The Rotator Interval: A Review of Anatomy, Function, and Normal and Abnormal MRI Appearance

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OBJECTIVE. The purpose of this article is to review imaging of the rotator interval, an anatomically complex region in the shoulder that plays an important role in the normal function of the shoulder joint. The rotator interval can be difficult to evaluate by imaging, and it is not routinely evaluated arthroscopically unless the clinical examination or imaging findings suggest an abnormality of the rotator interval. Rotator interval pathology is implicated in glenohumeral instability, biceps instability and adhesive capsulitis—entities which remain a challenge to diagnose and treat.

CONCLUSION. Imaging can play an important role in increasing suspicion for injury to the rotator interval so that this region can be evaluated and appropriate treatment can be initiated.

The rotator interval is a complex anatomic region containing several structures important for the stability and proper biomechanical functioning of the shoulder. Diagnosis of abnormality of the rotator interval is often difficult because of its complex anatomy and the difficulty in visualizing the structures within it on both imaging studies and arthroscopy. In addition, treatment of rotator interval abnormality remains controversial. This article will discuss the anatomy and function of the rotator interval, the imaging and appearance of the normal rotator interval, and the imaging appearance of rotator interval abnormality.

Anatomy

The rotator interval is that portion of the shoulder joint where the rotator interval capsule is reinforced externally by the coracohumeral ligament (CHL) and internally by the superior glenohumeral ligament (SGHL) and traversed by the intraarticular biceps tendon. It is a triangular anatomic area in the anterosuperior aspect of the shoulder, which is defined by the coracoid process at its base, superiorly by the anterior margin of the supraspinatus tendon, and inferiorly by the superior margin of the subscapularis tendon (Fig. 1).

The rotator interval capsule is the anterosuperior aspect of the glenohumeral joint capsule, which merges with the CHL and SGHL insertions medial and lateral to the bicipital groove, maintaining the biceps tendon within the groove. The CHL arises outside the glenohumeral joint from the lateral aspect of the base of the coracoid process of the scapula. It broadens to merge with the rotator interval capsule and inserts on both the lesser and greater tuberosities; in doing so, it spans the bicipital groove (Figs. 1 and 2). The fibers of the CHL cannot be separated from those of the anterior supraspinatus and superior subscapularis tendons with which it interdigitates or from the rotator interval capsule. The ligament divides into two major functional (although not always clearly distinct) bands, a smaller medial band and a larger lateral band (Fig. 3). The medial band of the CHL (MCHL) blends with the fibers of the SGHL to form a ligament (SGHL–MGHL) complex that surrounds the medial and inferior aspect of the intraarticular portion of the long head of the biceps tendon before it inserts on the lesser tuberosity of the humerus and merges with the rotator interval capsule along with the superior fibers of the subscapularis tendon. The lateral band of the CHL surrounds the superior and lateral aspect of the intraarticular long head of the biceps tendon before inserting on the greater tuberosity of the humerus and on the anterior margin of the supraspinatus tendon. The CHL is lax with the arm in internal rotation and adduction. It is a relatively constant structure, found to be hypoplastic or absent
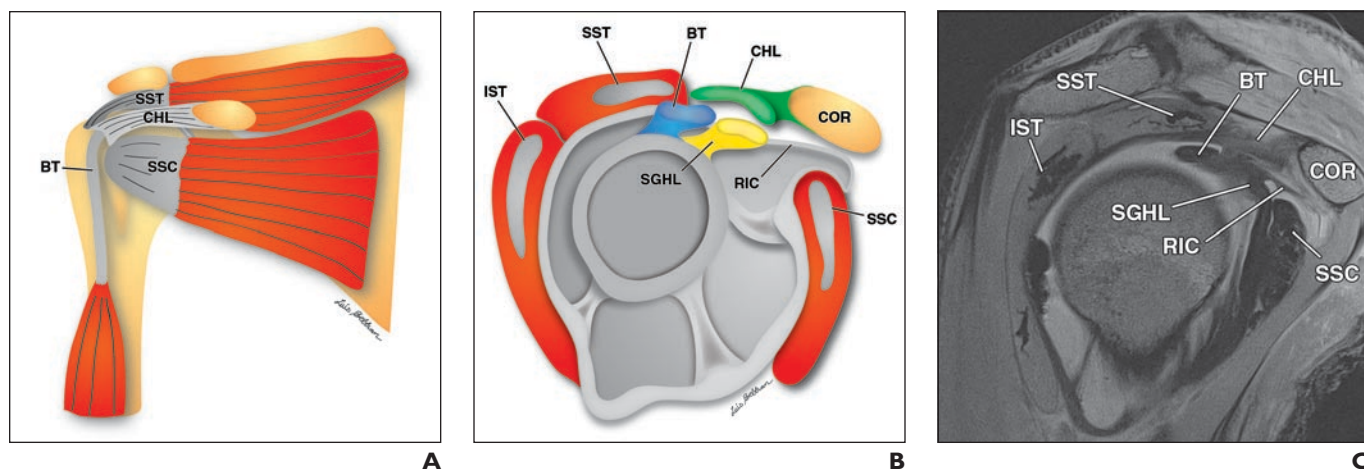
Fig. 1—Rotator interval.
A–C, Illustrations in coronal (**A**) and sagittal (**B**) planes and corresponding sagittal MR proton density–weighted arthrogram (**C**) show boundaries of rotator interval, which are defined by coracoid process (COR) at its base, superiorly by anterior margin of supraspinatus tendon (SST) and inferiorly by superior margin of subscapularis tendon (SSC). Contents of rotator interval include long head of biceps tendon (BT), coracohumeral ligament (CHL), superior glenohumeral ligament (SGHL), and rotator interval capsule. Rotator interval capsule (RIC) is anterosuperior aspect of glenohumeral joint capsule, which merges with CHL and SGHL insertions medial and lateral to bicipital groove. CHL arises from base of coracoid process, traverses through subcoracoid fat, and inserts on anterior humerus. IST = infraspinatus tendon.
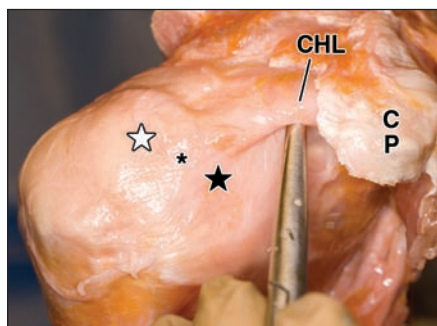
Fig. 2—Photograph of anterior view of cadaver dissection shows stout coracohumeral ligament (CHL); it suspends shoulder joint from coracoid process. CHL arises from lateral base of coracoid process (CP) and broadly inserts on lesser and greater tuberosities where it becomes confluent with subscapularis tendon (*black star*), supraspinatus tendon (*white star*), and rotator interval capsule (*asterisk*).

in only four of 63 (6%) shoulder dissections by Neer et al. [1].

The SGHL is a fold–focal thickening of the glenohumeral joint capsule (Fig. 4). It is variable in origin (supraglenoid tubercle, superior labrum, long head biceps tendon, middle glenohumeral ligament, or some combination) and may be absent in 3% of patients at arthroscopy [2]. The SGHL is anterior to and maintains a close relationship with the biceps tendon along its course. Before it inserts into a small depression above the lesser tuberosity (known as the fovea capitis of the humerus), it contributes to the biceps reflection pulley.

The long head of the biceps tendon may arise from the posterosuperior labrum, supraglenoid tubercle, or a combination of both [3]. The

tendon takes an oblique intraarticular course through the rotator interval and must make a 30° to 45° turn along the anterior surface of the humeral head before it exits the joint between the lesser and greater tuberosities in the intertubercular groove. Anomalies of the biceps brachii muscle are common, with 9–23% showing supernumerary heads [4]. More rare variants, particularly of the intraarticular portion of the long head of the biceps tendon, also have been reported in the literature [5].

The CHL and SGHL form a slinglike band surrounding the long head of the biceps tendon proximal to the bicipital groove (Fig. 5). This biceps reflection pulley plays an important role in the stability of the intraarticular biceps tendon. When the arm is abducted and externally rotated, the pulley limits medial subluxation of the biceps tendon. Medially, the superior glenohumeral ligament lies anterior to the biceps tendon. Further laterally, the ligament folds into a cup-shaped structure that cradles the biceps tendon. At the opening of the bicipital groove, SGHL fibers are joined by medial fibers of the CHL to form the biceps reflection pulley [6]. The subscapularis and supraspinatus tendon insertions along the lesser and greater tuberosities, respectively, blend with those of the CHL and are thus intimately associated with the biceps pulley [7, 8]. Injuries to any of these components of the sling are referred to as "pulley lesions" [9, 10].

Imaging

Imaging the components of the rotator interval poses several challenges. The small

size of the structures requires high spatial resolution; this is optimized by the use of strong gradients, surface coils, and high-field-strength MR scanners that maximize the signal-to-noise ratio. Even after imaging parameters are optimized, evaluating the structures of the rotator interval can be difficult. Most often, the SGHL, CHL, and rotator interval capsule appear as intermediate-weighted soft tissue filling the rotator interval and surrounding the biceps tendon. These normally coapted structures also can appear spuriously thickened when the shoulder is internally rotated; in this position, the rotator interval structures are not taut. Intraarticular fluid, either a joint effusion or intraarticular contrast material, separates the folds of tissue in this region and allows better structure delineation. It is for this reason that MR arthrography is recommended when abnormality of the rotator interval is suspected [11], and this is our preference. Nonarthrographic images, however, do allow evaluation of the extraarticular portion of the CHL and the subcoracoid fat that surrounds it.

At our institution, MR arthrography is done after 10–12 mL of dilute (1:200 solution) gadopentetate dimeglumine (Magnevist, Bayer HealthCare Pharmaceuticals) is injected into the joint and follows standard arthrography protocol: axial fat-saturated T1-weighted (TR/TE, 514/8.6), coronal oblique fat-saturated T1-weighted (754/8.6), sagittal oblique T1-weighted (450/9.4), coronal oblique fat-saturated T2-weighted (5,880/79), and abduction and external rotation
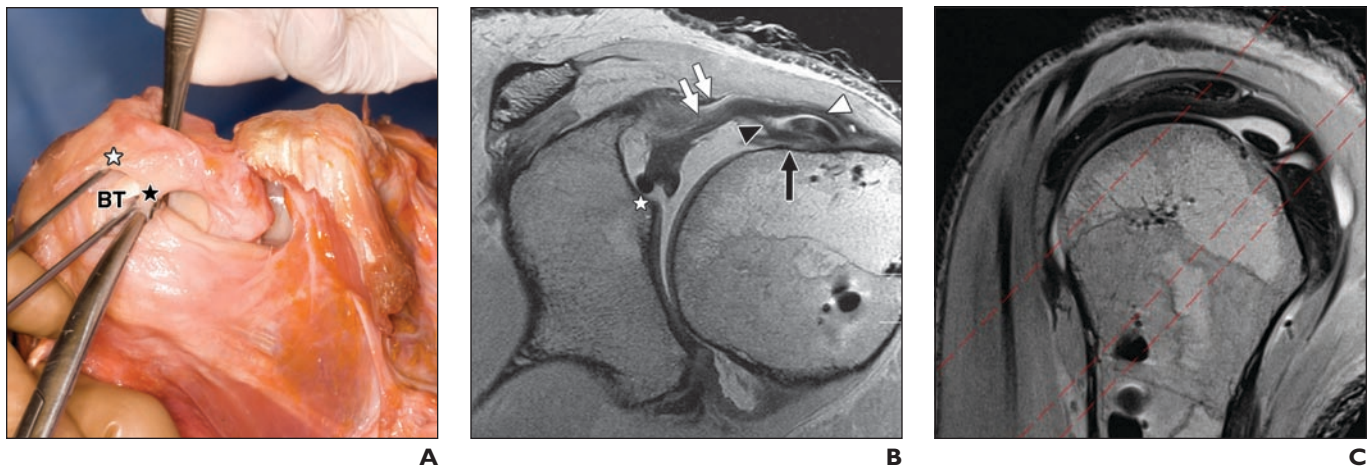
Fig. 3—Cadaver dissection.
A, Small incision was made in rotator interval capsule–coracohumeral ligament (CHL) at opening of bicipital groove. Photograph of anterior view shows biceps tendon (BT) between lateral (*white star*) and medial (*black star*) bands of CHL.
B and **C,** Oblique axial MRI (**B**) and proton density–weighted saline arthrogram (**C**) of cadaver specimen obtained perpendicular to plane of rotator interval capsule show medial band (*black arrowhead*) and lateral band (*white arrowhead*) of CHL insert on lesser and greater tuberosities, respectively. Superior glenohumeral ligament (*white arrows*) is seen from its origin (*white star*) to its insertion (*black arrow*). Dashed lines in **C** indicate plane of section.
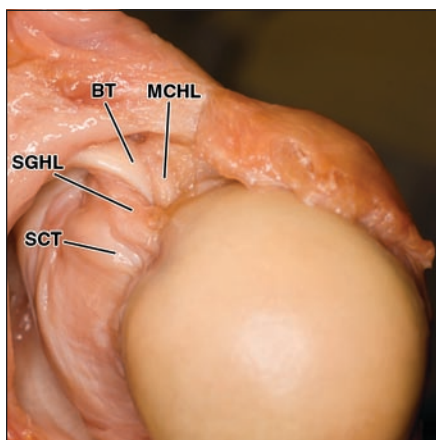

Fig. 4—Photograph shows posterior intraarticular view in internal rotation of cadaver dissection. Slinglike appearance of biceps pulley, formed by medial coracohumeral ligament (MCHL) and superior glenohumeral ligament (SGHL) is well visualized in internal rotation. SCT = subscapularis tendon, BT = biceps tendon.
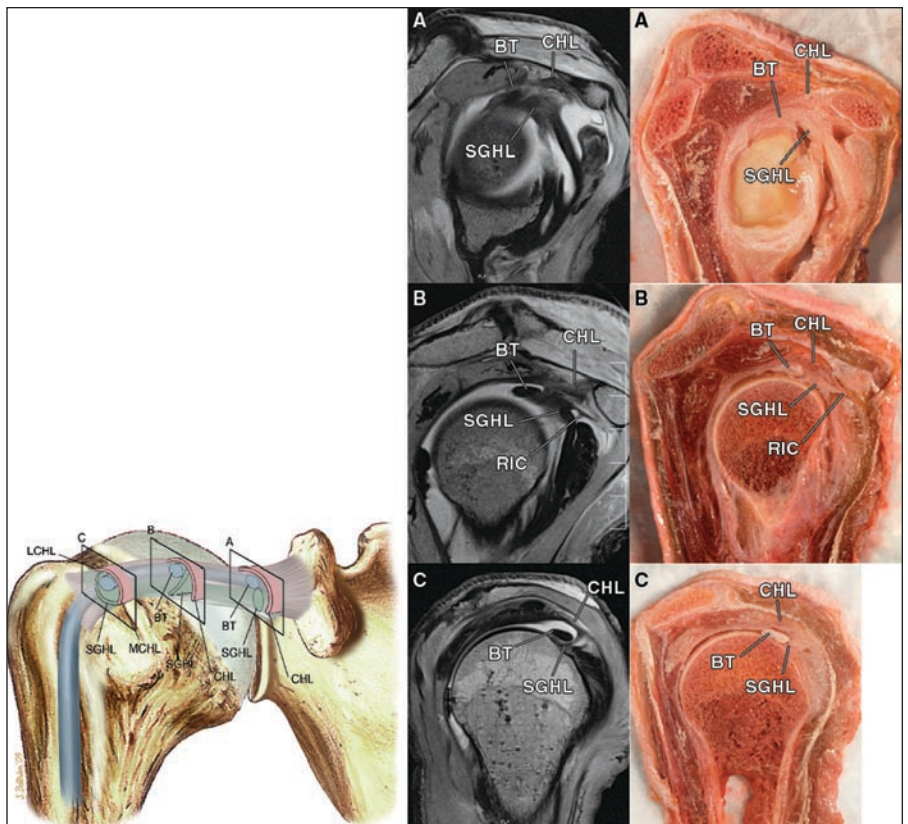
Fig. 5—Biceps pulley.
A–C, Illustration (*left*) of biceps pulley with corresponding proton density–weighted saline MR arthrograms and bandsaw sagittal sections of cadaver specimen through medial (**A**), middle (**B**), and lateral (**C**) rotator interval. SGHL = superior glenohumeral ligament, BT = biceps tendon, CHL = coracohumeral ligament, MCHL = medial coracohumeral ligament, LCHL = lateral coracohumeral ligament, RIC = rotator interval capsule.

fat-saturated T1-weighted (514/8.6) images are acquired. The slice thickness is 3–4 mm, spacing between slices is 0.3–0.4 mm, and acquisition matrix is 256 × 256.

Because of the small size, our cadaveric specimens were scanned using a 32-channel head coil on a 3-T MR scanner (Magnetom Verio 3 T, Siemens Healthcare). Saline arthrography rather than gadolinium arthrography was used because of the lack of concern for motion. Proton density–weighted images (2,450/46) were obtained at a 2-mm slice thickness using a 384 × 384 acquisition matrix in standard sagittal, axial, and oblique coronal imaging planes. The closely spaced proton density–weighted images allowed us to directly visualize the rotator interval structures with great detail. We found that specialized planes, referenced to the plane of the rotator interval capsule, did not provide added benefit with respect to evaluating the rotator interval.

CHL

On MRI, the CHL is a linear, laterally downsloped low-signal structure traversing the fat above the shoulder joint on the first few coronal oblique sections after the coracoid process is seen. On sagittal images lateral to the coracoid process, it can be seen traversing the fat above the joint capsule. Further laterally, it merges with the rotator interval joint capsule, where it cannot be identified as a separate struc-
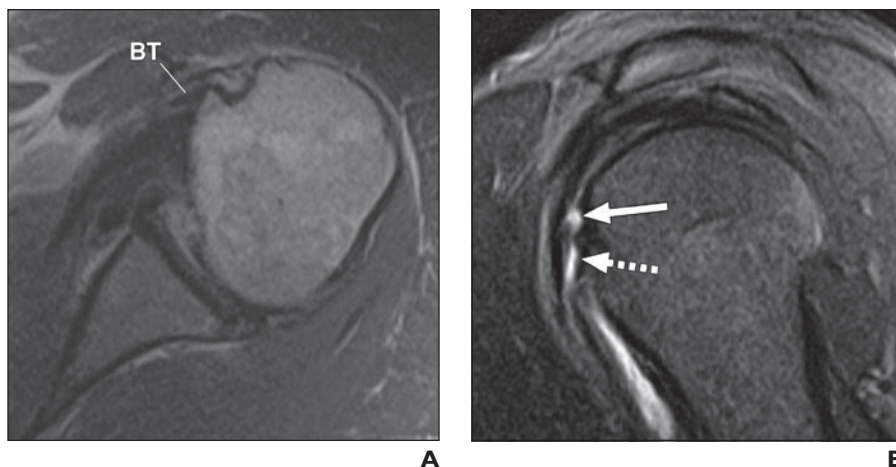

A **B**

Fig. 6—40-year-old man with arthroscopically proven traumatic biceps tendon dislocation.
A, Proton density–weighted axial image shows medial biceps tendon (BT) dislocation.
B, Sagittal STIR image shows empty fovea capitis (*solid arrow*) and empty lesser tuberosity (*dashed arrow*) related to rupture and retraction of superior glenohumeral ligament and subscapularis tendon, respectively.

ture (Fig. 5). The fused CHL–anterosuperior joint capsule also can be seen on axial sections just below the level of the coracoid process.

SGHL

Near its origin, sagittal images depict the round or oval cross section of the SGHL anterior to the biceps tendon (Fig. 5). When very thin sections are obtained through the rotator interval, this structure can be seen to run close to the course of the biceps tendon

from its origin at the supraglenoid tubercle to its insertion just above the subscapularis tendon at the lesser tuberosity. At the midportion of the rotator interval, it merges with the medial band of the coracohumeral ligament to form the biceps reflection pulley. Surprisingly, its distal insertion often can be seen in the sagittal plane when sought; it is of slightly higher signal intensity than the subscapularis and inserts on a small depression on the humeral head (Fig. 5).

Fig. 7—Bennett classification of biceps instability. Illustration shows normal appearance and lesions involving biceps pulley in axial plane. Rotator interval capsule is composed of coracohumeral ligament, superior glenohumeral ligament (SGHL), and capsular fibers, which all blend together along insertions medial and lateral to bicipital groove, maintaining biceps tendon within groove. Medial aspect of coracohumeral ligament (MCHL) blends with SGHL forming medial sheath (or MCHL–SGHL complex), which along with superior fibers of subscapularis tendon form medial supporting structures of bicipital groove. Lateral aspect of coracohumeral ligament (LCHL) blends with most anterior fibers of supraspinatus tendon forming lateral supporting structures of bicipital groove. Type 1 lesions are isolated tears of superior fibers of subscapularis tendon, resulting in medial subluxation of biceps tendon within groove. Type 2 represents tear of medial sheath (MCHL–SGHL complex), allowing medial subluxation of biceps tendon. Type 3 represents lesion of both medial sheath and subscapularis tendon, allowing medial dislocation of biceps tendon from bicipital groove. Type 4 involves LCHL and most-anterior fibers of supraspinatus tendon, allowing biceps tendon to dislocate anterior to subscapularis and coracohumeral ligament. Type 5 combines all structures (subscapularis tendon, medial sheath, LCHL, and supraspinatus tendon), which allows biceps tendon to dislocate either anteriorly or medially.
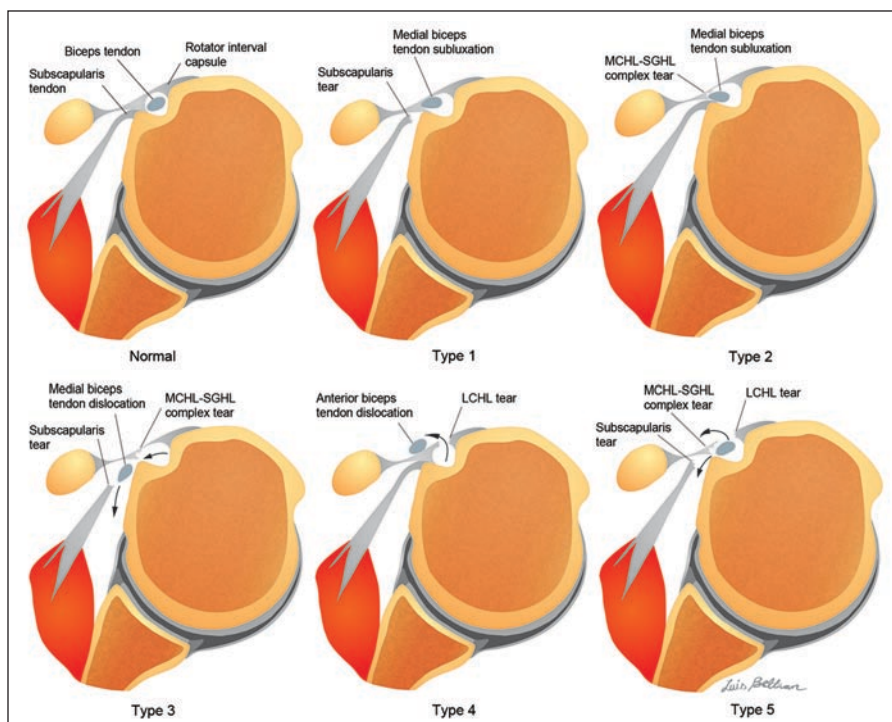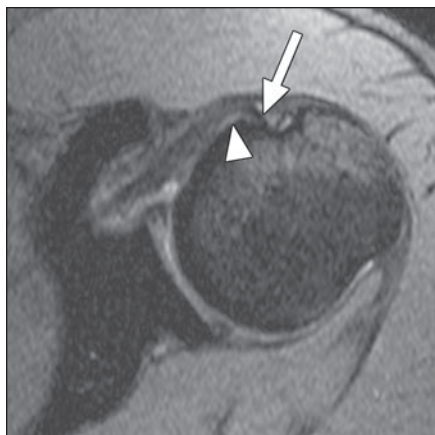
Fig. 8—26-year-old man with biceps instability, Bennett type 1. Axial gradient-recalled echo image shows type 1 lesion with tiny articular surface tear of cranial fibers of subscapularis tendon along lesser tuberosity attachment (*arrowhead*) associated with mild medial subluxation of biceps tendon (*arrow*) within bicipital groove.
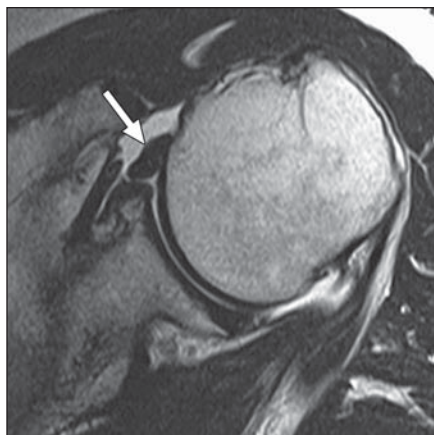

Fig. 9—39-year-old woman with biceps instability, Bennett type 3. Axial proton density–weighted MR image of type 3 lesion shows medial dislocation of biceps tendon (*arrow*) from bicipital groove.

Fig. 10—52-year-old man with biceps instability, Bennett type 4. Fat-saturated axial proton density–weighted MR image shows type 4 lesion with dislocation of biceps tendon anterior (superficial) to subscapularis tendon and coracohumeral ligament. LCHL = lateral coracohumeral ligament.

The reflection pulley represents a coalescence of the distal SGHL and medial band of the CHL. Before forming the pulley, the SGHL and CHL are seen as distinct and separate structures (Fig. 5A). As the two structures approach the lesser tuberosity, they form a T-shaped (Fig. 5B), then U-shaped structure (Fig. 5C), which provides support to the biceps pulley, the medial CHL forming the superior then anterior borders and the SGHL forming the anterior then inferior border as one progresses from medial to lateral. The pulley is best seen on sagittal images.

Rotator Interval Abnormality
Biceps Pulley Lesions

The biceps reflection pulley maintains the position of the biceps tendon within the bicipital groove. The reflection pulley can be injured with or without antecedent trauma. Traumatic injuries have been described after a fall on the outstretched arm in combination with full external or internal rotation as well as a backward fall on the hand or elbow [12, 13] (Fig. 6). Chronic injury can occur in patients with repetitive overhand activity [14].

The reflection pulley also may be secondarily affected in the setting of rotator cuff tears. Because of intimate insertion patterns, tears of the far anterior supraspinatus tendon footprint and superior subscapularis footprint (known as anterosuperior rotator cuff tears) may dissect to involve the CHL and SGHL, respectively. Destabilization of the

biceps tendon in this manner has been well published [14, 15]. In their study of 56 patients diagnosed with articular-side anterior supraspinatus tears by clinical examination and MRI, Habermeyer et al. [15] reported 73% involvement of the CHL at arthroscopy. The superior insertion of the intraarticular subscapularis tendon also provides medial support of the biceps tendon by contributing to the medial wall of the bicipital groove.

Biceps pulley injury can be difficult to diagnose; for this reason such injuries have been referred to as "hidden lesions" because they can be missed during open and arthroscopic examination [16]. Biceps pulley

injuries have been classified arthroscopically [6, 8] on the basis of lesions involving the subscapularis tendon, SGHL–MCHL complex, and the lateral CHL.

Bennett [8, 17] was the first to classify biceps subluxation–instability in 2001 and has since modified his classification on the basis of subsequent experience with arthroscopic diagnosis and treatment (Fig. 7). With this technique, injuries are described as involving the intraarticular subscapularis tendon (type 1) or the "medial sheath" (composed of the SGHL–MCHL ligament complex) (type 2) (Fig. 8), both the medial sheath and subscapularis tendon (type 3) (Fig. 9), the supraspi-
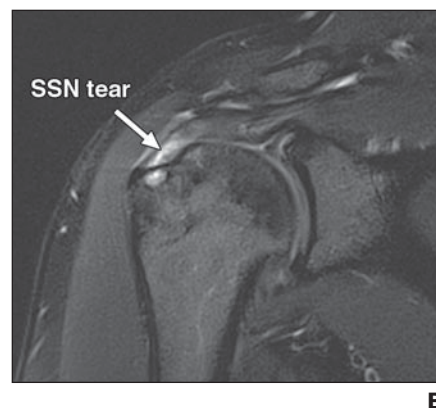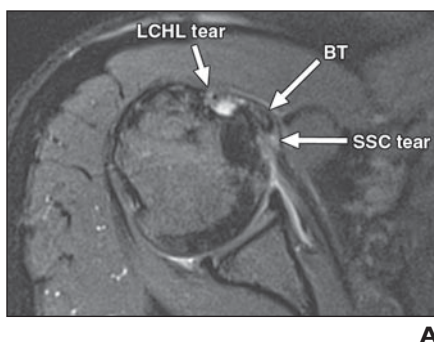

Fig. 11—46-year-old woman with biceps instability, Bennett type 5.
A and **B,** Axial fat-suppressed proton density–weighted (**A**) and coronal fat-suppressed T2-weighted (**B**) images show type 5 lesion with tearing of subscapularis tendon (SSC) along lesser tuberosity attachment associated with medial dislocation of biceps tendon (BT) from bicipital groove. There is also tearing of lateral coracohumeral ligament (LCHL) and supraspinatus tendon (SSN) along anterior leading-edge attachment. When both medial and lateral supporting structures of biceps pulley are disrupted, BT can dislocate either medially deep in relation to subscapularis tendon or anteriorly relative to coracohumeral ligament.
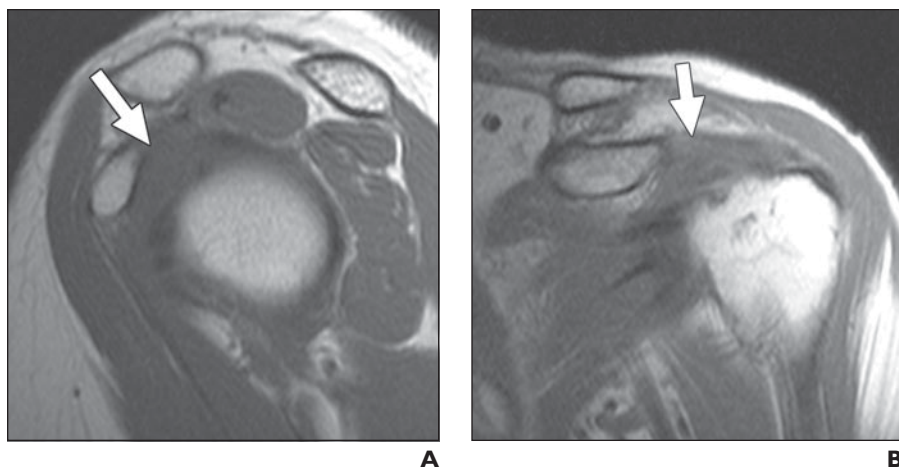
Fig. 12—65-year-old woman with adhesive capsulitis.
A and **B,** Sagittal T1-weighted (**A**) and coronal proton density–weighted (**B**) images show obliteration and replacement of normal rotator interval fat by hypointense soft-tissue signal (*arrow*) secondary to granulation or fibrous tissue.
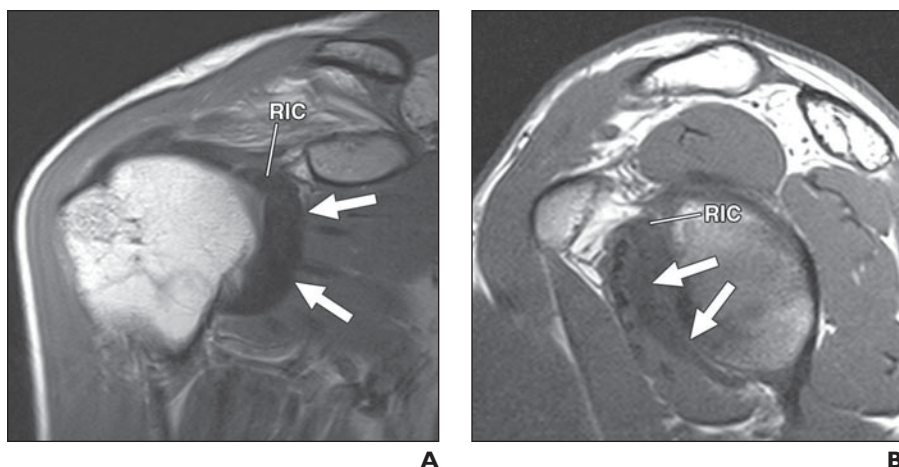

Fig. 13—28-year-old woman with adhesive capsulitis.
A and **B,** Coronal proton density–weighted (**A**) and sagittal T1-weighted (**B**) images show diffuse marked thickening of rotator interval capsule (RIC) and anterior joint capsule, which appears as thickened band of hypointense signal (*arrows*).

natus and lateral CHL (type 4) (Fig. 10), or all structures—intraarticular subscapularis tendon, medial sheath, supraspinatus tendon, and LCHL (type 5) (Fig. 11).

Type 1 lesions (Fig. 8) affect the subscapularis tendon and allow the biceps tendon to sublux into the medial aspect of the bicipital groove. When only the medial sheath is torn (type 2), the biceps tendon is also allowed to sublux medially within the bicipital groove. It is often not possible to distinguish between type 1 and type 2 lesions at MRI, and in both cases the biceps tendon is often "perched" on the lesser tuberosity. When the intraarticular subscapularis tendon and medial sheath are compromised (type 3), the biceps tendon can dislocate into the joint (Fig. 9).

Anterosuperior rotator cuff tears—that is, tears of the supraspinatus tendon that propagate into the rotator interval—are at risk for injuring the lateral coracohumeral ligament. Although not considered to be part of the biceps reflection pulley, this structure (and its relationship to the rotator interval capsule) provides important superolateral tension on the medial CHL with which it is contiguous. The combination of rupture of the LCHL and loss of the normal tension of the MCHL allows the biceps tendon to sublux superficial to the subscapularis tendon and coracohumeral ligament (type 4) (Fig. 10). When both lateral and medial stabilizing structures are injured, the biceps tendon is free to dislocate anteriorly or into the joint (type 5) (Fig. 11).

MRI has the potential to serve a vital role in preoperative diagnosis and surgical planning in the treatment of lesions of the biceps reflection pulley [10, 11, 18–21]. When the biceps tendon is perched on the lesser tuberosity, medially subluxed, or dislocated, injury to the biceps pulley structures can be inferred. Unfortunately, identifying abnormality in the component structures may not always be possible at MRI. This point was underscored by Weishaupt et al. [22], who in their review of 14 surgically proven biceps pulley lesions found that abnormalities of the superior border of the subscapularis tendon, seen on axial or parasagittal images, had high sensitivity, specificity, and positive and negative predictive values in diagnosing abnormalities of the reflection pulley. However, they were unable to reliably identify pulley lesions on the basis of imaging abnormalities of the component structures. Certainly, the discovery of a nonanatomically located biceps tendon should prompt close scrutiny of the structures of the rotator interval. We have found that, as a result of our cadaveric studies, our understanding of the anatomy of this complex region has evolved, and our ability to identify and delineate the SGHL, upper border of the subscapularis tendon, and CHL has allowed us to make more detailed assessments of this region. Undoubtedly, biceps pulley abnormality will be increasingly recognized by radiologists as imaging technology advances and as the use of higher-field-strength MR scanners and multichannel coils in routine clinical practice becomes more widespread.

Treatment

The choice of treatment of biceps pulley lesions remains controversial. Though there are few published studies on surgical treatment and repair of the biceps pulley [23, 24], the literature supports biceps tenodesis in these patients, and biceps tenodesis is the preferred method of treatment by the orthopedists in our institution.

Adhesive Capsulitis

Adhesive capsulitis is a relatively common clinically diagnosed condition that primarily affects middle-aged women. Patients present with painful restriction of motion that is worse at night. The pathology in adhesive capsulitis is felt to be the result of a cascade of events leading to thickening, contraction, and adhesion of the glenohumeral capsule, synovium, and glenohumeral ligaments, with resultant decreased capsular compliance [25, 26]. The CHL is considered
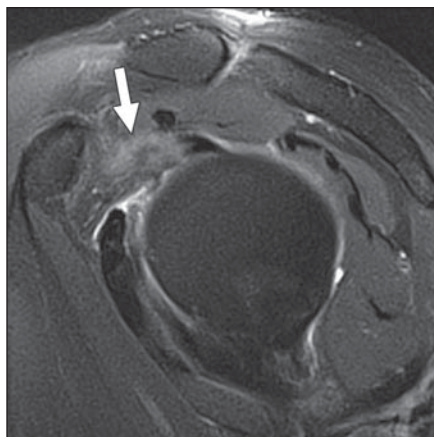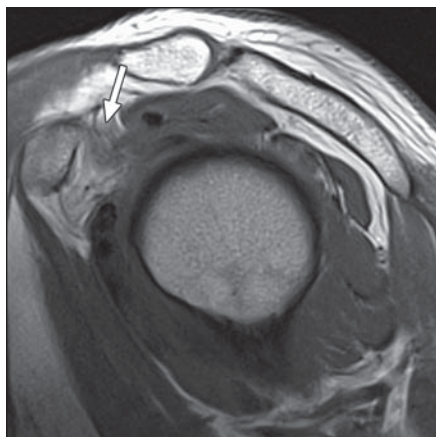
Fig. 14—43-year-old man with adhesive capsulitis. (Courtesy of Ilaslan H, Cleveland, OH)
A and **B,** Sagittal T1-weighted (**A**) and fat-suppressed T2-weighted (**B**) images show poor definition of coracohumeral ligaments with abnormal intermediate amorphous signal intensity (*arrow*) surrounding ligament.
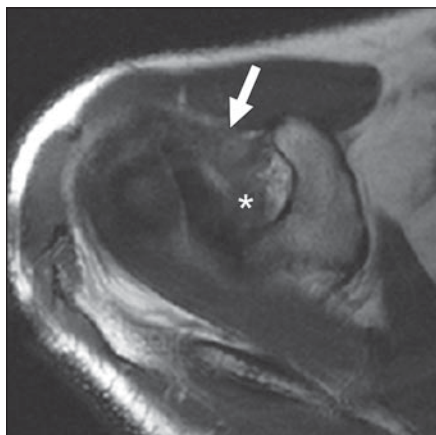

Fig. 15—45-year-old man with adhesive capsulitis. Axial T1-weighted image shows thickened coracohumeral ligament (*arrow*) and superior glenohumeral ligament (*asterisk*).
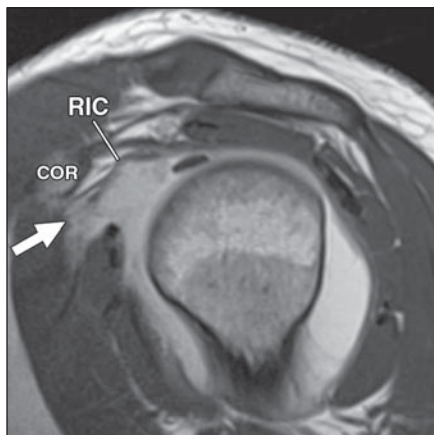

Fig. 16—30-year-old woman with arthroscopically proven lax rotator interval. Sagittal oblique T1-weighted MR arthrogram shows patulous and irregular contoured rotator interval capsule (RIC) (*arrow*) with contrast material extending into subcoracoid space. COR = coracoid process.

to be the key structure involved in the pathologic changes that lead to adhesive capsulitis [1, 27–29]. This normally pliant structure becomes rigid and inelastic, thus limiting external rotation.

Although the cause of adhesive capsulitis is unclear, studies have shown immunocytochemical evidence of both proliferative fibroblasts and acute and chronic inflammation [30–33].

Imaging Adhesive Capsulitis

Many imaging findings have been described in the diagnosis of adhesive capsulitis [26, 34–38]. These include synovial hypertrophy and debris [22] within the ro-

tator interval that can be seen on MRI as replacement of the normal rotator interval fat by granulation tissue or fibrous tissue (Fig. 12); thickening of the rotator interval capsule (Fig. 13); thickening of the ligaments of the rotator interval, SGHL, and CHL (Figs. 14 and 15); thickening of the joint capsule along the axillary pouch; and increased width of the axillary recess. Although several articles have tried to evaluate the sensitivity, specificity, and accuracy of these findings at MRI or ultrasound in the diagnosis of adhesive capsulitis [22, 34–40], the individual studies differ on measured endpoints, and meaningful conclusions about their findings are difficult to make. We performed our own

meta-analysis of these studies, which has not previously been published in the literature. Our analysis revealed that measures of axillary recess width and thickness of axillary recess capsule–synovium were the only commonly measured variables, and even these were only evaluated in three studies (Lefevre-Colau et al. [36], Jung et al. [34], and Lee et al. [35]). Although these endpoints were also evaluated in two additional studies by Mengiardi et al. [37] and Sofka et al. [38], they could not be included in the meta-analysis because the study by Mengiardi et al. did not provide the SD of results and the study by Sofka et al. did not evaluate control subjects. The Hedges *g* statistic was used to derive a standardized mean difference between cases (patients with a clinical or surgical diagnosis of adhesive capsulitis) and control subjects (patients without adhesive capsulitis) under a random effects model. The standardized mean distance represents an estimate of the difference between cases and control subjects in terms of the mean level of the outcome measure standardized using a pooled estimate of the SD and adjusted for heterogeneity among studies and small sample bias. The results indicated that there was no significant difference ($p > 0.5$) between cases and control subjects in terms of axillary recess width (standardized mean distance = 0.43; 95% CI for standardized mean distance, −3.12 to 3.98). However, the results indicated that the thickness of capsule–synovium in the axillary recess was significantly higher ($p < 0.001$) for patients than for control subjects (standardized mean distance = 1.54; 95% CI for standardized mean distance, 0.93–2.15). Other MRI findings specific to the rotator interval, such as thickening of the CHL and thickening of the rotator interval capsule were also evaluated by Mengiardi et al. [37]; however, there are not enough additional comparative studies evaluating the same endpoints available to perform a meta-analysis on the sensitivity and specificity of these findings.

In summary, we believe the role of MRI in the diagnosis of adhesive capsulitis at this time is limited. Adhesive capsulitis is primarily a clinical diagnosis and MRI serves only a supportive role in confirming the suspected diagnosis in clinically equivocal cases or early disease without significant symptoms.

Treatment

Adhesive capsulitis is considered to be a self-limited process progressing through distinct stages. Although complete resolution

is rare, adhesive capsulitis is permanently debilitating in only a small percentage of individuals. Treatment in nearly all cases is conservative and consists of supportive therapy, supervised and home-based exercise regimens, and nonsteroidal antiinflammatory drugs [41–44]. In the early stage of the disease, adhesive capsulitis can present without significant limitations in motion and easily can be mistaken for other shoulder abnormalities. In these cases, MRI may allow early diagnosis of the disease, which can then be clinically confirmed. Subsequently, treatment and progression of the disease are based on clinical findings, and MRI does not play a significant role.

Lax Rotator Interval With Instability

The glenohumeral joint is a ball-and-socket joint with the greatest range of motion of all human joints. Unlike the hip joint, where the femoral head is constrained by the bony acetabulum and motion is relatively restricted, the small arc and surface of the glenoid allow significantly greater freedom of humeral movement at the expense of rigid stability. Despite its relative lack of bony constraint, the shoulder is a stable joint, maintained by the balanced work of static and dynamic stabilizers.

In the midrange of motion, dynamic stabilizers are most responsible for joint stability. Muscle contraction compresses the humeral head against the concave surface of the glenoid and the labral rim; this generates concavity–compressive forces that maintain the humeral head in the center of the glenoid during physiologic range of motion. In contrast, the capsuloligamentous structures are redundant and lax throughout the midrange of motion and are only placed under tension once the joint approaches the limits of its range of motion.

It is believed that the glenohumeral ligaments, particularly the inferior glenohumeral ligament, are the most important static stabilizers of the joint [2]. The glenoid labrum functions more as a site of ligamentous attachment in contributing to shoulder stability than by providing increased depth to the glenoid fossa as previously believed [2, 45]. The middle and superior glenohumeral ligaments are thought to play a more minor role because they are frequently underdeveloped or absent [45, 46].

Rotator interval capsular lesions are believed to result in posterior and inferior instability of the glenohumeral joint [47]. In a cadaveric study by Harryman et al. [48], resection of the rotator interval capsule result-

ed in inferior and posterior translation of the adducted shoulder. Additionally, imbrication of the rotator interval capsule resulted in increased resistance to inferior and posterior translation. It is believed that the rotator interval capsule contributes to passive stability by providing an anatomic seal to the anterior superior joint capsule, allowing the generation of negative pressure between the humeral head and glenoid fossa [7, 8, 49]. The CHL is thought to contribute to joint stability by providing structural support; however, it plays a more minor role in static stabilization [10]. Other authors have reported that injury to the ligamentous structures of the rotator interval, particularly the SGHL and CHL, results in inferior instability [49–51]. Individuals with congenital defects of the rotator interval may be predisposed to glenohumeral instability [50, 52].

Imaging of Rotator Interval Laxity

An in vivo quantitative study of the rotator interval utilizing MR arthrography by Kim et al. [53] revealed statistically significant differences in the size and volume of the rotator interval between asymptomatic individuals and those with shoulder instability. A retrospective study of 120 shoulders, divided into groups with and without clinical chronic anterior instability, showed statistically larger rotator interval dimensions and calculated volume based on MR arthrography in those with clinical instability. Tears of the rotator interval capsule may manifest as thinning, irregularity, or focal discontinuity of the rotator interval capsule [18]. Extraarticular contrast material in the region of the rotator interval (such as in the subcoracoid space) may be identified at MR arthrography (Fig. 16) but not at routine MRI [19, 21] and can suggest loss of integrity of these structures, particularly in the setting of an intact rotator cuff. In their study, Vinson et al. [40] showed extension of intraarticular gadolinium to the cortex of the undersurface of the coracoid in five patients with arthroscopically proven lesion of the rotator interval who presented with shoulder instability.

Treatment

The rotator interval should be examined during any stabilization procedure of the shoulder joint. For patients with clinical findings consistent with an interval defect (i.e., excessive inferior translation with the shoulder in adduction and external rotation), the rotator interval tissue may be imbricated.

This can be performed arthroscopically or with an open exposure [52, 54–56]. In most of the patients, rotator interval closure represents a supplemental stabilization technique that is performed in conjunction with repair of other lesions. In select patients who show symptoms consistent with instability without a prior dislocation, isolated interval closure may also be considered to stabilize the shoulder joint [52]. However, this is a rare clinical presentation. For most patients with shoulder instability, there is capsular redundancy or a labral tear that must be surgically addressed. If these patients also show rotator interval defects, detected clinically or visually, the interval is tightened appropriately. It must be noted that the sutures for the rotator interval closure should be placed at least 1 cm lateral to the glenoid surface. Otherwise, the suture may also grasp and tighten the CHL and limit external rotation [57]. In theory, overtightening of the rotator interval could also lead to loss of other motion, but this has not been described in the literature.

Conclusion

Although a small and relatively difficult region to image, the rotator interval is an important region of the shoulder with respect to normal function. As its anatomy and function have become clearer through ex vivo and clinical studies over the past 20 years, interest in the diagnosis and treatment of pathologic conditions of the rotator interval has grown. Although further investigation is needed, it is clear that rotator interval abnormality will be increasingly recognized clinically and on imaging. With new technologic advancements in MR scanning capabilities, improved detection of rotator interval lesions including the individual structures that make up the biceps pulley is becoming a possibility. This improvement in imaging-based diagnosis will require better coordination between radiologists and orthopedic surgeons in terms of surgical planning. Further studies, including clinical effectiveness research, are necessary to establish MRI as a key component of presurgical workup in the clinical management of rotator interval abnormality.

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