The rotator interval (RI), composed of many of the vital structures that play a critical role in the function of the shoulder, is implicated in a range of pathologic conditions. Its complex anatomy can make a correct diagnosis challenging. Knowledge of the normal appearance and common pathology of the RI is important for the treatment of many shoulder conditions.

Anatomy

During its intra-articular course, the long head of the biceps (LHB) passes through the RI, a triangular defect in the rotator cuff formed by the protrusion of the coracoid process between the tendons of the supraspinatus and subscapularis. It is located in the anterosuperior part of the shoulder and was first defined by Neer et al.1

This space is bordered superolaterally by the anterior edge of the supraspinatus tendon and inferiorly by the upper edge of the subscapularis tendon. The base of the triangle corresponds to the base of the coracoid process and the apex of the transverse ligament. Within the RI, the long head of the biceps tendon (LHBT) is stabilized by a capsuloligamentous complex called the bicipital pulley, formed by the coracohumeral and superior glenohumeral ligaments.2

The coracohumeral ligament (CHL) originates from the glenohumeral joint capsule. It broadens to merge with the RI capsule, and splits laterally into two bands that span the bicipital groove. The larger lateral band of the CHL inserts on the anterior edge of the supraspinatus tendon and greater tuberosity. The smaller medial band inserts on the subscapularis tendon (SSC), the transverse humeral ligament, and the lesser tuberosity (►Figs. 1, 2, 3, 4). The fibers of the CHL interdigitate with those of the superior supraspinatus and superior subscapularis tendon.

The superior glenohumeral ligament (SGHL) is a thickening of the glenohumeral joint capsule that typically originates from the superior glenoid labrum next to the supraglenoid tubercle. It crosses the floor of the RI deep to the CHL and LHBT, and inserts onto a small depression called the fovea capitis of the lesser tuberosity3 (►Fig. 1). As the SGHL crosses the floor of the RI, it may fuse with the medial CHL. The LHBT is between the capsuloligamentous sling formed by the CHL and the SGHL. These structures help maintain the LHB tendon within the bicipital groove.4

A less widely studied part of the RI is the coracoglenoid ligament that arises from the superior glenoid labrum next to the supraglenoid tubercle. It crosses the floor of the RI deep to the CHL and LHBT, and inserts onto a small depression called the fovea capitis of the lesser tuberosity. As the SGHL crosses the floor of the RI, it may fuse with the medial CHL. The LHBT is between the capsuloligamentous sling formed by the CHL and the SGHL. These structures help maintain the LHB tendon within the bicipital groove.5

Distal to the biceps pulley, the LHBT exits the articular joint space and enters the bicipital groove between the greater and lesser tuberosities. The LHBT sheath is a synovial reflection.
that is in continuity with the glenohumeral joint capsule.\textsuperscript{6} Within the proximal bicipital groove, the LHB is covered by the “transverse ligament” that represents the confluence of the subscapularis, supraspinatus, and coracohumeral ligaments.\textsuperscript{5,7} However, the transverse ligament is thought to play a relatively minor role in stabilizing the LHBT.\textsuperscript{8}

Within the LHBT sheath, a membrane extending from the humeral groove to the LHB, known as a vinculum, may be seen (\textsuperscript{\textasteriskcentered}Fig. 5). It is believed to contribute to the vascular supply of the tendon and may have a role in preventing tendon reaction and Popeye deformity following intra-articular LHBT rupture.

**Long Head Biceps Tendon Variants**

A wide range of anatomical variants of the LHBT have been reported, such as aberrant intra- and extra-articular tendon origins and congenital absence. These variants are reported
to occur with a frequency of 1.9 to 7.4%. Dierickx et al developed a classification of 12 variant types. The most common subgroup is mesotenons, where a synovial band connects the LHB to the adjacent rotator cuff or capsule that may act as a block to tendon sliding. Another well-recognized subtype is adherent variants, where the LHBT adheres to the adjacent rotator cuff tendons or capsule. The relationship between these variants and the incidence of shoulder joint pathology remains controversial. However, absence of the LHBT was reported to be associated with other congenital abnormalities, such as spina bifida, congenital inguinal hernia, limb abnormalities, VATER syndrome, glenoid dysplasia, and multidirectional instability.

Accessory heads of the LHBT are present in 9.1 to 22.9% of people, depending on ethnic group (►Fig. 6). Supernumerary bicipital heads may arise from the superior joint capsule of the glenohumeral joint, from the greater or lesser tuberosities, or from the coracoid process. In cases of a double tendon origin, some authors have suggested that the structure arising from the superior joint capsule represents an aponeurotic expansion rather than a true tendinous structure.

Instability of the Long Head of Biceps Tendon

Instability of the LHBT is a common cause of shoulder pain secondary to disruption of the RI structures. The capsuloligamentous sling formed by the SGHL and CHL stabilizes and buttresses the LHB as it curves sharply from the intertubercular groove to the supraglenoid tuberosity (►Figs. 1 and 8).

In addition to the biceps pulley ligaments, the superior insertion of the subscapularis tendon is thought to play a crucial role in maintaining integrity of the biceps pulley. The superior most fibers of the SGHL attach to the upper margin of the lesser tuberosity. A further tendinous slip extends superiorly and passes deep to the LHBT, inserting onto the fovea capitis of the humerus (►Figs. 7, 8, and ►Fig. S1). This helps prevent anteromedial displacement of the LHBT. Disruption of the rotator cuff tendons may often extend to involve the biceps pulley structures with associated LHBT instability.

The initial findings of biceps instability may be subtle with minor medial displacement of the LHBT. However, severe subluxation or dislocation of the LHBT from the bicipital groove may be readily shown on imaging. Several classification systems have been proposed. Bennett described five patterns of biceps instability; Walch et al and Habermeyer et al proposed four types of bicipital dislocation. More recently, Martetschläger et al proposed a simplified
classification of direct pulley lesions: type 1, lesion of the
medial pulley (medial CHL and/or SGHL); type 2, lesion of the
lateral pulley (lateral CHL); and type 3, lesion of the medial
and lateral pulley slings.  

In type II injuries, there is injury to the medial SGHL part
of the bicipital pulley with mild medial subluxation of the
LHBT. However, the intact subscapularis tendon prevents further dislocation (► Fig. S3).

In type III injuries, there is injury of the SGHL and an
intrasubstance tear of the subscapularis tendon that allows
for extra-articular dislocation of the LHBT. However, intact
depth fibers of the subscapularis tendon prevent intra-artic-
ular dislocation (► Fig. S4).

In type IV injuries, disruption of the CHL roof of the
bicipital pulley allows for extra-articular tendon dislocation
that may lie superficial to the subscapularis tendon. These
injuries of the lateral parts of the bicipital pulley are often
associated with partial- or full-thickness tears of the supra-
spinatus tendon (► Fig. S5).

In type V injuries, tears of the medial and lateral limbs of
the coracohumeral and superior glenohumeral ligaments,
combined with a full-thickness tear of the subscapularis tear
of the SGHL, allows for intra-articular dislocation of the
LHBT. Intact inferior fibers of the subscapularis tendon mean
that the LHBT moves from a dislocated intra-articular posi-
tion anterior to the subscapularis inferiorly (► Fig. S6).

In type VI injuries, there is a tear of the medial parts of
the bicipital pulley and detachment of the subscapularis tendon
that allows the medially dislocated LHBT into the glenohum-
eral joint space (► Fig. S7).

Imaging of Long Head of Biceps Pulley

In isolated pulley lesions, although the LHB may be unstable
in its intra-articular course, it may not show gross instability
of the extra-articular tendon. One indirect sign of a pulley
injury is the so-called chondral print where increased mo-
bility of the LHB erodes the humeral chondral surface.
Although initially an arthroscopic finding, 23 Zappia et al
reported this sign could be detected on ultrasonography
(US) with high rates of sensitivity, specificity, and diagnostic
accuracy for chondral print confirmed on arthroscopy 24
(► Fig. 9).
The ligaments of the bicipital pulley are often difficult to show on standard magnetic resonance imaging (MRI) sequences, and MR arthrography may allow for more accurate evaluation of the LHB and bicipital pulley injuries (\textsuperscript{►}Fig. 2). Schaeffeler et al showed high accuracy in the detection of isolated pulley lesions using anterior and inferior displacement of the LHBT on sagittal oblique MR arthrogram images called the “displacement sign” on the oblique sagittal sequences.\textsuperscript{25}

Both US\textsuperscript{4,26} (\textsuperscript{►}Fig. 10) and MRI\textsuperscript{27,28} (\textsuperscript{►}Fig. 11) have excellent diagnostic accuracy in identifying dislocation and subluxation of the LHBT. Frank subluxation of the LHB can be identified with medial displacement of the LHBT over the lesser tuberosity. It can then be seen anterior to the subscapularis tendon (\textsuperscript{Fig. 12}), within a tear of the subscapularis tendon (\textsuperscript{►}Figs. 13 and 14), or deep to the subscapularis tendon within the glenohumeral joint (\textsuperscript{►}Fig. 15). Rarely, the LHB tendon may be seen to dislocate posteriorly, which may be associated with a history of anterior glenohumeral dislocation (\textsuperscript{►}Fig. 16).

MR arthograms may also be useful in showing tears of the superior subscapularis tendon that may extend to involve the medial limb of the biceps pulley or the anterior leading edge of the supraspinatus tendon that may involve the lateral parts of the bicep pulley.
Degeneration of the LHBT may occur secondary to a wide variety of pathologies, such as subacromial impingement, tendon instability, or tendon entrapment. Tendon degeneration may occur at any level of the tendon and is most commonly secondary to repetitive mechanical stresses. In addition, a consistent zone of hypovascularity is reported to be found in the region of the LHBT most often prone to rupture. This area extends from midway through the gleno-humeral joint to the proximal intertubercular groove. It may contribute to the risk of tendon degeneration that results in a range of histopathologic changes characterized by mucoid fibrous changes, increased vascularization, infiltration and replacement by adipocytes, and frequent chondrocytic/chondrometaplasia differentiations. These tendinopathic changes are often associated with insidious, progressive chronic pain. Continued repetitive stresses may result in progressive tendon fibrillation, macroscopic partial tears, and eventually complete rupture.

Tendon thickening, flattening, or deficiency of the tendon may all be associated with tendon degeneration. In tendinopathy, the LHB may appear hypoechoic on US, and color Doppler imaging may be useful to identify local tenosynovitis. LHBT MRI signal intensity may be difficult to assess due to the magic angle artifact and the abruptly curved course of the tendon. Magic angle artifact typically involves a short segment of the LHBT where the intratendinous collagen fibers are closest to the magic angle of 55 degrees relative to the main magnetic field, with focal increased signal on short TE images but normal signal on the corresponding T2-weighted images (Figs. 7 and 8).

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Rupture of the LHB tendon is rare in a healthy tendon and usually associated with chronic tendon degeneration. Partial- and full-thickness tears typically occur within an area of relative hypovascularity, extending from midway through the glenohumeral joint to the proximal intertubercular groove. A challenging subgroup of these injuries are partial-thickness tears of the LHBT proximal to the bicipital groove, known as “groove entry lesions.” Magic angle artifact may obscure these injuries, and they are best detected on US or on long TE sequences.

Complete rupture of the LHBT may result in a “Popeye” deformity where retraction of the LHBT and muscle results in a prominent bulge over the anterior lateral part of the proximal arm. Complete rupture of the LHBT often results in resolution of pain from underlying tendinosis, partial tear, or instability. Arthroscopic tenotomy is widely practiced, and more recent articles have described a technique for percutaneous US-guided tenotomy of LHBT. Partial-thickness tears of the LHBT may be detected as a focal change in tendon caliber. Intrasubstance delamination tears appear as longitudinally oriented intratendinous splits on US or longitudinally oriented intratendinous hyperintense fluid signal on MRI.
Frozen Shoulder

Frozen shoulder is a common condition characterized by globally restricted shoulder movement where radiographic findings other than osteopenia are absent. The underlying pathology is not well understood; however, it is believed to result in adhesion, thickening, and contraction of the glenohumeral capsule and glenohumeral ligaments that results in reduced capsular compliance. The key structure usually affected first is the CHL that forms the roof of the rotator cuff interval. Tension of the CHL has an important role in the stability and range of motion (ROM) of the glenohumeral joint, particularly in external rotation. The CHL normally consists of loose connective tissue and therefore is relatively flexible. However, this may be altered in frozen shoulder when there is contracture of the CHL, consisting of a dense matrix of type III collagen populated with fibroblasts and myofibroblasts. Contraction of the CHL limits external rotation of the arm and is then followed by thickening and contraction of the glenohumeral joint capsule, which may further limit the ROM. Surgical studies reported that coracohumeral release is associated with significant recovery of shoulder range of movement.

Although frozen shoulder is primarily a clinical diagnosis, several typical imaging findings have been reported, such as thickening of the CHL (Fig. 21), replacement of the normal RI fat, and thickening of the axillary recess or soft tissue edema within the RI and axillary recess soft tissues (Fig. 22). However, the end points used in earlier imaging studies have been highly variable, preventing further meta-analysis. Recent studies also showed that the stiffness of the CHL can be assessed using shear wave elastography and is increased in cases of frozen shoulder, although further research is indicated to investigate the clinical utility of this finding.

Although conservative treatment is often pursued, frozen shoulder may result in permanent disability in a small number of individuals. Physiotherapy, steroid injections, manipulation under general anesthesia, and arthroscopic capsular release are commonly used treatments. Targeted injection of the RI (Fig. 24) has been proposed as a potential treatment for frozen shoulder with promising results. More recently, the combination of RI injection and immediate manipulation of the glenohumeral joint under local anesthetic block was reported to result in significant improvement in ROM and patient pain.

Rotator Interval Laxity

The glenohumeral joint has the greatest ROM of all human joints. Stability depends on a balance of static and dynamic stabilizers. Muscle contraction compresses the humeral head against the glenoid during physiologic ROM. The normal capsuloligamentous structures of the shoulder are redundant and lax throughout the midrange of shoulder motion and are only under tension when the joint approaches the limits of its ROM. The inferior glenohumeral ligament is believed to be the most important static stabilizer of the shoulder. This contrasts with the middle and superior glenohumeral ligaments that are often underdeveloped or congenitally absent and believed to play a relatively minor role in maintaining joint stability. Some authors have suggested that RI capsule injuries may result in posterior and inferior glenohumeral joint instability and that patients with congenital deficiency of the RI may be at increased risk of inferior instability. Injury or deficiency of the RI structures may be well shown on MR arthrogram. Extension of intra-articular contrast into the subcoracoid space has been associated with arthroscopically confirmed RI injuries in patients with shoulder instability.
Conclusion

The RI is an important anatomical region that plays a critical role in normal shoulder function. Although this area of complex anatomy can be difficult to image, further understanding of the typical and pathologic radiologic appearances of the RI structures and LHBT may allow for more accurate diagnoses and improved treatment options.

Conflict of Interest
None declared.

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Fig. 23 Measurement of the elastic modulus of the coracohumeral ligament using shear wave elastography.

Fig. 24 Transverse ultrasonography image of the rotator interval (RI) with the biceps tendon at the center of the image. Using a long-axis needle approach, the needle tip lies between the coracohumeral ligament, as it forms the roof of the RI, and the long head of biceps tendon below.
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