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## Role of Biomechanics in Rotator Cuff Pathology: North American Perspective

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### Abstract

The biomechanical properties of, and mechanical environment surrounding, the rotator cuff continue to be a research area of great interest as injury and failure of these tendons are among the most common of shoulder pathologies, with incidence increasing in older populations. Recent literature has refined our knowledge of the anatomy, the role of the various tendons in movement and shoulder stability, and structural and material properties of the human rotator cuff. Furthermore, animal models have characterized the effects of mechanical loading on rotator cuff properties, the biomechanical interactions among cuff tendons in uninjured and deficient rotator cuffs have been described, and more recent imaging studies have provided novel insights into the function of the rotator cuff *in vivo*. Research to advance our understanding of the biomechanical factors contributing to rotator cuff disease is needed, as the etiology, prognostic indicators, and reasons for treatment failure are poorly understood. We summarize published biomechanical literature on the rotator cuff to provide a current perspective on potential mechanisms involved in cuff pathology.

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### Rotator Cuff Failure in Biomechanical Literature

Although the epidemiology of rotator cuff tears is not well established, rotator cuff tears, either symptomatic or asymptomatic, approach a prevalence of 7–30% [1–12]. Moreover, rotator cuff tears pose a challenging problem with regard to clinical management and diagnosis as they can be symptomatic or asymptomatic, and can be accompanied by pain, loss of muscle strength, and range of motion deficits [1, 13–18]. In contrast to the reported dissociation between symptoms and the presence of rotator cuff tears, cadaveric and animal models have shown an alteration in the biomechanics of the shoulder in the presence of tears [8, 19–23].

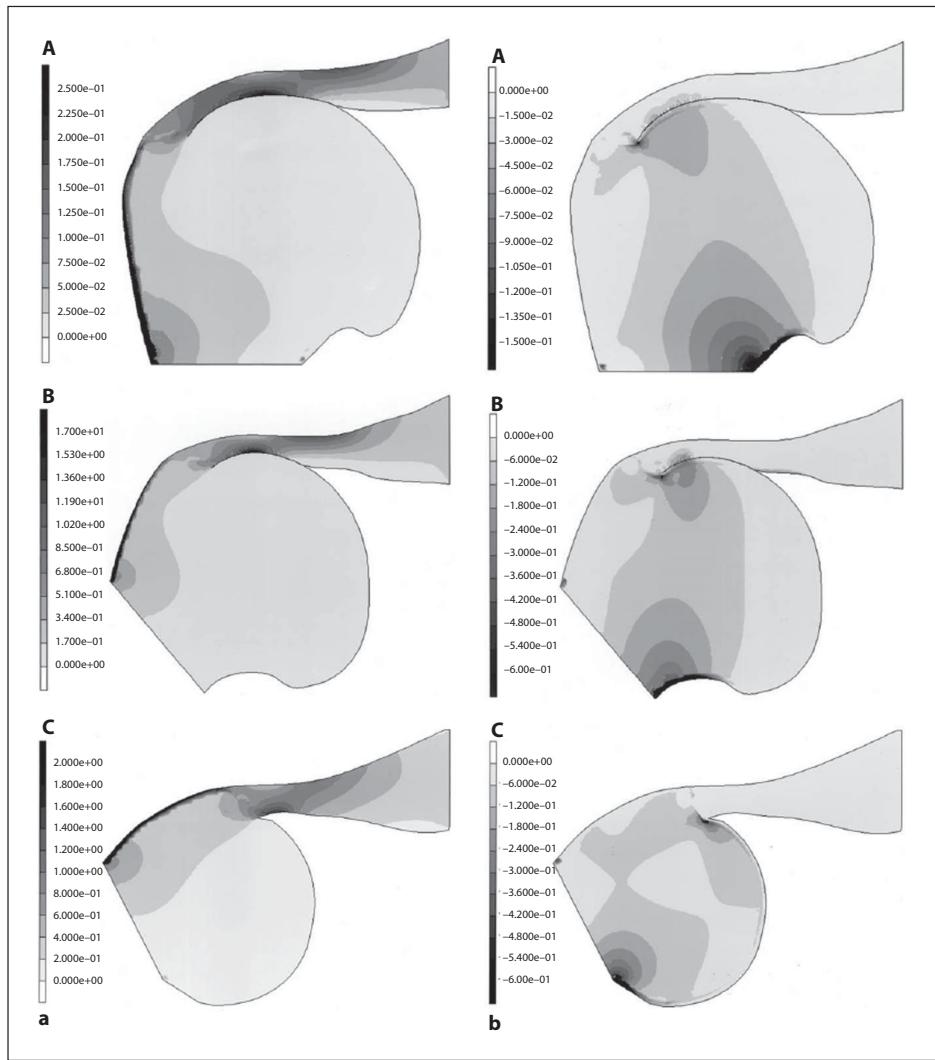
In a study of the spatially varying material properties of the human supraspinatus tendon, Lake et al. [24] showed a significant difference in the tensile modulus of the posterior portion of the tendon when compared to the medial and anterior portions.

Despite these differences, histological scores of degeneration did not correlate to their biomechanical findings. However, a study of normal versus torn supraspinatus tendons found a significant increase in infiltration of mast cells and macrophages in torn tendons with mild to moderate tendinopathy [25]. This finding contradicts earlier studies that show degeneration of the tendon rather than infiltrate within the tendon [26, 27]. Interestingly, there is a greater cellular and vascular response in small tears of the rotator cuff, while large and massive tears are primarily degenerative in nature [28]. However, it is unclear whether the latter observations are applicable to both partial and full-thickness tears, and whether these findings vary with tear location.

There exists a stress concentration within the articular side of the supraspinatus tendon in a finite element model [29]. This finding has since been corroborated in both physiologic [30–32] and pathologic states [32]. Sano et al. [30] used finite element analysis derived from MRI and histologic data to model the biomechanical properties of intact tendon (fig. 1). They then modeled an articular, bursal, and intratendinous tear and show increased stress concentration at these levels [32]. Despite biomechanical data which suggest strain gradients within the rotator cuff tendon [20, 33], to date, clinical evidence does not demonstrate failure of the tendon proper in a specific location as the primary mechanism of failure. In revision of rotator cuff repairs, the anchor or the suture tendon interface is commonly the focal point of failure [34–36]. In tests of native humerus-tendon-muscle constructs in cadavera, insertion site failure or musculotendinous junction failure occurs before rotator cuff tendon tear [33]. This evidence suggests that, under conditions of uniaxial tension applied along the tendon fibers, in its uninjured state, the rotator cuff tendon proper is biomechanically more robust than its bony insertion on the lateral side and its muscular junction on the medial side. Sano et al. [37] showed a negative correlation between the ultimate tensile strength of the supraspinatus tendon and the tendons' histologic properties at the bony insertion. Sano also concludes that microscopic degeneration is more common at the insertion than in the middle portion (from lateral at insertion to medial at musculotendinous junction) of the supraspinatus. Finally, Fukuda et al. [38] postulated that intratendinous mechanical shear forces may be responsible for early tendon defects. Itoi et al. [39] divided the supraspinatus tendon into thirds and noted that, despite a similar modulus of elasticity between the articular and bursal sides, the anterior portion of the tendon exhibited a significantly higher elastic modulus than that of the middle and posterior thirds.

### **Impingement: Biomechanical Considerations**

Neer and Poppen first described subacromial impingement in an intraoperative study of 400 patients with rotator cuff tears [40]. They postulated that the etiology of the rotator cuff tears in 95% of their patients was subacromial impingement on the cuff



**Fig. 1.** Finite element results for tensile (a) and compressive (b) stress at abduction angles of 0° (A), 30° (B), and 60° (C). Darker color denotes greater stress. Reprinted with permission from Elsevier [30].

by the anterior third of the acromion [41]. Subsequently, the shape of the acromion [42], the presence of arthritic changes in the acromioclavicular joint [43], and spurs on the acromion and distal clavicle [44] have been found to result in narrowing of the subacromial space.

It is likely that subacromial contact in and of itself is not detrimental, but a narrowed acromiohumeral distance predisposes an individual to rotator cuff disease [41,

42, 45]. This point was most recently made in a meta-analysis of *in vivo* imaging of the rotator cuff with ultrasound [45]. Seitz et al. [45] identified five contemporary studies that compared the acromiohumeral space in patients with and without diagnosed rotator cuff tears and impingement. They concluded that patients with full-thickness rotator cuff tears have a significantly smaller acromiohumeral space than those without pathology.

The anatomic shape of the acromion is associated with the severity of rotator cuff disease with type III or hooked acromial shape having the highest penchant for pathology [42, 46, 47]. The slope of the acromion is another measure used to assess acromial shape, and has also been implicated in symptomatic impingement [48] and increased tendon compression [49, 50].

However, it is unclear whether impingement induces cuff damage or whether weakness, dysfunction, and fatigue or overuse of the rotator cuff lead to subacromial impingement (e.g., via the resulting superior humeral translation). Recent cadaveric evidence suggests that non-pathologic contact pressure between 0.04 and 0.11 MPa from 0 to 100° of flexion exists beneath the coracoacromial arch. Similarly, acromial contact pressure reached 0.43 MPa in flexion [51]. Contact occurred in otherwise grossly intact tendons devoid of pathologic changes. Although the authors did not present histologic or biochemical results for the tendons, Yamamoto et al. [51] suggest that contact of the rotator cuff tendons with the coracoacromial arch is not necessarily a pathologic phenomenon. A study evaluating the effect of acromioplasty on glenohumeral translation and rotator cuff contact pressure, even with complete resection of the coracoacromial ligament, showed a pressure of at least 0.02 MPa on the rotator cuff at all times [52].

Mechanical overuse of the supraspinatus was explicitly tested by Soslowsky et al. [53] by examining tendon alterations in a rat model of decline treadmill running. The tendon in their overuse model exhibited an increased cross-sectional area, hypercellularity, and collagen disorganization. Biomechanically, maximum stress and elastic modulus were significantly lower in the overuse group when compared to cage control rats. Impingement of the tendons was not included in the study design, and yet these tendons showed signs of biomechanical and cellular degradation [53].

Schneeberger et al. [54] tested an alternative hypothesis of rotator cuff pathology in an *in vivo* rat model by producing impingement on the supraspinatus with surgically implanted plates and then analyzing the tendon grossly and histologically. They found that the tendons showed signs of bursal sided degeneration in every instance of simulated impingement, demonstrating that impingement may indeed have a detrimental effect on rotator cuff tendons.

Soslowsky et al. [55] used a combination of the aforementioned approaches by subjecting rats to the overuse model with the addition of simulated subacromial impingement with the use of an Achilles tendon allograft. They examined three groups of rats: an overuse, an impingement, and an overuse + impingement group. The authors found that both impingement and overuse had detrimental effects on



the supraspinatus. In addition, the combined group showed a significant increase in cross-sectional area and a significant decrease in maximum stress and elastic modulus when compared to either of the individual groups.

### **Rotator Cuff Pathology: Age and Symptoms**

A prospective clinical study of 411 patients found a tear in 23% of subjects with an increase in incidence that correlated directly with age. Specifically, 38% (52 of 136) of asymptomatic patients older than 70 had full-thickness tears appreciable on ultrasound [12]. This study along with MRI findings by Sher et al. [56] in asymptomatic patients of a younger cohort, suggest that tears in the rotator cuff, especially with advanced age, could be classified as ‘normal aging’. Moosmayer et al. [1] found the prevalence of rotator cuff tears to be 7.6% overall, and 15% in ages of 70–79, a smaller prevalence overall yet also an increase with age. Neer et al. [57] described three stages of rotator cuff lesions characterized by age: <25 years for stage I, between 25 and 40 for stage II, and stage III for those >40. The stages correspond to the most common characteristics of impingement, with stage three including rotator cuff tears. Although heavily biased toward impingement as the primary cause of rotator cuff tears, Neer’s stages suggest that age is a component to rotator cuff pathology.

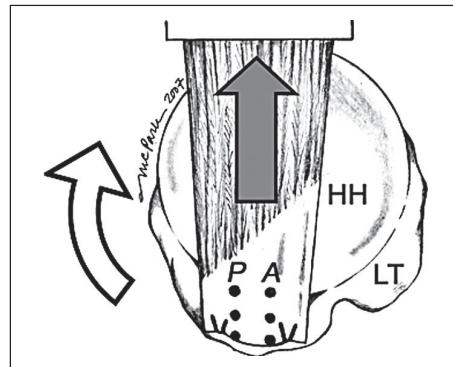
As a microscopic correlate, Kumagai et al. [58] found that elderly cadavera without tendon tears (52–90 years) were characterized by calcification, fibrovascular proliferation and microtears in the tendon, and patients in a younger cohort did not demonstrate these histologic findings.

Despite the increasing prevalence of RC tears with age, clinical symptoms do not always accompany a tear [2, 59, 60]. In fact, across all ages, there is still debate as to what factors cause symptoms. Based on imaging studies of symptomatic and asymptomatic patients with rotator cuff tears, size of tear [2, 59, 60] and fatty infiltration [60] are indicators of symptomatic tears.

Karas et al. [3] recently reviewed the biomechanical effects of rotator cuff tears in cadaveric models. The *in vivo* biomechanics of the shoulder, however, whether in symptomatic or asymptomatic patients, are altered in the presence of a rotator cuff tear based on electromyography and kinematics [61, 62]. In confirmed rotator cuff tears, muscle activation is increased when compared with non-pathologic shoulders. In asymptomatic subjects, the subscapularis had increased activity whereas, in symptomatic subjects, the rotator cuff muscles associated with torn tendons had increased activity. Yamaguchi et al. [62] analyzed the three-dimensional kinematics of the glenohumeral joint in patients with symptomatic and asymptomatic rotator cuff pathology and found statistically significant humeral head migration in both groups, which suggests that symptoms do not result from a change in kinematics.

Results from translational scientific studies on the rotator cuff have led to an improved understanding of potential biomechanical mechanisms of cuff injury. The

**Fig. 2.** Schematic of custom testing configuration allowing for dynamic humeral external rotation during cyclic loading of the supraspinatus tendon. The straight arrow represents the vector for tendon loading. The curved arrow represents the direction of rotation. A = Anterior tendon region; P = posterior tendon region; LT = lesser tuberosity; HH = humeral head. Modified from Park et al. [64].



reliability of ongoing and future work to more closely simulate clinically observed pathology will not only provide further insight into the etiology of rotator cuff disease, but also facilitate potential approaches for injury prevention, diagnosis and treatment. For example, it is possible that experimental methods such as applying fatigue loading until failure, or utilizing more complex loading directions/modes (e.g., superposing impingement with tensile loading of the tendon, or loading the tendon in more physiologic directions [63, 64]) may be required in cadaveric studies to produce pathology more consistent with clinical and/or imaging results (fig. 2). Furthermore, refinements of recent technological advancements such as arthroscopically insertable force probes [10, 65] for in vivo measurement of tendon forces as well as imaging methods for in vivo kinematic studies [9, 66] and soft tissue deformation analyses [67] will likely provide critical insight into the biomechanical environment of rotator cuff tendon and surrounding soft tissue stabilizers. Collectively, advances in our understanding of the in vivo, temporal biomechanical function of the rotator cuff has strong implications for rehabilitation protocols and guidelines for the design of tissue engineered constructs for healing and repair of RC defects.

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