

Current Concepts With Video Illustration

The Rotator Interval: Pathology and Management

Trevor R. Gaskill, M.D., Sepp Braun, M.D., and Peter J. Millett, M.D., M.Sc.

Abstract: The rotator interval describes the anatomic space bounded by the subscapularis, supraspinatus, and coracoid. This space contains the coracohumeral and superior glenohumeral ligament, the biceps tendon, and anterior joint capsule. Although a definitive role of the rotator interval structures has not been established, it is apparent that they contribute to shoulder dysfunction. Contracture or scarring of rotator interval structures can manifest as adhesive capsulitis. It is typically managed nonsurgically with local injections and gentle shoulder therapy. Recalcitrant cases have been successfully managed with an arthroscopic interval release and manipulation. Conversely, laxity of rotator interval structures may contribute to glenohumeral instability. In some cases this can be managed with one of a number of arthroscopic interval closure techniques. Instability of the biceps tendon is often a direct result of damage to the rotator interval. Damage to the biceps pulley structures can lead to biceps tendon subluxation or dislocation depending on the structures injured. Although some authors describe reconstruction of this tissue sling, most recommend tenodesis or tenotomy if it is significantly damaged. Impingement between the coracoid and lesser humeral tuberosity is a relatively well-established, yet less common cause of anterior shoulder pain. It may also contribute to injury of the anterosuperior rotator cuff and rotator interval structures. Although radiographic indices are described, it appears intraoperative dynamic testing may be more helpful in substantiating the diagnosis. A high index of suspicion should be used in association with biceps pulley damage or anterosuperior rotator cuff tears. Coracoid impingement can be treated with either open or arthroscopic techniques. We review the anatomy and function of the rotator interval. The presentation, physical examination, imaging characteristics, and management strategies are discussed for various diagnoses attributable to the rotator interval. Our preferred methods for treatment of each lesion are also discussed.

Neer is widely credited with describing the anterosuperior space between the subscapularis and supraspinatus tendons as the rotator interval. The rotator interval has since become recognized as a distinct anatomic space. The early, classic contributions of Neer and Foster,¹ Rowe and Zarins,² and Nobuhara

and Ikeda³ proposed biomechanical roles for structures within the rotator interval and fostered curiosity in its dysfunction. Subsequently, considerable effort has been devoted to better understanding the rotator interval, yet its pathoanatomical role remains controversial. As our knowledge of the biomechanical sig-

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nificance of the rotator interval structures evolves, so does our understanding of the disability it can create. This provides an opportunity to advance our ability to therapeutically intervene (Video 1, available at www.arthroscopyjournal.org).

ANATOMY

The rotator interval is a triangular space located in the anterosuperior portion of the glenohumeral joint. It is bounded by the supraspinatus superiorly and the subscapularis inferiorly, and the coracoid process forms its medial base. Contained within this triangular space are the coracohumeral ligament (CHL), middle glenohumeral ligament (MGHL), and superior glenohumeral ligament (SGHL), long head of the biceps tendon, and anterior joint capsule.

The configuration of these structures has been the subject of several anatomic descriptions. The presence and prominence of structures and foramen within the rotator interval exhibit considerable variability. Jost et al.⁴ offered 1 of the more comprehensive depictions based on 22 cadaveric shoulder specimens. They describe the medial and lateral rotator interval as unique anatomic structures (Fig 1). Laterally, the first layer consists of the CHL fibers following the subscapularis and supraspinatus to their respective insertions into the humeral tuberosities. Layer 2 is composed of meshing fibers of the bounding rotator cuff musculature and CHL. Layer 3 is primarily deep fibers of the CHL inserting on the greater tuberosity. The deep

layer is composed of the joint capsule and SGHL. Medially, 2 layers are described. The CHL makes up the superficial layer, and the deep layer comprises the SGHL and joint capsule.

The CHL is a broad, thin structure originating from the lateral coracoid base. It has an irregular trapezoidal shape and a variable insertion.⁵ Recent reports suggest that the CHL is histologically more similar to capsule than ligamentous tissue.⁵ Others have concluded that it is an essential biomechanical component of the rotator interval.⁶⁻¹⁰ Thus controversy remains regarding the physiologic role of the CHL.^{4,7,10,11}

The SGHL originates at the superior glenoid tubercle and inserts near the lesser tuberosity at the fovea capitis.¹⁰ It crosses the floor of the rotator interval and may merge with the CHL.⁵ These ligaments, with contributions from the bounding rotator cuff tendons, form the biceps reflection pulley. Traditionally, it was thought that the SGHL or CHL (or both) was the primary stabilizer of the long head of the biceps tendon.¹²⁻¹⁴ More recent anatomic reports challenge this belief and suggest that subscapularis tendon integrity is responsible for biceps tendon stability.¹¹

FUNCTION

The structures within the rotator interval are the subject of multiple in vivo and in vitro biomechanical studies, and many of these are contrasting.^{3,4,6,8-11,15-19}

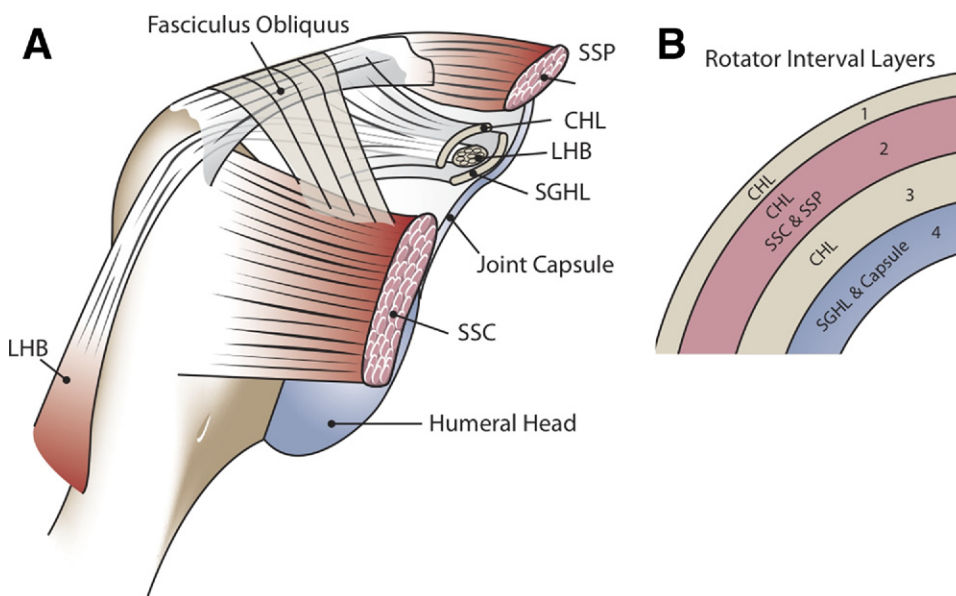


FIGURE 1. Depiction of rotator interval anatomy. (A) Gross representation of described rotator interval structures. (B) Cross-sectional representation of lateral rotator interval layers as described by Jost et al.⁴ The supraspinatus (SSP), CHL, long head of the biceps tendon (LHB), SGHL, and subscapularis (SSC) are depicted.

Harryman et al.¹⁰ classically reported 1 of the first comprehensive cadaveric studies examining the function of rotator interval structures. They showed that sectioning of the rotator interval capsule and ligamentous structures increased passive glenohumeral flexion, extension, external rotation, and adduction in 80 shoulders. Conversely, they showed that medial-to-lateral interval imbrication caused a reciprocal decrease in these motions. They concluded that the interval functions as a “check-rein” against excessive motion and limits posterior-inferior glenohumeral translation. This study substantiated a report from Nobuhara and Ikeda,³ who showed clinically that tightening the rotator interval, by externally rotating the shoulder, resulted in decreased posterior-inferior glenohumeral instability. Numerous clinical studies citing altered motion with interval closure support this proposed function, although the magnitude and direction of limitations are debated.²⁰⁻²² Therefore it is thought that structures within the rotator interval provide some degree of stability to the glenohumeral joint (Table 1).

Rotator interval structures also contribute to the stability of the long head of the biceps tendon. The CHL, SGHL, and subscapularis form a reflection pulley, supporting the bicipital tendon as it exits the glenohumeral joint. Evidence supports each as critical to the integrity of this structure.^{11,12,23} The transverse humeral ligament is an inconsistent anatomic structure.²⁴ When present, it appears to provide little biomechanical support within the groove despite its prominent location.²⁵ A third and, perhaps, less obvious role of the rotator interval is its function in maintaining negative intra-articular pressure. Venting of the glenohumeral capsule contributes to greater translation.^{26,27} Similarly, lesions of the rotator interval involving the capsule could also contribute to instability.

CONTRACTURES OF ROTATOR INTERVAL

Rotator interval contractures constitute a spectrum of disease ranging from mild rotator cuff impingement to debilitating adhesive capsulitis. In its most incapacitating form, it is characterized by painful motion and

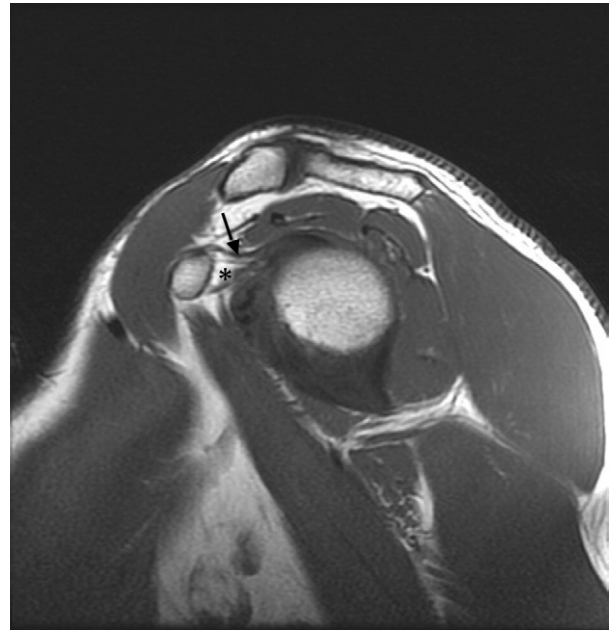


FIGURE 2. Sagittal oblique T1-weighted MRI scan showing normal appearance of CHL (arrow) and subcoracoid fat plane (asterisk). The normal laxity of the CHL should be noted.

frequently causes rest and night pain. It is thought to occur more frequently and respond less readily in diabetic populations.^{28,29} It can also occur in the post-operative setting. Active and passive motion limitations are characteristic, and abnormal scapulothoracic motion may exist. Discomfort is frequently localized to the deltoid insertion, and tenderness in the area of the coracoid is common.¹⁵

Radiographs are typically normal, and decreased capsular volume may be apparent with arthrography. Magnetic resonance imaging (MRI) studies report obliteration of the subcoracoid fat plane and anatomic changes in the rotator interval^{30,31} (Fig 2). Studies by Kim et al.^{31,32} showed significant decreases in rotator interval height, base, and area compared with normal shoulders. Others have identified thickening of the CHL and joint capsule within the rotator interval compared with normal age- and sex-matched controls^{30,33} (Fig 3).

The etiology of adhesive capsulitis is not completely understood. Injury, causing inflammation and scarring, has been proposed.^{3,17} Histologically, fibroblasts and myofibroblasts are prevalent, depositing dense matrices of collagen within the capsule.³⁴ Work is being conducted to identify abnormal expressions of cytokines, proteases, or growth factors that may be responsible for the abnormal regulation of these

TABLE 1. Proposed Rotator Interval Function

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| <ul style="list-style-type: none"> • Contributes to glenohumeral stability • Increases stability of long head of biceps tendon • Limits excessive glenohumeral motion |
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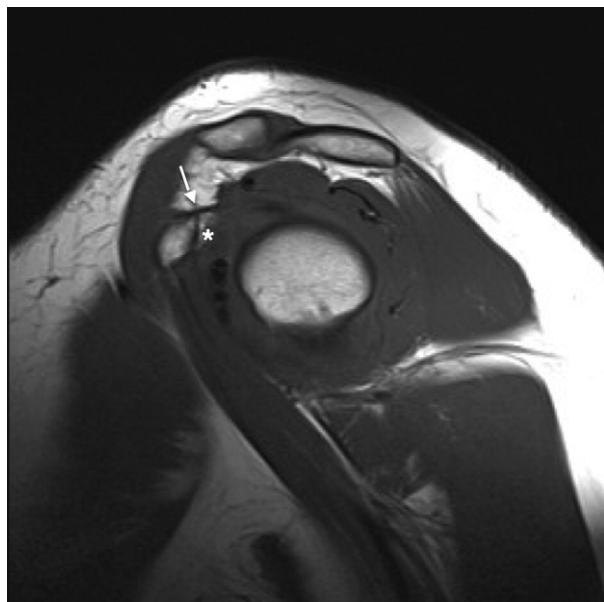


FIGURE 3. Sagittal oblique T1-weighted image showing thick, contracted CHL (arrow) and obliteration of normal subcoracoid fat plane (asterisk) in a patient with adhesive capsulitis.

cells.^{34,35} Regardless of its etiology, some believe that, ultimately, a contracted CHL is the “essential” lesion responsible for this entity.⁹

Symptomatic management is frequently all that is necessary to successfully treat adhesive capsulitis, because it is generally a self-limiting disorder. Various nonsurgical modalities are used including nonsteroidal agents, local injections, and physical therapy.²⁸ Satisfactory outcomes are reported in 90% of patients with idiopathic capsulitis after local steroid injection, whereas oral steroid administration appears less efficacious.^{28,36,37}

Recalcitrant cases may require manipulation or surgical soft-tissue release. Promising results have been reported for manipulation alone and both open and arthroscopic release techniques.³⁸⁻⁴² Post-manipulation gains in shoulder motion and function appear to be sustained long-term as noted by Farrell et al.³⁹ in their follow-up of 26 shoulders 15 years after manipulation. More recently, reports of combined manipulation and arthroscopic release have also yielded good results.⁴⁰⁻⁴³ In addition, hydrodilatation has been shown to be effective in properly selected patients.⁴⁴

Preferred Technique

The initial management is nonsurgical. Gentle motion exercises are prescribed, typically supervised by a

physical therapist. Aggressive therapy may worsen symptoms, especially in the early, painful stages of adhesive capsulitis. Corticosteroid injections are used to provide local symptomatic control and attempt to limit any inflammatory process.

If these measures fail, an arthroscopic interval release followed by a gentle manipulation under anesthesia is recommended. Introduction of the arthroscope must be done carefully in the contracted shoulder to avoid iatrogenic cartilage damage. Less force is required during manipulation, however, if the arthroscopic release is performed first. The beach-chair position and interscalene regional anesthesia are used. Indwelling catheters can be useful to facilitate early passive motion and decrease narcotic requirements.

The joint is insufflated before the surgeon carefully introduces the arthroscope. Visualization of the anterior-superior portion of the joint is usually possible, and an anterior working portal is established. A 4.5-mm oscillating shaver and electrocautery are used to debride synovitis and exuberant scar tissue (Fig 4). Once adequate visualization is established, a thick, robust capsule is usually confirmed. A hooked electrocautery device is used to divide the capsule just lateral to the glenoid labrum extending from the biceps tendon to the subscapularis. This release increases glenohumeral volume, allows the humeral head to lateralize, and improves visualization of the anterior-inferior gle-

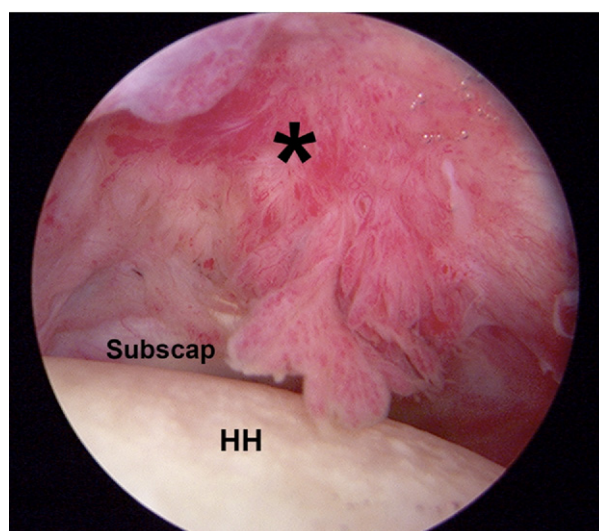


FIGURE 4. Rotator interval before arthroscopic release with synovitis (asterisk) in left shoulder viewed from posterior with patient in modified beach-chair position. (Subscap, subscapularis; HH, humeral head.)

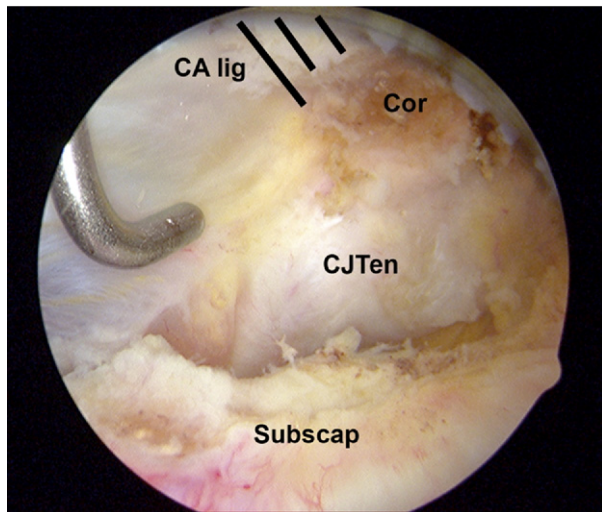


FIGURE 5. Rotator interval after arthroscopic release in left shoulder viewed from posterior with patient in modified beach-chair position. The following structures can be seen: coracoid (Cor), coracoacromial ligament (CA lig) with lines indicating its course, conjoined tendon (CJTen), and subscapularis (Subscap).

nohumeral joint. Additional release is performed in this area as necessary to ensure mobility of the subscapularis (Fig 5). A gentle manipulation is then performed to provide appropriate motion.

Postoperatively, adequate analgesia is critical to maintain motion established intraoperatively. Passive motion is begun shortly after surgery and continued at least daily for the first 2 weeks. Patients are then transitioned to a home exercise program with intermittent supervision individualized to the their progress.

ROTATOR INTERVAL LAXITY

The opposite end of the spectrum encompasses redundancy of rotator interval structures. Laxity may contribute to instability and resultant pain.^{4,7,10} Rowe and Zarins² reported an association between rotator interval lesions and patients with recurrent instability of the shoulder. They therefore recommended addressing these lesions during surgery. Despite this correlation, the precise contribution of structures within the rotator interval to glenohumeral stability is debated.

Patients presenting with laxity of rotator interval ligamentous or capsular structures often report a history of acute trauma or overuse injuries that eventually lead to tissue insufficiency and laxity. Many of these patients will complain of instability or early fatigue.

The sulcus sign may be present if inferior humeral translation is excessive and normally disappears in external rotation because of tightening of the rotator interval. The presence of a persistent sulcus sign despite glenohumeral external rotation may suggest pathologic laxity when symptoms are elicited.^{3,10,16,17}

Subtle inferior subluxation may be appreciated on radiographs, and although arthrograms are infrequently used, they may show contrast extravasation from interval lesions or abnormal contrast filling in abduction and external rotation.³ More recently, MRI and magnetic resonance arthrography have become popular imaging modalities because of the added soft-tissue detail these techniques afford. Increased rotator interval dimensions have been described in patients with chronic instability compared with controls.³² Other authors suggest that these measurements are not significantly different between various instability patterns and controls.⁴⁵

Symptomatic patients may require imbrication of rotator interval structures to decrease laxity. Isolated rotator interval foramen closure for recurrent instability has been reported with excellent results.¹⁶ It is important to recognize, however, that rotator interval lesions are often not solely responsible for glenohumeral instability. Failure to recognize other causes of instability will yield unsatisfactory results.⁴⁶ Levine et al.⁴⁶ reported on 50 patients undergoing revision shoulder stabilization. External rotation was asymmetrically limited by tight anterior structures in more than 20% of patients in this series. Yet, these patients remained unstable in abduction and external rotation because a patulous inferior glenohumeral ligament capsular complex was not addressed at the primary surgery. Therefore isolated interval closure is indicated for only select patients.

Arthroscopic and open procedures have been described to imbricate rotator interval tissues. The results of open coracohumeral imbrication of Harryman et al.¹⁰ are often quoted to justify closure of the rotator interval in certain situations. They showed decreased posterior and inferior translation after open medial-to-lateral imbrication of the CHL. These findings are frequently extrapolated to arthroscopic closure despite fundamental differences in the interval closure techniques. This has resulted in an interesting controversy regarding the true function of, and indication for, a rotator interval closure. In contrast to the CHL imbrication described by Harryman et al., the typical arthroscopic SGHL-to-MGHL closure more accurately depicts closure of the subscapularis foramen. Recent analysis of this technique by Provencher and col-

TABLE 2. *Rotator Interval Closure Pearls*

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- External rotation during closure minimizes joint contracture
 - Begin just lateral to coracoid and proceed laterally as necessary
 - Tie closure stitches extra-articularly to capture CHL for more robust closure
-

leagues^{20,47} and other authors⁴⁸ has failed to replicate findings reported by Harryman et al. By contrast, Savoie et al.^{49,50} report excellent clinical results using an oblique suture orientation, propose that it more accurately reproduces the results of Harryman et al., and suggest that it provides an increased measure of success in many cases. To our knowledge, no biomechanical data currently exist to support this conclusion. To this end, Farber et al.⁵¹ compared a medial-to-lateral arthroscopic technique using suture anchors with a superior-to-inferior technique in a cadaveric multidirectional instability model. The medial-to-lateral closure technique more closely restored normal motion, but the findings of Harryman et al. could not be confirmed despite use of a similar interval closure orientation. Mologne et al.²⁰ reported improvement in anterior translation after an arthroscopic SGHL-to-MGHL rotator interval closure. No improvement in posterior or inferior glenohumeral stability was shown. Therefore arthroscopic SGHL-to-MGHL closure may have a role in instability repair, but clinical outcome studies are lacking and the long-term efficacy is unclear. If interval closure is performed, tensioning the closure in 30° of external rotation may avoid motion limitations.⁵²

Preferred Technique

Satisfactory results have been reported both with and without rotator interval closure in posterior and

multidirectional instability procedures. Although the biomechanical role of an arthroscopic closure remains controversial, such a closure may be indicated in cases of a persistent sulcus sign despite external rotation or when subluxation persists despite adequate plication. In these cases an arthroscopic SGHL-to-MGHL closure is used as an extension of the capsular shift.

A rotator interval closure may be performed with the patient in either the beach-chair or lateral decubitus position (Table 2). The rotator interval is visualized from the posterior portal. The arm is externally rotated to minimize motion loss. An 8.25-mm cannula is positioned just outside the joint capsule. A curved shuttling device is then passed through the cannula, penetrating the capsular tissue (typically the MGHL) just superior to the subscapularis, 3 to 4 mm lateral to the glenoid. A suture is advanced into the joint. The cannula is redirected superiorly, and a penetrating instrument is passed anterior to the supraspinatus tendon through the SGHL base. The previously passed suture is retrieved through the same cannula in the anterior portal to avoid capturing the deltoid. Sutures are tied extra-articularly, capturing as much capsular tissue as possible. Additional sutures are placed laterally as necessary (Fig 6). Typically, 2 sutures are used, although in more severe cases, 3 can be placed. Postoperative rehabilitation is dependent on leading pathology, but external rotation is usually limited to 30° for the first 3 to 4 weeks.

BICEPS REFLECTION PULLEY TEARS

Early work by Petersson⁵³ illustrated medial biceps tendon dislocation in 3.3% of cadaveric specimens and suggested the relation between subscapularis integrity and stability of the biceps tendon. Subsequently, the biceps reflection pulley was described as being formed by contributions from the CHL, SGHL

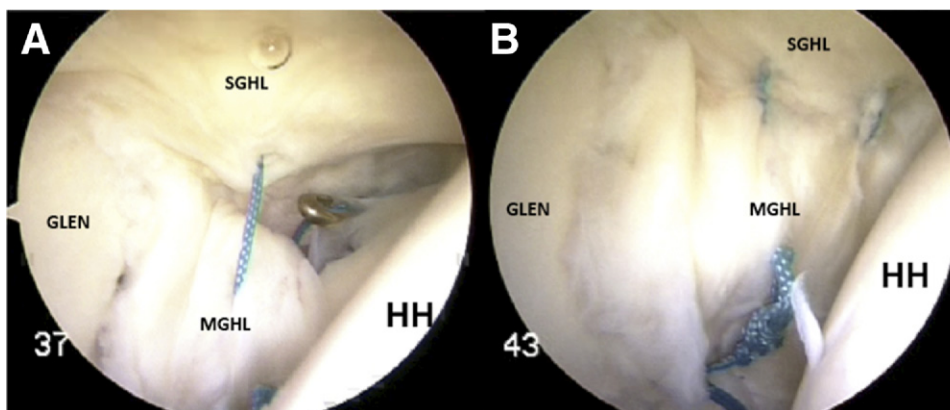


FIGURE 6. Right shoulder viewed from posterior before (A) and after (B) rotator interval closure with patient in modified beach-chair position. (A) Passage of first rotator interval suture from the SGHL to the MGHL. (B) Closed rotator interval after placement of 2 sutures. The humeral head (HH) and glenoid (GLEN) can also be seen.

and upper subscapularis.¹² In these patients the superficial CHL was always intact, and until the rotator interval was opened, the lesion was “hidden” and easily overlooked during both open and arthroscopic inspections.^{12,54}

It is established that injury to this pulley complex results in instability of the proximal biceps tendon.⁵⁵ Bennett⁵⁵ reported a classification system of biceps reflection pulley lesions based on the anatomic structure injured (Fig 7). If either the intra-articular subscapularis (type 1) or the medial head of the CHL (type 2) is incompetent, the biceps tendon will display increased intra-sheath mobility. When both the subscapularis and medial CHL are disrupted, the biceps dislocates intra-articularly (type 3). Less commonly, the biceps tendon may dislocate anterior to the subscapularis if the lateral CHL and leading edge of the

subscapularis are injured (type 4). Finally, when each structure is disrupted, complete loss of integrity of the bicipital sheath occurs (type 5). Although instability may occur in isolation, it is frequently associated with a subscapularis and leading-edge supraspinatus tear as an “anterosuperior” rotator cuff tear.^{6,55} In 1 series, internal anterosuperior impingement was found in 44% of patients with pulley tears.¹⁴ In addition to injury to the biceps reflection pulley, anterior-superior impingement is also thought to be responsible for injury to the biceps tendon. Boileau et al.⁵⁶ described a hypertrophic, hourglass-shaped biceps tendon entrapped within the bicipital groove. This impedes normal gliding during elevation and leads to mechanical block and subsequent pain.

Lesions of the biceps reflection pulley system are difficult to identify radiologically; however, several

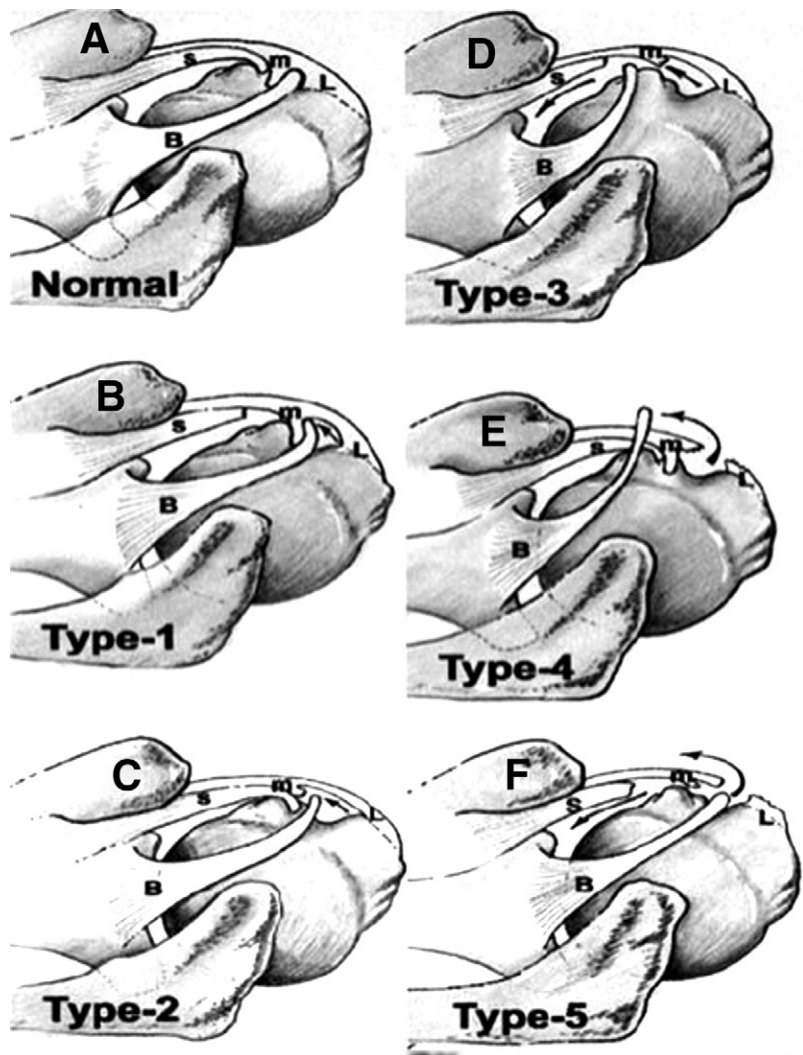


FIGURE 7. Classification system: normal (A); type 1, tear of subscapularis without involvement of medial head of CHL (B); type 2, tear of medial head of CHL without subscapularis involvement (C); type 3, tear of both medial head of CHL and subscapularis (D); type 4, tear of supraspinatus and lateral head of CHL (E); and type 5, tears of all stabilizing structures (F). (s, subscapularis; m, medial head of CHL; B, biceps; L, lateral head of CHL.) Arrows represent direction of biceps instability. (Reprinted with permission.⁵⁵)

subtle findings may be useful. Walch et al.¹² described the “pulley sign” as an extra-articular collection of contrast material anterior to the superior extent of the subscapularis. Its presence on magnetic resonance arthrography studies suggests injury to the biceps reflection pulley complex. Other authors suggest that extension of contrast to the cortex of the coracoid may be helpful in the preoperative diagnosis of rotator interval lesions.⁵⁷

Lesions of the biceps reflection pulley can be addressed surgically, but results are mixed. Reports of open repair by Walch et al.¹² included subscapularis reattachment and medial biceps sheath reconstruction. Of 22 patients in this series, 12 required scar removal or groove deepening to stabilize an enlarged tendon. This technique resulted in tendon rupture in 3 patients and modest pain improvement, thus leading the authors to conclude that biceps tenodesis is a more reliable treatment. Other authors, however, advocate an arthroscopic repair technique and report statistically improved pain, American Shoulder and Elbow Surgeons, and Constant scores at 2 years’ follow-up.⁵⁸ Although repair of the biceps reflection pulley remains controversial, most authors appear to support tenodesis of the biceps tendon to protect associated rotator cuff repairs. Biceps tenodesis can be accomplished through open or arthroscopic approaches by use of soft-tissue or bony repairs. The optimal technique has not been established, and many open and arthroscopic techniques have resulted in good reported outcomes.^{59,60}

Preferred technique

Our preference is to perform a subpectoral biceps tenodesis in patients with anterior shoulder pain and MRI or arthroscopic confirmation of damage to the biceps tendon or its pulley system. The beach-chair position with standard posterior viewing and anterior working portals are used for the arthroscopic portion of this procedure. The biceps tendon is advanced intra-articularly to visualize as much of the tendon as possible, and when indicated, it is cut at its base. The inferior border of the pectoralis major is palpated, and a longitudinal incision, centered over the inferior border of the pectoralis major tendon, is made. The reflected falx of the pectoralis major fascia is incised in line with the pectoralis tendon fibers at the inferior aspect of the pectoralis tendon. The horizontal fibers of the pectoralis tendon should be visualized to ensure appropriate location. Blunt dissection proximally, deep to the pectoralis major tendon and along the

anteromedial humerus, will reveal the long head of the biceps tendon in the bicipital groove. Retraction instruments are placed, and the biceps tendon is delivered into the surgical wound. It is trimmed approximately 2 cm proximal to its musculotendinous transition, and a No. 2 permanent suture is placed along its proximal 1.5 cm in a whipstitch fashion. A drill is used to fashion a tunnel approximately 15 mm in depth at the distal aspect of the bicipital groove. Care must be taken to ensure that the tunnel is drilled in the center of the humerus. An interference screw is used to fix the tendon within the bone tunnel and advanced until it is flush with the humeral cortex. The 2 ends of the suture are tied over the interference screw, enhancing screw-tendon security.

Postoperatively, a sling is used for no longer than 4 weeks, and the rehabilitation plan should follow that prescribed because of concomitant procedures. Resisted elbow flexion is not permitted for 6 weeks. If in isolation, passive glenohumeral and elbow motion should begin immediately and advance to active motion as the patient tolerates. Strengthening or heavy lifting should be withheld for the first 6 postoperative weeks.

CORACOID IMPINGEMENT

Coracoid impingement is a relatively well-established, yet less common cause of anterior shoulder pain (Table 3). Pain is presumed to occur because of impingement of the subscapularis between the coracoid process and lesser humeral tuberosity. It may be idiopathic in nature or result from trauma, instability, or iatrogenic causes.⁶¹ Although the coracoacromial interval involves the acromion, CHL, and coracoid process, it is the latter that is considered most responsible for altering the volume of the coracoacromial arch.⁶²

Russo and Togo⁶³ suggested that this syndrome occurs most commonly because of chronic overuse with the shoulder in a forward-flexed, adducted, and internally rotated position. These patients typically present with dull anterior shoulder pain exacerbated by bringing the lesser tuberosity into contact with the

TABLE 3. Coracoid Impingement

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- Tenderness in subcoracoid region
 - Pain in forward flexed, adducted, and internally rotated position
 - Coracohumeral interval <5 mm
 - Dynamic impingement at arthroscopy
-

TABLE 4. Key Points Regarding Coracoid Impingement

- Should be evaluated in any patient with other anterosuperior rotator cuff or biceps injury
- Subtle anterior instability should be assessed
- Diagnostic injection can be useful to localize symptoms
- Narrowed coracohumeral interval can be evaluated by computed tomography or MRI
- Dynamic examination confirms adequate resection of coracoid

coracoid process, and it appears to be more common in the midrange of flexion.⁶⁴ Tenderness can be elicited in the soft tissues surrounding the coracoid process and lesser tuberosity. Others have noted burning pain during aggravating exercise such as push-ups.⁶⁵ Injection of the subcoracoid region has been advocated as a diagnostic and therapeutic intervention.^{66,67} Validated diagnostic criteria do not exist, and therefore it remains largely a diagnosis of exclusion (Table 4).

Standard radiographs may detect some anatomic variations that can contribute to this syndrome; however, MRI or computed tomography is more useful in this respect. The coracoid index is measured by determining the distance the coracoid projects lateral to the glenoid articular surface on axial images. Dines et al.⁶⁷ reported the mean value in 67 normal shoulders to be 8.2 mm. A study by Friedman et al.,⁶⁸ using MRI to measure the coracohumeral interval, found that asymptomatic patients averaged 11 mm, with none less than 4 mm, in maximal internal rotation. By contrast, the mean coracohumeral interval in symptomatic patients was 5.5 mm, and it appears to narrow with internal rotation.^{68,69} Subsequently, this index on MRI was found to be only 5.3% sensitive but 97% specific for coracoid impingement.¹⁸ Other authors report no correlation between the coracohumeral interval and subscapularis injury in cadaveric speci-

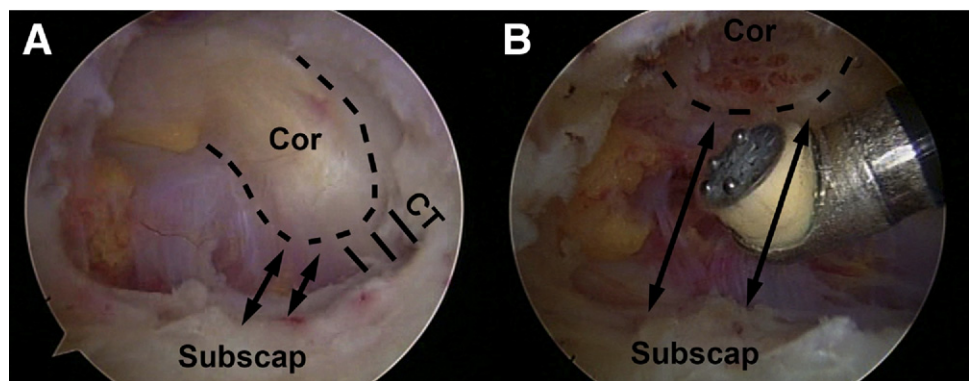
mens.⁷⁰ They propose that functional anterior instability narrows the coracohumeral interval and may be more important than static impingement in the setting of full-thickness rotator cuff tears.⁷⁰ Therefore imaging may support but cannot establish this diagnosis.

The first line of treatment should include a trial of activity modification and rotator cuff and scapular strengthening activities. If nonsurgical modalities fail, surgical decompression may be necessary. Options to accomplish this include open or arthroscopic coracoplasty. It is also intuitive that anterior instability could narrow the coracohumeral interval and, if indicated, should be appropriately stabilized before coracoplasty.⁷¹ Open coracoplasty is accomplished through a deltopectoral approach allowing removal of the lateral 1.5 cm of the coracoid. This requires protection of the musculocutaneous nerve and repair of the conjoint tendon more medially after its dissection.^{65,67} Other authors believe that isolated coracoid impingement is rare and advocate resection of the coracoacromial ligament and acromioplasty in addition to partial coracoid resection.⁶¹ More recently, arthroscopic techniques have been popularized.^{72,73} The described advantage is avoiding detachment of the conjoint tendon and less tissue dissection. Outcome studies detailing this relatively rare diagnosis are limited, but authors typically report reasonable outcomes for both open and arthroscopic procedures.^{67,73}

Preferred Technique

Arthroscopic coracoplasty is our preferred technique to address coracoid impingement. It avoids detachment of the conjoint tendon and facilitates treatment of other concomitant intra-articular pathology. The coracoid and subcoracoid space should be examined in patients with long head biceps and biceps reflection pulley tears, subscapularis tears, and ante-

FIGURE 8. Right shoulder viewing from posterior with patient in modified beach-chair position. (A) Coracoid (Cor) before coracoplasty. (B) Coracoid after coracoplasty. Arrows indicate the increased distance to the subscapularis (Subscap) after coracoplasty. (CT, conjoint tendon.)



rior supraspinatus tears because of the high association with coracohumeral impingement as suggested by Braun et al.^{74,75} We perform this procedure with patients in the beach-chair position under regional anesthesia using standard posterior viewing and anterior working portals. A coracoplasty can be performed by a transarticular approach or an extra-articular approach. Most commonly, we use a transarticular approach.⁷⁶ The capsule between the SGHL and MGHL is opened with a shaver or radiofrequency device. The coracoacromial ligament fibers serve as an excellent landmark leading to the lateral side of the coracoid safely. Radiofrequency ablation is used to clear the posterior and lateral aspects of the coracoid. The conjoint tendon and coracoacromial ligament are preserved, with removal of the lateral and posterior coracoid with an arthroscopic bur. The anterolateral 1 to 1.5 cm of the coracoid is typically resected, and dynamic examination should confirm sufficient resection. Excessive resection risks coracoid fracture, and neurologic injury is possible inferior to the coracoid (Fig 8).

Postoperatively, rehabilitation is tailored based on other attendant procedures. If performed in isolation, rehabilitation should be similar to that of an isolated subacromial bursectomy. Motion begins immediately, and active range of motion is encouraged as early as discomfort will allow. We recommend that positions of impingement be avoided for the first 2 postoperative weeks.

CONTROVERSIES AND FUTURE DIRECTIONS

The rotator interval continues to be an area of intense interest for shoulder surgeons. Although precise roles for the named ligamentous and capsular structures are debated, our ability to satisfactorily treat pathology of the rotator interval has become more reliable. Despite these advances, controversy remains in several areas. It is apparent that traditional arthroscopic rotator interval closure techniques have not to date successfully reproduced the biomechanical results of open imbrication. This, in combination with equivocal clinical results, has resulted in uncertainty regarding its utility. Other authors hold that rotator interval closure is a critical aspect of shoulder reconstruction for all patients. More detailed analysis is necessary to determine the indication and optimal technique for rotator interval closure.

Similarly, the etiology and prevalence of coracoid impingement remain contentious. Although coracoid

impingement appears to be highly associated with other anterior shoulder damage, it is unknown whether it contributes to or results from these associated injuries.^{70,74} Biomechanical and clinical validation of intervention techniques will be necessary to establish a more precise role for surgical treatment. We will also likely see increased utilization of biologic intervention, through growth factor and enzymatic regulation, to augment or avoid surgical management in many scenarios. These efforts will ultimately continue to improve outcomes for many complex shoulder patients.

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