5 Etiopathogenesis of Rotator Cuff Arthropathy

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5.1 Definition and Historical Review

In the early 1980s, Neer et al. [1, 2] have coined the term "rotator cuff arthropathy" to indicate a nosological condition characterized by arthritic degeneration of the glenohumeral joint consequent to the massive posterosuperior rotator cuff tear. However, more than a century earlier, Adams [3], in his book on rheumatic gout, and Smith [4, 5] had described cases of shoulder arthropathy characterized by erosion of the upper portion of the humeral head, of the acromion, of the distal third of the clavicle, and of the rotator cuff tear. Codman [6], in his monograph published in 1934, had described the case of a woman, 51 years old, whose shoulder underwent rotator cuff tear, glenohumeral arthropathy, loose bodies, and swelling for the abundant articular synovial fluid.

Further papers have not been published until the end of the 1950s when Galmiche and Deshayes [7], Burman et al. [8], Banna and Hume [9], Shepard [10], and Snook [11] reported a total of 30 cases of shoulder arthropathies, some of them with the characteristics of cuff tear arthropathy.

In 1968, De Seze [12] described the hemorrhagic shoulder of three elderly women whose clinical (blood streaked recurrent effusion; rotator cuff tear) and radiographical (severe degenerative glenohumeral humeral arthritis) characteristics suggested a rotator cuff arthropathy. One year later, Bauduin and Famaey [13] described an analogous case.

Jensen et al. [14], in a prestigious publication of 1999, described the three main clinical and radiographical characteristics of the cuff arthropathy: (a) massive tear of the rotator cuff, associated with shoulder pain, supra and infraspinatus atrophy, and loss of motion (Fig. 5.1a–c); (b) degenerative changes of the glenohumeral joint (Fig. 5.2a, b); (c) upward migration of the humeral head observable on AP view (Fig. 5.3a). Humeral head collapse (Fig. 5.3b), erosive changes of superior glenoid or acromion, periarticular soft tissue calcifications, and subdeltoid effusion are other possible features that may be present [15].

5.2 Etiopathogenesis

Mechanical theory. Neer et al. [2] hypothesized that mechanical factors were at the origin of cuff arthropathy. According to this theory, loss of downward force performed by a healthy rotator

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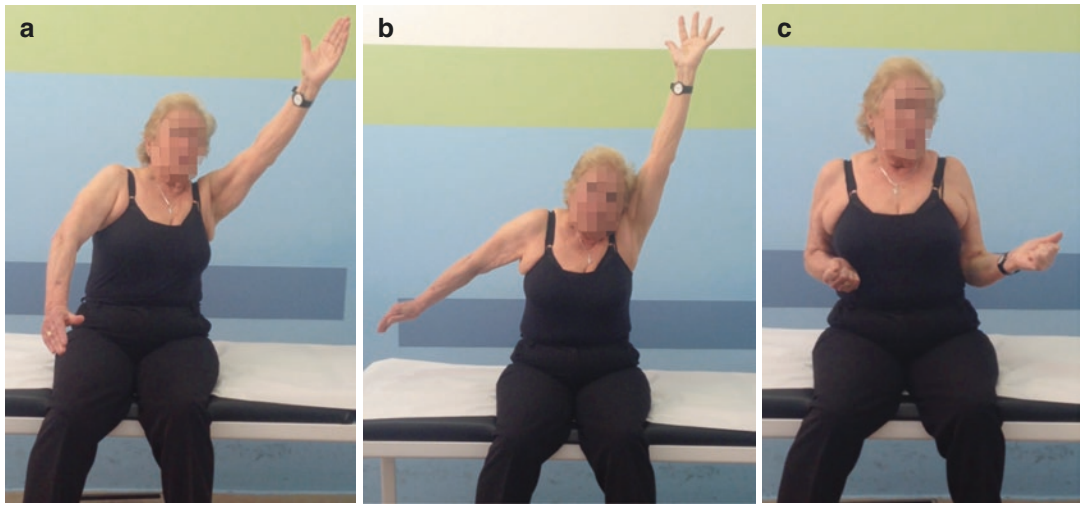

Fig. 5.1 (**a**–**c**) Decrease in range of motion in a 75-year-old female patient with cuff tear arthropathy

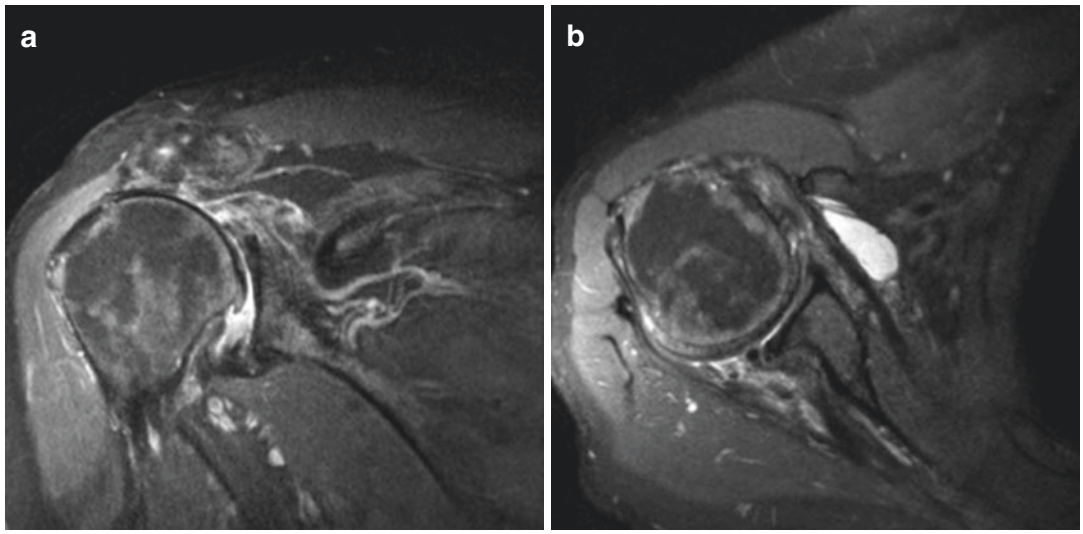

Fig. 5.2 MRI of a right shoulder of a 77-year-old male patient with Hamada 3 cuff tear arthropathy. (**a**) Coronal T2 fat suppressed FSE. Acromiohumeral distance <5 mm with acetabularization of acromion. (**b**) Axial PD fat suppressed FSE: Walch A1 glenoid morphology (humeral head centered with minimal erosion)

cuff on the humeral head would result in a superior migration of the humerus. This might facilitate an erosion of the superior surface of the glenoid and of the anteroinferior aspect of the acromion. In addition, the upward migration of the humeral head could cause a joint instability, an eccentric work of the humeral head, and, consequently, a premature wear of the articular cartilage in the areas of higher glenohumeral compression. Since 21 of the 26 patients cited in the paper had the rupture of the long head of the biceps tendon, Neer thought that this injury would help the upward migration of the humeral head.

Burkhart's hypothesis [16] seems to support the mechanical theory. The author believes that the healthy inferior portion of the rotator cuff (below the center of rotation) creates a moment that must balance the deltoid moment (force coupling). Furthermore, the subscapularis is anteriorly balanced against the infraspinatus and teres minor posteriorly. Uncoupling of the essential force couples results in anterior superior

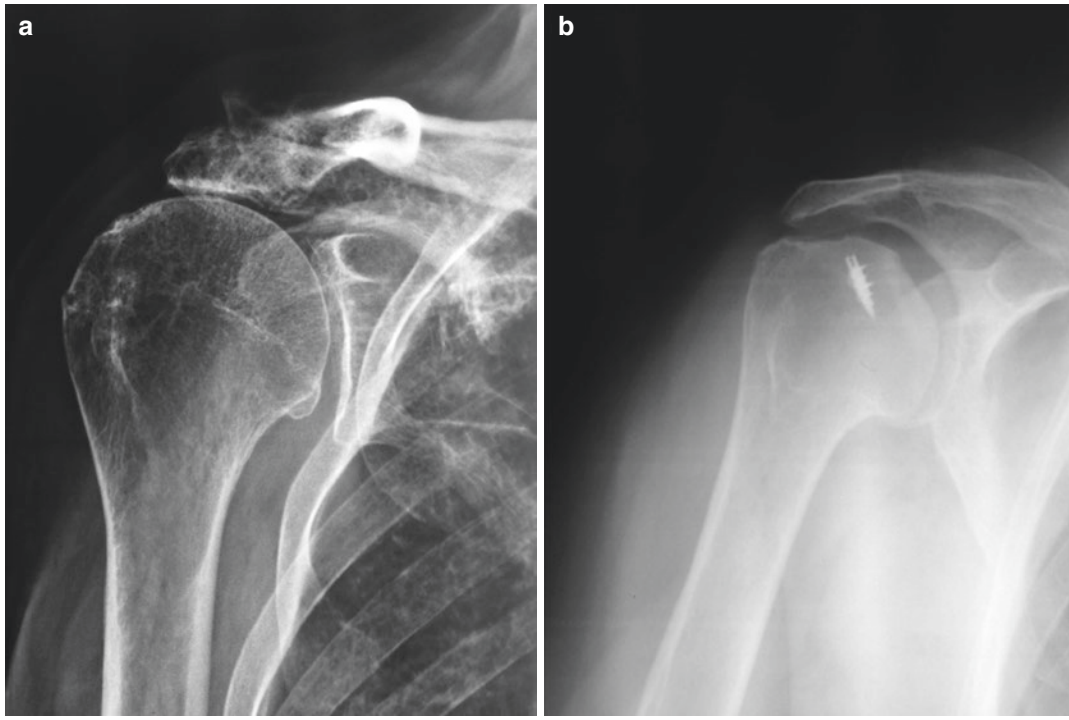

Fig. 5.3 (**a**) True AP X-ray view of a right shoulder: Hamada 3 cuff tear arthropathy. (**b**) AP X-ray view of a right shoulder: Hamada 5 cuff tear arthropathy bony destruction-humeral head collapse after a cuff repair failure

translation of the humeral head with attempted elevation of the shoulder.

In 1997, Collins and Harryman [17] hypothesized that cuff arthropathy was initially due to supraspinatus tear and later to the infraspinatus lesion; the complex tendon tear would cause the upward migration of the humeral head and, consequently, the contact of articular cartilage of the humeral head against the anteroinferior margin of the acromion. Cartilage fragmentation results in particulate debris, which causes synovial thickening and effusion as well as calcium phosphate crystal formation. The enzymatic response to the crystals furthers the damage of the articular surfaces.

Concavity-compression mechanism, suggested by Hurov [18], further corroborates the mechanical theory. According to the author, the healthy cuff compresses the convexity of the humeral head against the pseudo-concavity of the glenoid; therefore, the cuff, with other periscapular muscles, would act as an important dynamic stabilizer of the joint. This action may be even more important in the presence of severe laxity of the static stabilizers of the shoulder (capsule, labrum, and glenohumeral ligaments).

Oh et al. [19] identified that critical tear sizes responsible for disrupted joint kinematics are those with full-thickness supraspinatus tears and 50% detachment of the infraspinatus.

Nutritional theory. Neer et al. [1, 2] have also suggested that the osteoarthritis could depend on the loss of the "water tight" effect (loss of negative pressure normally existing inside the shoulder joint in normal conditions) due to the cuff tear.

This would cause dispersion of synovial fluid, normally contained in the joint, in the subacromial space. The dispersion would make the diffusion of synovial fluid into the joint cartilage difficult; consequently, the cartilage would be poorly nourished and would easily run into atrophy. Furthermore, diffusion of the fluid into the cartilage should be further hindered by the decrease in range of motion caused by the shoulder pain due to the cuff tear (loss of water and mucopolysaccharides content). In addition, decrease in mobility, resulting in pain, would lead the subchondral bone to be osteoporotic and more exposed to possible collapse.

It is known that cytokine and catabolic enzyme concentration increases in the early phases of osteoarthritis. Many studies have also proved that the rotator cuff tear leads to an increased production of interleukin 1β and TNF, which helps to explain the presence of pain and inflammation. It was also noted that the production of many cartilage matrix-specific matrix metalloproteinases (MMPs) increased, including MMP-1, MMP-2, MMP-3, MMP-8, and MMP-13 [20, 21]. The presence of MMP-3 is important because it is implicated in the proteolytic activation of the other MMPs. Yoshihara et al. [22] observed that there is a correlation between the concentration of these cytokines, collagenases, and aggrecanases and accelerated cartilage degeneration after a cuff tear.

These observations redimension the nutritional theory in the genesis of cuff tear arthropathy. In fact, with the evacuation of a part of the synovial fluid through the tendon lesion, inflammation factors and proteolytic enzymes should be removed, and, thus, health status of articular cartilage should be preserved.

In 2012, Reuter et al. [23] sonographically assessed the articular surface of the glenohumeral joint in rats with a rotator cuff tear and observed a thickness decrease in the cartilage. Kramer et al. [24] histologically studied the glenohumeral cartilage of rats, respectively, submitted to detachment of the posterosuperior rotator cuff and to suprascapular nerve root transection passing through the trapezoid (joint capsule was kept intact). The animals were killed 12 weeks after surgery. In the first case, if there had been degenerative changes of the cartilage, it would have been attributed, in accordance with Neer's hypotheses [1, 2], to the altered mechanical loading and to the nutritional theory, instead, in the second case, only to the mechanical hypothesis. Surprisingly, the amount of cartilage degeneration was similar between the groups. This result suggests that aberrant mechanical forces are the primary causes of articular cartilage degeneration in the setting of cuff tear arthropathy.

Crystalline-induced arthritis of the shoulder theory. In orthopedic literature, almost simultaneously to Neer's hypotheses, a nosologic entity similar to the cuff arthropathy has been described: the "Milwaukee syndrome" [25]. Although it is responsible for a clinical condition similar to that of the cuff arthropathy, this disease has been attributed to the presence in the synovial fluid of basic calcium phosphate crystals encapsulated into microspheroids without apparent inflammatory cell response. Indeed an altered capsular degenerated cartilage and synovium, possibly with a macrophage response and subsequent release of collagenase and neutral proteases, are associated with this condition, resulting in the attack and subsequent destruction of the joint.

In 1985, Dieppe and Watt [26] noted that basic calcium phosphate crystals could be found in arthritic and neuropathic joints and in apparently healthy joints of elderly subjects. In addition, the apatite crystals are found especially in the most destructive atrophic situations. Therefore, the authors hypothesized that the crystals are produced by the processes that are secondary to joint degeneration. This hypothesis redimensions the inflammatory theory and suggests that the syndrome is a form of cuff arthropathy.

Autoimmune rheumatic diseases. Cuff arthropathy could be considered an autoimmune rheumatic disease. As well as for scleroderma or systemic lupus erythematous, patients with cuff tear arthropathy are frequently females. No study has ever confirmed this hypothesis. We are conducting a study to verify the reliability of this hypothesis; however, available data do not allow us to formulate conclusions.

Idiopathic theory. It is possible that the cuff arthropathy is the result of a fortuitous coincidence between rotator cuff tear, which is frequently found in elderly patients [27, 28], and idiopathic glenohumeral arthropathy. In other words, arthropathy would occur regardless of cuff tear.

Upward migration of the humeral head consequent to the cuff tear would only be responsible

for the fast evolution of the arthritic process, and it would only cause a more precocious wear of the upper portion of the glenoid surface. If this hypothesis is correct, the cuff arthropathy should not have a clear preference for sex, and patients should have an average age similar to that of patients with concentric arthropathy; instead, the cuff arthropathy is predominantly found in females and in older patients.

Theory related to joint laxity. Since cuff tear arthropathy and youth joint laxity are significantly more frequent in females, we hypothesized that these two conditions are associated with each other. If the rotator cuff tear occurs in patients who have/had joint laxity, it is possible that the involved shoulder could develop a severe static instability that might be responsible for a premature wear of the cartilage of the superior glenoid. This assumption justifies the evident difference in the prevalence of cuff arthropathy due to gender.

In order to verify this theory, 133 consecutive patients with glenohumeral osteoarthritis [(48M, 85F; mean age (SD): 72.32 (7.05)] were divided into 2 groups: Group I (patients with CTA) and Group II (patients with concentric shoulder arthropathy) composed of 71 (22M–49F) and 62 (26M–36F) patients, respectively. The presence of current or previous joints hypermobility in all participants was assessed by two standardized methodologies: the Beighton criteria/score and a 5-item self-report questionnaire [29, 30]. The questionnaire investigates, using major and minor criteria, patient's ability to perform uncommon activities, the presence of joint diseases, or the tendency to dislocation.

Beighton criteria led to a diagnosis of joint hypermobility in 16 patients (22.5%) in Group I and in 15 patients (24.2%) in Group II. According to the 5-item self-report questionnaire, juvenile joint laxity was diagnosed in 11 (15.5%) and 12 (19.4%) patients belonging to Groups I and II, respectively. No significant association between the two groups and both Beighton criteria [$\chi^2(1) = 0.051$, $p = 0.82$] and 5-item self-report questionnaire [$\chi^2(1) = 0.67$, $p = 0.41$] was found.

Our data excluded this possible correlation. In fact, surprisingly, in the two groups, percentage of patients who, in juvenile period, have been considered subjects with joint hyperlaxity was the same (Group I, 16 pts. on 71 = 22:53%; Group II, 14 pts. on 62 = 22:58%). Therefore, it is presumable that hyperlaxity condition runs out before the onset of tendon rupture and cannot enhance the joint instability consequent to cuff tear.

5.3 Clinical Presentation

Generally, patients with cuff tear arthropathy are older than 65. They refer shoulder pain; rarely pain intensity is marked. Patients typically are women with shoulder symptoms of long duration. The dominant side is most commonly affected. Usually, pain is distributed in the anterolateral region of the shoulder; rarely it is also at the neck base; it does not extend beyond the elbow; scapular region is not interested; pain is not accompanied by paresthesia. The pain characteristically interferes with sleep and intensifies with activity.

Many patients experience audible crepitus. When these are present, it is easy to evocate them during the Jobe or the Full can test maneuvers.

In thin patients, it is sometimes possible to observe shoulder profile deformity, because of the humeral head upward migration. Occasionally the shoulder is swollen by the presence of abundant synovial fluid that is spread in the subacromial space and glenohumeral and acromioclavicular joints.

Atrophy of infraspinatus and supraspinatus muscles is constantly observable. Weakness of the external rotators may be marked; generally Full can test and Patte test are positive. Very often, the lag signs are also positive.

In the vast majority of patients, the active and passive range of motion is severely limited because of soft tissue contractures or fixed glenohumeral subluxation [14]. Patients who maintain a stable core can keep mobility in flexion and abduction.

5.4 Differences Between Concentric and Eccentric Glenohumeral Arthritis

5.4.1 Histology

At our knowledge, no studies have been conducted regarding histological differences between shoulder arthropathies with or without cuff tear. Actually, the vast majority of the studies have considered histological and ultrastructural characteristics of the idiopathic arthritis, assuming that there were no differences between the two conditions. In both, articular cartilage layer is thinned or, as in the areas submitted to higher mechanical stress, has deep and broad splits or is completely absent, leaving wide exposition of the subchondral bone. In the most severe cases, cells are arranged in clusters in the deeper layer of the cartilage; sometimes chondrocyte lacunae are empty, surrounded by thickened collagen fibers [31]. The living cells are in intense activity and have well-developed cytoplasmic granules. They are enclosed in lacunae that contain numerous fibrils and mature collagen fibers. Matrix is represented by thickened collagen fibers, arranged in all directions, often perpendicularly disposed with respect to articular surface. Colloidal iron staining shows the presence of mucopolysaccharides around the living chondrocytes.

Neer [2] histologically described 26 shoulders with cuff arthropathy. Authors observed three consistent findings: areas with atrophic cartilage and osteoporotic subchondral bone in the humeral head; areas where cartilage is denuded and subchondral bone is sclerotic; and fragments of articular cartilage in the subsynovial layer. A histological study performed by Jensen et al. [14] on specimens of patients with cuff arthropathy revealed foci of calcific deposits in synovial microvilli.

Kramer et al. [24] performed an elegant study on rats whose cuff tendons were previously excised. The histological analysis was performed 12 months after surgery. Authors observed significant cartilage changes in the humeral head compared with the control side. Applying the modified Mankin score [32] (widely used for histologic evaluation of osteoarthritis), they obtained a value of 5.7 ± 1.9 in the involved shoulder and 2.0 ± 1.0 in the control side ($P < 0.001$). The score considers the structure, cellularity, safranin O staining, and tidemark integrity. Analogously, glenoid values were, respectively, 5.1 ± 1.9 and 2.4 ± 0.8 ($P < 0.001$).

CT studies [33–35] have demonstrated that bone density, below the superficial cartilaginous layer of the glenoid, varies with the different forms of arthropathy. In particular, the calcified cartilage layer, which is deeper than the noncalcified layer, is thicker in cuff arthropathy with respect to the concentric arthropathy; instead, the subchondral bone is thinner [35].

Kekatpure et al. [36] submitted to histopathologic analysis the humeral head of nine women who underwent total shoulder arthroplasty for a rapidly destructive arthrosis (rapid collapse of the humeral head with no evidence of other nonseptic articular arthropathy). Of the nine cases, seven had a rotator cuff tear (however fatty infiltration of the rotator cuff muscles were not indicative of a chronic condition), whereas tendinosis in the supraspinatus tendons was found in two cases. Analysis showed absence of articular cartilage. In the subchondral zone, both fragmentation and regeneration of bone matrix, which represented fracture healing, were observed. There was no evidence of inflammatory changes, microorganisms, or crystal-induced arthropathy. Authors did not observe typical AVN findings in the marrow, medullary bone, and cortex.

5.4.2 Age and Gender

It is known that patients with cuff tear arthropathy usually are older than those with concentric arthropathy and are very often females. To check the reliability of these data, we reviewed

all the scientific papers published in English from 2000 to date, relative to shoulder arthropathy without rotator cuff tear. We excluded all the papers conducted on patients with rheumatologic diseases, traumas, infections, previous surgical treatments, and cohorts of less than 20 patients. We were able to have demographic information on about 2761 patients with concentric arthropathy [37–45]. Data obtained were compared with those of a meta-analysis conducted by Samitier et al. [46] in 2015 relative to patients with cuff tear arthropathy. This cohort consisted of 581 patients. Data were not statistically analyzed. The weighted average age of the 2761 patients was 66.7 years while that of patients with cuff arthropathy was 72.0 years. These differences reflect my personal experience. In fact, in my series, the mean age of cuff intact arthropathy patients was 70.1 years while that of patients with cuff tear arthropathy was 75.6 years. The different age justifies a different etiology.

Analyzing the 2761 patients with concentric shoulder arthropathy, the weighted percentage of females was 48.8%; that relative to cuff tear arthropathy was 74%. In my series, the percentages were 56% and 70.3%, respectively.

Literature data indicate that the prevalence of cuff tear does not vary between genders. These data reflect our experience. In our series of 586 patients with different sized cuff tear, males and females were, respectively, 280 and 306 [47]. Nevertheless, cuff tear arthropathy is much more common in females. Different hypotheses may be formulated to explain this sexual predisposition:

(1) The percentage of females with joint hyperlaxity is higher than that of males [48–54]; therefore, in absence of cuff tendons and with less effective static stabilizers, the shoulder could result excessively unstable.

(2) Muscle mass in females is less represented [55]; also in this case, the shoulder, in absence of cuff tendons, could be less stable.

(3) Cuff tear arthropathy might be an autoimmune disease and therefore belong to those diseases that notoriously are more frequent in females. In this case estrogens would play a primary role. In fact, estrogen receptors are present on cells of the immune system involved in the pathogenic mechanism of the autoimmune disease.

(4) Genetics.

(5) Environmental factors and lifestyle.

5.4.3 Functional Evaluation

Absolute values of ASES and SST scores are lower in patients with cuff arthropathy than those reported for patients with concentric arthropathy (Table 5.1). This is partly due to the fact that patients with cuff arthropathy are older. However, the marked difference between the mean values of flexion, abduction, and external rotation recorded in the two groups of patients (Table 5.2) indicates an actual functional difference. In addition, patients with cuff arthropathy have a decrease in external rotation strength that further compromises shoulder function. Surprisingly, it shows no significant differences between the two groups when the shoulder function is evaluated with the constant score.

Table 5.1 Functional evaluation in patients with shoulder arthropathy

	Concentric arthropathy	Cuff tear arthropathy Data relative to 581 patients [46]
	Weighted average	Weighted average
Constant score	27.9 (57 patients) [40, 44] 26.8 (210 patients) [56] 26.3 (41 patients) [57] 30.1 (41 patients) [57] 37.3 (62 patients) [58]	30.5
ASES score	39.3 (635 patients) [37, 42, 43, 45]	31.8
SST	3.3 (57 patients) [42, 45]	1.8

Comparison between concentric arthropathy and cuff tear arthropathy

Table 5.2 Range of motion in patients with shoulder arthropathy

	Concentric arthropathy	Cuff tear arthropathy Data relative to 581 patients [46]
	Weighted average	Weighted average
Forward flexion	90.1° (771 patients) [37, 38, 42–45]	63.7°
Abduction	76.2° (488 patients) [43–45]	51.2°
External rotation	21.8° (771 patients) [37, 38, 42–45]	12.5°

Comparison between concentric arthropathy and cuff tear arthropathy

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