

RELATIONSHIP BETWEEN THE CRITICAL SHOULDER ANGLE AND SHOULDER DISEASE

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Abstract

» In this review, we describe the history of the Critical Shoulder Angle (CSA), the utility of the CSA in identifying rotator cuff tears and glenohumeral osteoarthritis, and the association between the CSA and patient-reported outcomes. Additionally, we address some of the controversies surrounding the CSA with an updated literature review.

» The CSA is the angle between the plane of the glenoid fossa (the line from the inferior edge of the glenoid to the superior edge of the glenoid) and a line drawn from the inferior edge of the glenoid to the lateral edge of the acromion on a true anteroposterior (Grashey) shoulder radiograph.

» An increased CSA ($>35^\circ$) is thought to alter deltoid vectors, which results in increased superior shear forces on the rotator cuff muscles. This increased loading of the rotator cuff may be a risk factor for the development of rotator cuff tears.

» A decreased CSA ($<30^\circ$) is associated with glenohumeral arthritis due to the increased compressive forces across the glenohumeral joint.

» Reports in the literature have both supported and refuted the associations between the CSA, shoulder disease, and clinical treatment outcomes. These conflicting findings may be attributable to the lack of standardized radiographic methods for measuring CSA and/or to measurement errors.

» Prospective longitudinal cohort studies involving a standard and reproducible method of CSA measurement are needed to elucidate the true relationship between the CSA and shoulder disease.

Introduction

Acromial morphology has been linked to the development of rotator cuff disease for decades. Multiple methods for quantifying and measuring acromial and scapular anatomy and morphology have been proposed and studied; however, the precise acromial morphologies that predispose the shoulder to impingement syn-

drome, subacromial bursitis, and rotator cuff tears continue to be debated. Previous analyses by Bigliani et al. and Neer identified the anterolateral acromial morphology (including the coracoacromial ligament) as a source of subacromial pain and a causative factor in the development of bursal-sided rotator cuff tears^{1,2}. The findings of Neer and Bigliani et al. resulted in the widespread adoption of either

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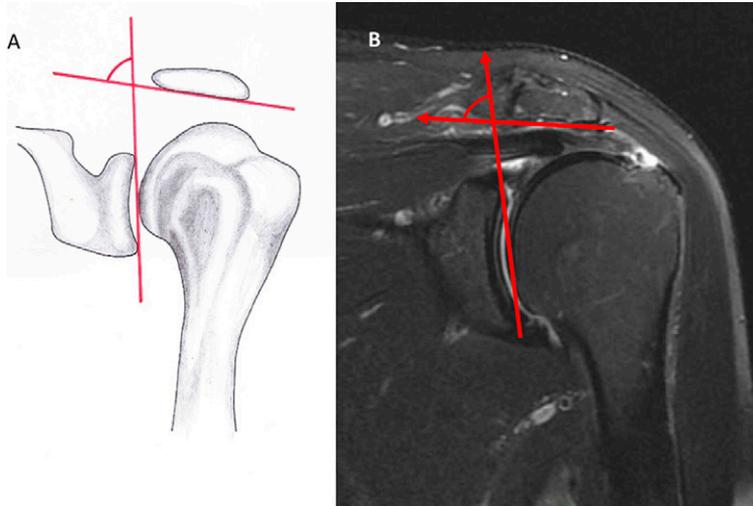


Fig. 1
Fig. 1-A Illustration depicting the lateral acromion angle, which is formed by the intersection of a line drawn on the subacromial surface and a line tangent to the glenoid articular surface. **Fig. 1-B** Coronal T2-weighted MRI scan showing a lateral acromion angle of 80.1°.

arthroscopic or open subacromial decompression of the anterolateral part of the acromion as a treatment for rotator cuff disease and impingement-related symptoms³.

Others, including Watson-Jones⁴, Armstrong⁵, and, most recently, Moor et al.⁶, have implicated the lateral extension of the acromion as a risk factor for the development of rotator cuff disease. Moor et al. introduced the concept of the Critical Shoulder Angle (CSA), which is thought to be predictive of both rotator cuff disease and glenohumeral arthritis. Thus, reviewing the historical acromial indices and measurements is instructive in understanding how the CSA can be used as a tool for determining and predicting various shoulder abnormalities.

History

Most acromial indices were introduced with the goal of better identifying and directing the treatment of rotator cuff-related disorders. Bigliani et al.¹, who developed what has become the most popular system for the prediction of impingement and the development of rotator cuff tears, classified acromial morphology into 3 types: flat (Type 1; 17% of shoulders), curved (Type 2; 43%), and hooked (Type 3; 40%). Various other methods of quantifying acromial morphology and the associated effect on the development of different shoulder abnormalities have also been introduced. The lateral acromion angle,

as described by Banas et al.⁷, primarily takes into account glenoid and acromial inclination as measured on a coronal oblique magnetic resonance imaging (MRI) scan (Fig. 1). Those authors compared measurements from patients with rotator cuff disease of varying severity and found a significant association between full-thickness rotator cuff tears and a lateral acromion angle of $\leq 70^\circ$.

Similar to Bigliani et al.¹, Aoki et al.⁸ described decreased acromial tilt, as seen on a lateral scapular radiograph or supraspinatus outlet view, as a risk factor for the development of rotator cuff disorders (Fig. 2). Those authors furthered the concept that anterolateral acromial morphology was a source of rotator cuff disease, hypothesizing that changes to the acromial morphology that decreased the area of the supraspinatus outlet led to narrowing between the acromion and the greater tuberosity, thus creating an abrading effect on the rotator cuff insertion and leading to a subsequent tear. However, other authors have proposed that adaptive or acquired changes within the acromion and coracoacromial ligament are an effect of rotator cuff disorders rather than a cause⁹⁻¹². Additionally, a meta-analysis¹³ and a randomized trial¹⁴ evaluating the effect of acromioplasty on the outcomes of arthroscopic rotator cuff repairs revealed no difference in outcomes and failure rates between repairs that included an

anterolateral acromioplasty and those that did not.

The association between a large lateral acromial extension and symptomatic rotator cuff tears was further studied radiographically by Nyffeler et al.¹⁵. Those authors described a radiographic parameter, the acromial index, and found that a higher index (indicating increasing amounts of lateral acromial extension) was associated with rotator cuff tears. The authors theorized that the increased acromial extension resulted in a larger vertically directed force on the deltoid muscle during abduction. As a result, the supraspinatus

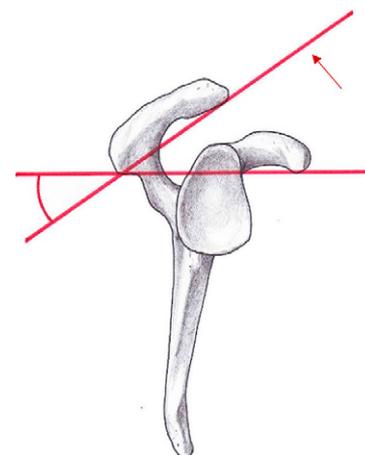


Fig. 2
Illustration depicting acromial tilt, which is formed by the intersection of the line extending from the anterior tip of the acromion to the acromial angle (arrow) and the line extending from the tip of the coracoid process to the acromial angle.

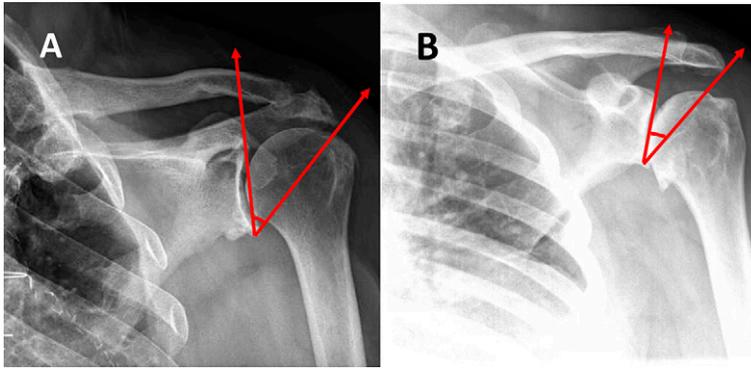


Fig. 3

Figs. 3-A and 3-B Radiographs demonstrating the CSA, which takes into account both lateral acromial overhang and glenoid inclination.

Fig. 3-A A shoulder with a full-thickness rotator cuff tear and a CSA of 46.1°. **Fig. 3-B** A shoulder with osteoarthritis, an intact rotator cuff, and a CSA of 30.1°.

was subjected to increased strain or shear forces because a larger horizontal force was needed to stabilize the humeral head onto the glenoid. This association was further refined with the description of the CSA (Fig. 3)⁶.

Although similar to the acromial index, the CSA measurement does not include the shape or orientation of the humeral head. Including the humeral head in measurements evaluating acromial morphology can lead to a misleading value in the setting of osteoarthritis, when the humeral head becomes flattened from arthritic wear and subsequently medializes. The CSA does, however, take into account glenoid inclination, which also has been implicated as a risk factor for the development of rotator cuff tears¹⁶⁻¹⁸. The CSA therefore accounts for contributions from both glenoid inclination and lateral acromial length in a measurement that includes components of both the lateral acromial angle and the acromial index. It must be noted that the lateral acromial angle is a 2-dimensional (2D) measurement, and the CSA does not account for the complex 3D morphology of the scapula or the variable anterior-to-posterior position of the lateral part of the acromion, both of which may impact the measurement and also may have pathological implications¹⁹.

Radiographic Measurement

The original description of the CSA required a true anteroposterior (Grashey) shoulder radiograph with minimal overlap of the anterior and posterior

glenoid rims. A line is drawn from the inferior edge of the glenoid to the superior edge of the glenoid (across the plane of the glenoid fossa) and is subtended by a second line drawn from the inferior edge of the glenoid to the lateral most extent of the acromion (Fig. 3). Moor et al.⁶ found that malrotation in the coronal and/or sagittal plane up to 20° from the true anteroposterior view had an effect of <2° in the CSA measurement variability. However, Suter et al.²⁰ found that a radiograph viewing angle beyond 5° of anteversion or 8° of retroversion resulted in a >2° deviation in the CSA in comparison with the true anteroposterior view. Flexion and extension viewing angles were better tolerated according to the authors, with viewing angles >15° of flexion and >26° of extension being required to produce a >2° change in the CSA in comparison with the true anteroposterior view. That study also incorporated a novel radiographic classification system (the Suter-Henninger system) for the evaluation of malorientation of the scapula, which affects the accuracy of CSA measurements on the basis of the glenoid double-contour sign and inverted teardrop patterns at the upper glenoid rim (Figs. 4-A through 4-D). Scapular flexion and extension were also assessed using the coracoglenoid overlap patterns (Figs. 4-E, 4-F, and 4-G). After ruling out any glenoid double contour patterns involving >50% of the glenoid height (patterns D1, D2, and D3) and any inverted teardrop patterns involving <50% of the glenoid height (patterns B1, B2, and B3),

the authors found an 89% probability of accurately assessing the true CSA within 2° compared with the true anteroposterior view. Because obtaining a reproducible true anteroposterior radiograph to accurately measure the CSA is difficult, utilizing the Suter-Henninger criteria for measuring the adequacy of the anteroposterior radiograph to accurately assess the CSA is critical in any future studies.

Chalmers et al. found that only 21% of 1,552 radiographs were of sufficient quality to utilize the CSA accurately, suggesting a high degree of variability in the 3D morphology of the scapula or rotation of the radiographs that may affect the accuracy of the CSA measurement²¹. In an effort to overcome this limitation, Spiegl et al.²² developed a method for measuring the CSA with use of a coronal T1-weighted oblique MRI scan, but they found that using conventional radiography according to the method described by Moor et al.⁶ was more accurate and reproducible than using MRI. Other authors²³ have found that radiographs showing anterior-to-posterior glenoid rim overlap of >11 mm are an indication of >20° of malrotation as defined by Moor et al.⁶. However, as shown by Suter et al.²⁰, much of the recent research on the CSA may be invalid if only 5° to 8° of change in anteversion or retroversion, instead of the previously accepted 20° of change, can affect the accuracy of measurements. In summary, because of the variation in radiographic angulation and the complex 3D morphology of the scapula and lateral part of

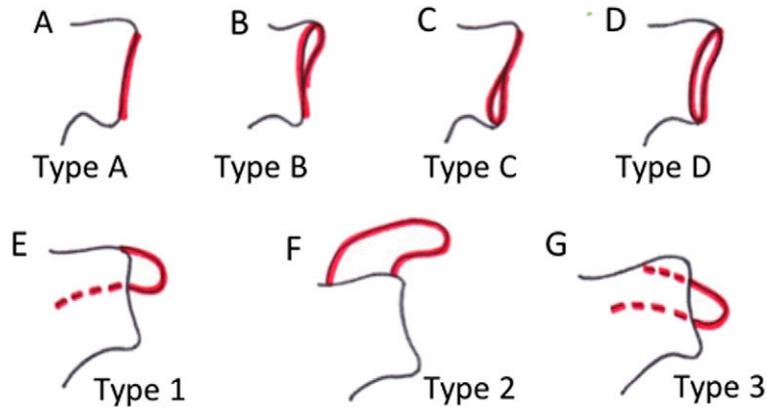


Fig. 4

Figs. 4-A through 4-G The Suter-Henninger (SH) classification system for verification of radiographic adequacy of a true anteroposterior radiograph to measure the CSA. **Figs. 4-A through 4-D** Illustrations depicting the assessment of rotation of the radiograph. The glenoid rim is outlined in red. Rotation is classified as Type A (no double contour), Type B (superior or inverted teardrop), Type C (inferior teardrop), or Type D (double contour). **Figs. 4-E, 4-F, and 4-G** Illustrations depicting the coracoglenoid overlap, which is an assessment of the scapular flexion or extension. The coracoid is outlined in red. Overlap is classified as Type 1 (optimal overlap of the upper glenoid rim and the inferior edge of the coracoid), Type 2 (no overlap, with the coracoid above the superior glenoid rim), or Type 3 (inferior overlap, with the coracoid below the superior glenoid rim).

the acromion, future research must focus on standardizing how the CSA is measured to help ensure greater accuracy and reproducibility.

Critical Shoulder Angle

The initial evaluations of the CSA in the literature indicated that normal values were between 30° and 35°, with values of <30° being implicated in the development of glenohumeral osteoarthritis and values of >35° being considered a risk factor for rotator cuff tears^{6,23-26}. In their original study, Moor et al.⁶ evaluated the CSA in a control group of 94 asymptomatic shoulders with normal rotator cuff musculature and no osteoarthritis, 102 shoulders with MRI-documented full-thickness rotator cuff tears and no osteoarthritis, and 102 shoulders with primary osteoarthritis and no cuff tears noted during shoulder-replacement surgery. The mean CSA was 33.1° (range, 26.8° to 38.6°) in the control group, 38.0° (range, 29.5° to 43.5°) in the cuff-tear group, and 28.1° (range, 18.6° to 35.8°) in the osteoarthritis group. Furthermore, of the patients with a CSA of >35°, 84% were in the cuff-tear group, and of those with a CSA of <30°, 93% were in the osteoarthritis group.

Spiegel et al.²², in a study comparing 3 groups of 10 age-matched patients with rotator cuff tears, osteoarthritis, or

neither abnormality, reported significant differences among the 3 groups in terms of the mean CSA ($p < 0.001$). Similarly, Heuberger et al.²⁵, in a study involving 100 patients with rotator cuff tears and no osteoarthritis and 100 patients with osteoarthritis, reported that the mean CSA (and standard deviation) was $36.3^\circ \pm 2.7^\circ$ in the rotator cuff tear group and $27.3^\circ \pm 3.5^\circ$ in the osteoarthritis group. Additionally, in a subset of patients with cuff tear arthropathy ($n = 100$), the mean CSA was $35.2^\circ \pm 2.8^\circ$. Mantell et al.²⁷, using the receiver operating characteristic (ROC) curve, showed that a CSA of >35° was 90% specific and 52% sensitive for a full-thickness cuff tear in the setting of osteoarthritis. Contrary to the above findings, Chalmers et al.²¹ found that the mean CSA was $34^\circ \pm 4^\circ$ in patients with cuff tears, compared with $32^\circ \pm 4^\circ$ in patients with normal rotator cuffs, and concluded that the difference was small enough that it could have been influenced by measurement error.

Rotator Cuff Tears and CSA

The etiology of atraumatic, degenerative rotator cuff tears has been the topic of great controversy in both sports medicine and shoulder surgery. In their landmark retrospective study comparing shoulders with no pathology and shoulders with full-thickness rotator cuff

tears, Moor et al.⁶ reported that the cuff tear group had a larger mean CSA compared with controls (38.0° versus 33.1°, respectively). Those authors concluded that the CSA, which demonstrated a sensitivity of 0.82 and a specificity of 0.92, may be of value for predicting rotator cuff tears and that anatomical differences in the CSA may be risk factors for the development of degenerative rotator cuff tears.

When compared with other acromial metrics such as the lateral acromial angle, the acromial index, or the acromioglennoid angle, the CSA has been found to be a more discriminatory metric for distinguishing between shoulders with degenerative full-thickness tears and control shoulders by producing the largest area under the ROC curve and the highest levels of sensitivity and specificity^{25,26,28}. Additionally, both Moor et al. and Heuberger et al. demonstrated that the combined risk factors of age and CSA were highly predictive of degenerative rotator cuff tears^{25,26}. Multiple subsequent studies have reaffirmed this relationship between a CSA of >35° and the incidence of atraumatic rotator cuff tears^{22,24,28-31}.

However, despite the association between the CSA and rotator cuff pathology, the CSA may only be helpful for diagnosing full-thickness tears and not partial tears. In a subsequent study,

Moor et al.³² demonstrated that a larger CSA was significantly associated only with full-thickness rotator cuff tears ($p < 0.0001$). Pandey et al.³³ further substantiated this relationship by showing that while the average CSAs for shoulders with full-thickness and partial-thickness tears (41.01° and 38.83° , respectively) were higher than that for control shoulders (37.28°), only the full-thickness tear group was significantly different from the control group ($p < 0.001$). Furthermore, Chalmers et al.²¹ noted that only 21% of 1,552 radiographs were of adequate quality to measure the true CSA on the basis of the Suter-Henninger grading scale and concluded that the 2° difference that they noted between their study groups may have been clinically irrelevant because it could have been due to measurement error alone. These findings suggest that while the CSA may be correlated with atraumatic full-thickness rotator cuff tears, diagnosis should not be based on this measurement alone and that further advanced imaging may be needed to confirm the presence of rotator cuff pathology.

While there have been many retrospective observational studies evaluating the association between a large CSA and degenerative rotator cuff tears, there have been few biomechanical studies investigating this relationship. Gerber et al.³⁴ utilized a robotic shoulder model to assess the biomechanical association between the CSA and supraspinatus tendon load during shoulder abduction. The investigators found that a smaller CSA was associated with greater compressive joint forces and lower shear forces, especially during early abduction. Consequently, a large CSA (38°) when compared with a smaller control CSA (33°) led to a greater instability ratio (defined as joint shear to joint compression forces) between 6° and 61° of abduction, with the largest instability ratio difference occurring between 33° and 37° of elevation. To further explain the association between increased CSA and larger vertical shear forces on the shoulder joint, Gerber

et al. suggested that an increased CSA can cause the deltoid forces to be directed relatively more superiorly in the coronal plane, which would require a 13% to 33% increase in supraspinatus force to achieve normal levels of joint stability between 33° and 37° of active abduction. Thus, a larger CSA would lead to degenerative rotator cuff tears over time due to the abnormal loading patterns of the supraspinatus tendon.

In a separate biomechanical study, Moor et al.³⁵ examined the effects of glenoid inclination-dependent variations of the CSA with use of an experimental simulator and cadaveric shoulders. The investigators found that as the CSA increased, both the instability and shear forces of the joint increased. As a result, the supraspinatus muscle is needed to compensate for glenohumeral joint instability in shoulders with an increased CSA, especially with shoulder abduction angles of $>16^\circ$. Because balanced mechanical loading of the shoulder joint is needed for normal shoulder physiology, these biomechanical studies suggest that a large CSA may lead to a disequilibrium of forces on the glenohumeral joint, particularly between the humeral head and the rotator cuff muscles. Thus, the biomechanical studies discussed above suggest that the supraspinatus muscle must be activated to stabilize the joint in shoulders with a large CSA^{34,35}. This increased activation of the supraspinatus could explain, in part, the increased observation of degenerative rotator cuff tears in association with a large CSA.

While the combination of these observational and biomechanical studies supports the possible association between a large CSA and an increased likelihood of rotator cuff tears, the clinical utility of the CSA remains questionable in the setting of rotator cuff disease. Moreover, because most of the studies evaluating this association were performed retrospectively with either a case-control or case series study design, further prospective studies are needed to solidify the association between CSA and rotator cuff tears.

Shoulder Osteoarthritis and CSA

The etiology of glenohumeral osteoarthritis has been reported to be associated with a wide range of risk factors, such as age and trauma^{36,37}. Recently, attention has been paid to scapular morphology in the search for a reliably predictive measurement related to shoulder osteoarthritis. Measurements such as glenoid inclination and lateral extension of the acromion have been studied to determine whether there is a relationship between osseous anatomy and the development of osteoarthritis over time, but no statistical difference was seen when these measurements were compared between arthritic and control subjects^{6,15}. Recently, in continued attempts to better understand the etiology of glenohumeral osteoarthritis, attention has been paid to the CSA, which has been shown to be significantly different when patients with osteoarthritis have been compared with controls without osteoarthritis^{6,22,25,28,30,31}.

In their 2013 landmark study, Moor et al.⁶ found a significant 5° difference between control subjects and patients with osteoarthritis in terms of the CSA (mean, 33.1° versus 28.1° , respectively; $p < 0.0001$). Those authors concluded that a CSA of $<30^\circ$ was associated with osteoarthritis, but, because of the retrospective nature of the study, they were unable to find a causative relationship between the CSA and osteoarthritis. However, several subsequent studies have shown, with strong interobserver and intraobserver reliability, that a CSA of $<30^\circ$ has a significant association with osteoarthritis^{22,25,28,30,31}. Heuberger et al.²⁵, in a retrospective matched-cohort study of 1,000 patients with and without shoulder osteoarthritis, found that the CSA in the osteoarthritis group was significantly smaller than that in the control group ($p < 0.001$). In that study, the CSA measurement also demonstrated the highest sensitivity (82.0%) and specificity (76.1%) of all the indices assessed²⁵. That study is the largest to date in which the CSAs of patients with osteoarthritis have been compared with those of

controls. While the study established a high sensitivity and specificity for the CSA in relation to osteoarthritis, its retrospective nature limited its ability to show causation between CSA and osteoarthritis. Recently, Bjarnison et al.²³ reported that the CSA was associated with shoulder osteoarthritis but not rotator cuff tears. The authors reported an odds ratio for the development of osteoarthritis of 2.25 for patients with a CSA of $<30^\circ$ ($p = 0.002$), whereas the odds ratio for the development of a cuff tear was 1.12 for patients with a CSA of $>35^\circ$ ($p = 0.63$). Furthermore, 1 study evaluating the effect of the CSA on patient outcomes after anatomic total shoulder arthroplasty demonstrated no relationship between the CSA and the subsequent development of late rotator cuff tears.³⁸

Although the above studies supported an association between a decreased CSA and the development of osteoarthritis, we are not aware of any prospective studies that have suggested causation^{6,22,25,28,30,31}. It is believed that as the CSA decreases, forces on the deltoid shift, causing the forces on the glenohumeral joint to become more compressive, thereby resulting in higher loading of the cartilage and subsequent osteoarthritis. Several studies have demonstrated that as the CSA decreases, the force vector of the deltoid is directed more horizontally, increasing the compressive force of the humeral head on the glenoid^{34,35,39}.

The major limitation of those biomechanical studies is their use of pulleys and wires in cadaveric models to replicate the shoulder musculature; such simulations are unable to replicate the dynamic relationships between the rotator cuff muscles. Consequently, they do not assess the integrity of the axial rotator cuff force couple, which plays a role in the development of osteoarthritis. A recent study by Naidoo et al.¹⁹ demonstrated the variability in the 3D morphological anatomy of the scapula and the position of the lateral part of the acromion as measured on a supraspinatus outlet-view radiograph.

This variable anatomy, which may not be captured by the CSA, could affect the deltoid and rotator cuff force couple and be related to the development of osteoarthritis. Additionally, those studies, like the CSA itself, do not account for the osseous glenoid morphology and version of the scapula in the axial plane, which can further affect the true relationship between the CSA measurement and shoulder osteoarthritis.

In summary, much attention has been placed on finding a reliable measurement to predict the development of shoulder osteoarthritis. However, no studies to date have shown a causal relationship between the CSA and osteoarthritis, most likely because of the retrospective nature of most studies. Randomized controlled trials with standardized radiographic measurements are needed to assess whether patients are at risk for developing osteoarthritis in association with a lower-than-normal CSA or whether low CSAs are the result of the disease process itself changing the glenoid and acromial morphology. Additionally, research is needed to determine how overall 3D shoulder morphology affects the development of degenerative glenohumeral joint disease.

Patient Outcomes and CSA

A limited number of studies have evaluated the influence of the CSA on patient outcomes and retear rates after rotator cuff repair. In a Level-III study, Garcia et al.⁴⁰ retrospectively reviewed the records for 76 patients who had undergone primary arthroscopic rotator cuff repair. After a mean duration of follow-up of 26.1 months, those authors found that a higher CSA was significantly associated with a full-thickness retear as assessed with ultrasound 6 months postoperatively ($p < 0.01$). Specifically, a CSA of $>38^\circ$ was associated with a 15-fold increased risk of a postoperative full-thickness tear. Furthermore, an increased CSA was significantly associated with worse postoperative American Shoulder and Elbow Surgeons (ASES) scores ($p <$

0.03). Although that study was limited by its retrospective nature and short-term follow-up, it provided the initial evidence to support a correlation between the CSA and outcomes after rotator cuff repair.

Kirsch et al.⁴¹ reported contrasting results in a Level-II prospective study of 53 patients (mean age, 61 years) who underwent arthroscopic rotator cuff repair for the treatment of atraumatic full-thickness rotator cuff tears. Postoperatively, clinical outcomes improved significantly as assessed with Western Ontario Rotator Cuff, ASES, and visual analog scale (VAS) pain scores ($p < 0.0001$ for all); however, the CSA was not significantly associated with any of those outcomes at the 24-month follow-up visit ($p = 0.581, 0.458, \text{ and } 0.859$, respectively). Similarly, Lee et al.⁴² investigated the influence of the CSA and the acromial index on outcomes after arthroscopic rotator cuff repair. Although the Constant Shoulder Score, Oxford Shoulder Score, and University of California at Los Angeles Shoulder Rating Scale score were significantly worse for patients with a CSA of $>35^\circ$ as compared with those with a CSA of $\leq 35^\circ$ at 6 months ($p = 0.005, 0.030, \text{ and } 0.035$, respectively), there were no significant clinical outcome differences at 24 months. Given these conflicting preliminary results, further investigation is needed to determine the associations between the CSA and outcomes after rotator cuff repair. Future studies should also address the role of using the CSA measurement to predict which patients would benefit from an arthroscopic lateral acromial resection to optimize long-term surgical outcomes after rotator cuff repair. Reducing the CSA via arthroscopic lateral acromial resection has been shown to be feasible in cadaveric specimens^{43,44}.

There is also a paucity of evidence examining the role of CSA measurements in predicting the outcome of surgical treatment of irreparable rotator cuff tears with tendon transfer. Gerber et al.⁴⁵, in a study on the results of 46 latissimus dorsi tendon transfers in 44

patients with irreparable posterosuperior rotator cuff tears at a minimum of 10 years, found that larger CSAs were associated with inferior outcomes. The mean CSA in the group with unsatisfactory outcomes was significantly higher than that in the group with satisfactory outcomes ($39.5^\circ \pm 3.6^\circ$ [range, 34° to 44°] compared with $36.1^\circ \pm 2.6^\circ$ [range, 32° to 42°]; $p = 0.005$). In a subgroup analysis, the 22 shoulders with a CSA of $<36^\circ$ had a mean relative Constant score of 91 at the time of the latest follow-up, whereas the 24 shoulders with a CSA of $>36^\circ$ had a mean relative Constant score of 71 ($p < 0.0001$). Those authors concluded that shoulders with fatty infiltration of the teres minor, subscapularis insufficiency, and a larger CSA had inferior results after latissimus dorsi tendon transfer for the treatment of irreparable posterosuperior rotator cuff tears. Although the study by Gerber et al. provided further evidence of an association between an increased CSA and worse outcomes after tendon transfer for massive irreparable rotator cuff tears, in light of the conflicting results from the previously mentioned Level-II and III studies, this association is currently observational, without proven therapeutic consequences. Further prospective studies are needed to more definitively demonstrate outcomes-related relationships.

Overview

Abnormal acromial morphology, especially lateral acromial extension, contributes to the development of rotator cuff disease by creating altered mechanical vectors that affect both compressive and shear forces. The CSA has been linked to the development of both rotator cuff tears and osteoarthritis. An increased CSA ($>35^\circ$) is thought to result in increased superior shear forces on the rotator cuff muscles due to altered deltoid vectors, which may help to predict, and may be a risk factor for, the development of rotator cuff tears. A decreased CSA ($<30^\circ$) is associated with glenohumeral arthritis due to increased compressive forces across the joint. Pub-

lished studies have both supported and refuted these associations; the conflicting findings may be due to the lack of standardized radiographic methods for measuring the CSA and/or measurement errors. Prospective longitudinal cohort studies involving a standard and reproducible method of CSA measurement are needed to determine the true relationship between the CSA and shoulder disease.

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