 51]. Recent literature suggests that this wear pattern is not axisymmetric to the superoinferior axis of the glenoid, but rather orientated in the posteroinferior region [50, 51]. Over time, these erosion patterns may lead to significant glenoid bone loss, presenting a major challenge in reverse shoulder arthroplasty [50–52]. However, three-dimensional reconstruction has allowed further analysis of glenoid erosion patterns. This is much needed, since the two-dimensional CT images inaccurately represent the wear pattern in osteoarthritic glenoids [51]. Unfortunately, it still remains uncertain if osteoarthritis results in altered kinematics and subluxation, or if the changed kinematics with subluxation is instigating this inflammatory disease [51].

Finally, massive cuff tears may lead to cuff tear arthropathy (CTA), which is defined as muscle degeneration, including fatty infiltration and atrophy, along with bony alterations, such as humeral head erosion and acetabularization of the acromion [53]. The underlying pathway may be induced by a massive cuff tear with anterosuperior escape, followed by a mechanical conflict between the humeral head and the superior glenoid and acromion [54]. In addition to the collapse of cartilage and bony structures, enzymes may be released that impair the surrounding tissue, thus leading to pain and limited shoulder function [53]. Maintenance of a sufficient rotator cuff function has been shown to be vital to delay the development of glenohumeral arthritis, highlighting the necessity of a good repair technique [55]. However, in the presence of severe CTA, these repair techniques may be infeasible, calling for reverse total shoulder arthroplasty instead.

## 1.5   Conclusion

Due to the combination of impaired biological healing potential and joint affecting biomechanical changes, massive irreparable rotator cuff tears

**Table 1.1** Key factors

| Nature of the problem | Key factors |
|---|---|
| Structural | • Most massive tears follow distinct patterns (antero- and posterosuperior)<br>• Progressive thinning of the rotator crescent facilitates tear initiation<br>• Increased tensile load on remaining fibres leads to tear progression in both anterior and posterior directions |
| Biological | • Hypovascularity limits biological healing potential<br>• Tissue degeneration includes atrophy, fatty infiltration, retraction, and loss of elasticity making surgical repair difficult |
| Biomechanical | • Mechanical advantage of the deltoid muscle is comprised of higher tractive forces and loss of balanced concavity-compression<br>• Disruption of force couples leads to superior humeral head migration and shoulder dysfunction (pseudoparalysis)<br>• Glenohumeral instability causes humeral head decentralization<br>• Glenoidal erosion patterns may progress to severe CTA |

remain a major challenge in shoulder surgery. For the treatment of these patients and defining the underlying problems (Table 1.1), the interaction of biological and biomechanical pathomechanisms has to be considered. As biological healing may be impaired by hypovascularity as well as tissue degeneration including atrophy, fatty infiltration, and tendon retraction, concomitant biomechanical alterations of glenohumeral joint kinematics may result in shoulder dysfunction and lead to the development of cuff tear arthropathy in the long term.

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