The biceps pulley or “sling” is a capsuloligamentous complex that acts to stabilize the long head of the biceps tendon in the bicipital groove. The pulley complex is composed of the superior glenohumeral ligament, the coracohumeral ligament, and the distal attachment of the subscapularis tendon, and is located within the rotator interval between the anterior edge of the supraspinatus tendon and the superior edge of the subscapularis tendon. Because of its superior depiction of the capsular components, direct magnetic resonance arthrography is the imaging modality of choice for demonstrating both the normal anatomy and associated lesions of the biceps pulley. Oblique sagittal images and axial images obtained with a high image matrix are valuable for identifying individual components of the pulley system. Various pathologic processes occur in the biceps pulley as well as the rotator interval. These processes can be traumatic, degenerative, congenital, or secondary to injuries to the surrounding structures. The term hidden lesion refers to an injury of the biceps pulley mechanism and is derived from the difficulty in making clinical and arthroscopic identification. Pathologic conditions associated with pulley lesions include anterosuperior impingement, instability of the biceps tendon, biceps tendinopathy or tendinosis, superior labrum anterior and posterior lesions, and adhesive capsulitis. It is important to be familiar with the normal appearance of the biceps pulley so that abnormalities can be correctly assessed and effectively managed.

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Introduction
Abnormalities of the rotator interval are increasingly being recognized as causes of shoulder pain and discomfort. Within this anatomic area lies a complex pulley system that stabilizes the long head of the biceps tendon (LBT). The difficulties in clinical and arthroscopic evaluation of this region highlight the importance of both pre- and posttreatment imaging assessment (1). Although lesions of the biceps pulley can optimally be imaged with magnetic resonance (MR) arthrography, they are frequently overlooked or inaccurately assessed, and, if left untreated, they may result in persistent shoulder pain. Recognizing the complex anatomy of the pulley system and its close association with the surrounding structures, as well as understanding the mechanisms and patterns of injury, are essential for guiding appropriate case management.

In this article, we discuss the biceps pulley in terms of normal anatomy, function, MR imaging techniques, and MR arthrographic features. In addition, we discuss the MR arthrographic findings of various pathologic processes found in the anterosuperior aspect of the shoulder, and correlate these findings with arthroscopic findings.

Normal Anatomy

Glenohumeral Joint
The glenohumeral joint consists of the spheric head of the humerus and the shallow glenoid fossa of the scapula. It is known as the most mobile joint in the body and the most frequently dislocated large joint, owing to its relative lack of osseous constraints. Its stability, both static and dynamic, depends on the surrounding muscle and soft-tissue structures (2,3).

Glenoid Labrum
As a fibrocartilaginous extension of the glenoid fossa, the glenoid labrum deepens and increases the surface area of the glenohumeral articulation. It varies in size and thickness, with a base that is attached to the margin of the fossa and is triangular in cross section. The labral attachment is sometimes partially deficient anterosuperiorly (2,3).

Joint Capsule
A fibrous capsule is attached medially to the glenoid neck outside the glenoid labrum and laterally to the anatomic neck of the humerus (except inferiorly, where it attaches to the humeral shaft). Superiorly, the capsule encroaches on the coracoid process to include the attachment
Figure 2. Normal anatomy. Ac = acromion, Ssc = subscapularis tendon, Ssp = supraspinatus tendon. (a) Drawing illustrates the deeper structures of the rotator interval (circled) after the superficial ligaments have been removed. Cap = joint capsule, CHL = coracohumeral ligament, Cl = clavicle, Cp = coracoid process, Lat. band = lateral band of the CHL, Med. band = medial band of the CHL, Tr = transverse humeral ligament. (b) In the corresponding cadaveric photograph, the conjoined tendon of the coracobrachialis muscle and the short head of the biceps brachialis muscle (arrow) is flipped superiorly, the supraspinatus tendon is flipped posteriorly, and the coracoacromial ligament is cut at its insertion at the acromion (arrowhead). The rotator interval (dotted outline) is covered by the coracohumeral ligament (CHL) and the extraarticular portion of the LBT (*).

Figure 3. (a) Drawing illustrates the normal anatomy of the biceps pulley. The CHL is cut so that the superior glenohumeral ligament (SGHL), focal capsular thickening, and the intraarticular portion of the LBT can be seen. Ac = acromion, Cl = clavicle, Cp = coracoid process, GT = greater tuberosity, LT = lesser tuberosity, Ssc = subscapularis tendon, Ssp = supraspinatus tendon. (b) In the corresponding cadaveric photograph (anterolateral view), the subscapularis tendon (arrows) and the joint capsule (arrowhead) are cut. Cp = coracoid process, dotted line indicates the rotator interval.

of the LBT (3,4). The capsule of the glenohumeral joint is lax and somewhat redundant, and although this configuration maximizes the range of motion of the upper extremity, it also contributes to potential instability of the glenohumeral joint. There are several ligaments and tendons that reinforce the capsule, from both the inside and outside (Figs 1–3).
Figure 4.  (a) Drawing illustrates the intraarticular portion of the shoulder without the humeral head. Ac = acromion, Cl = clavicle, Cp = coracoid process, 1 = supraspinatus tendon, 2 = infraspinatus tendon, 3 = teres minor tendon, 4 = subscapularis tendon, 5 = LBT, 6 = superior GHL, 7 = middle GHL, 8 = anterior band of the inferior GHL, 9 = posterior band of the inferior GHL, 10 = glenoid labrum, 11 = CHL, 12 = superior subscapularis recess, 13 = subacromial bursa, 14 = subcoracoid bursa, 15 = short head of the biceps tendon, 16 = coracobrachialis tendon, 17 = coracoacromial ligament, 18 = coracoclavicular ligament. (b) Corresponding cadaveric photograph shows the proximal portion of the LBT (black *), the superior GHL (black arrow), and the middle GHL (arrowhead). The superior subscapularis recess (white *) is seen between the capsule and the subscapularis muscle (white arrow). The joint capsule is cut at its proximal portion (cf Fig 7).

Capsular Ligaments
The three glenohumeral ligaments (GHLs)—superior, middle, and inferior—are bandlike collagenous thickenings of the capsule that reinforce the (thin) capsule and can be seen only from within the joint (Fig 4). The superior GHL is the most consistently identified capsular ligament (2). It originates from the superior glenoid tubercle just anterior to the origin of the biceps tendon. The CHL originates at the coracoid process and courses posteriorly and laterally to fuse with the joint capsule. It runs parallel to the superior GHL and inserts into the lesser tuberosity (2).

Long Head of the Biceps Tendon
As its name implies, the biceps brachii muscle has two separate origins. The short head of the biceps tendon is extraarticular in location and arises from the apex of the coracoid process along with the coracobrachialis tendon. The LBT arises within the glenohumeral joint from the supraglenoid tubercle of the scapula, and the glenoid labrum within the capsule (Figs 1, 5). It traverses the rotator interval and descends through the intertubercular sulcus (bicipital groove). The conjoined distal tendon of the short and long heads inserts onto the radial tuberosity below the level of the elbow (3). Because of the anatomic position of the bicipital groove both medially and ventrally in relation to the scapula, the biceps serves as a rather ineffectual elevator of the arm. The site of origin of the long head is variable. Attachment to the posterosuperior labrum, the glenoid tubercle, or both may be seen. The labral attachment is further subdivided, arising mainly posteriorly, mainly anteriorly, or equally from both locations (5).

Rotator Cuff
The four constituent muscles of the rotator cuff originate from the scapula and wrap around the humeral head as they insert onto the proximal humerus. These muscles include the subscapularis muscle anteriorly, the infraspinatus and teres minor muscles posteriorly, and the supraspinatus muscle superiorly. The musculotendinous cuff is firmly attached to the underlying joint capsule, except at the rotator interval and axillary recess, and reinforces the joint capsule from the outside (Fig 2) (6).
Figure 5. Biceps anchor. Cadaveric photograph (lateral view) shows the origin of the biceps tendon (white arrow) and the superior GHL (black arrow). The CHL (black arrowhead) is seen just above the biceps tendon. White arrowhead indicates the cut subscapularis tendon (cf Fig 3b).

Figure 6. Cadaveric photograph (posterolateral view) shows the intraarticular ligaments and outer supporting structures. The roof of the rotator interval is covered by the CHL (arrowheads) and the joint capsule (Cap). The proximal portion of the LBT (★) courses into the bicipital groove (not shown) together with the middle GHL (straight arrow) and the anterior band of the inferior GHL (curved arrow). Isp = infraspinatus tendon, Ssp = supraspinatus tendon, TM = teres minor tendon.

Coracoacromial Ligament
The coracoacromial ligament is a strong, triangular fibrous band that extends from the coracoid process to the acromion (Fig 1). Together with the coracoid process and the acromion, it forms a protective arch superficial to the rotator cuff (3). The shape of the ligament varies (6).

Rotator Interval and Biceps Pulley
The triangular space between the superior border of the subscapularis tendon and the anterior border of the supraspinatus tendon is termed the rotator interval. The intraarticular portion of the LBT courses through the rotator interval (Fig 2) (7–10). In this article, the anterior rotator interval is referred to simply as the rotator interval. The apex and the base of the rotator interval are formed by the transverse ligament bridging the bicipital groove and the coracoid process with the origin of the CHL medially, respectively (7–10). The rotator interval represents a defect in the rotator cuff resulting from the protrusion of the coracoid process between the supraspinatus and infraspinatus tendons. This portion of the glenohumeral joint capsule is not reinforced by overlying rotator cuff muscles.

The CHL originates from the lateral aspect of the base of the coracoid process. It courses through the rotator interval and forms two discrete bands distally. The larger (lateral) band blends into the greater tuberosity and the fibers of the supraspinatus tendon. The smaller (medial) band crosses over the biceps tendon to insert at the proximal aspect of the lesser tuberosity, forming an anterior covering around the biceps tendon, where it blends with the fibers of the subscapularis tendon. Variable types of insertion are described, including (a) insertion into the rotator interval, (b) insertion to either the supraspinatus tendon or the subscapularis tendon, or (c) insertion to both the supraspinatus and subscapularis tendons (2,11). The CHL is the most superficial capsular structure of the rotator interval. It blends with the fibers of the subscapularis and supraspinatus tendons at their insertions.

The superior GHL originates from the superior glenoid tubercle just anterior to the biceps tendon. Laterally, it forms a U-shaped “sling” that crosses underneath the biceps tendon and inserts into the lesser tuberosity, where it blends with the CHL. The insertion fibers of the superior GHL extend inferiorly to the superior margin of the subscapularis tendon and blend with the fibers of the subscapularis tendon at the lesser tuberosity (8).

The LBT may arise from the posterosuperior labrum, the superior glenoid tubercle, or both (Figs 3, 5). As the tendon passes laterally through the rotator interval, it is surrounded by the CHL superiorly and the superior GHL anteriorly, forming a slinglike band (Figs 6, 7) (5,12). When the
biceps tendon approaches the distal end of the capsule, the bicipital groove, the conjoined fibers of the superior GHL and joint capsule form the floor of the tendon (Fig 8) (5). The role of the transverse humeral ligament remains controversial. Many authors do not believe that it is a major contributor to stabilization of the biceps tendon in the bicipital groove (1,10,13). Gleason et al (14) conducted a cadaveric study and discovered no separate anatomic structures bridging the bicipital groove. Instead, the fibers covering the bicipital groove are composed of a sling formed mainly by the fibers of the subscapularis tendon, with contributions from the supraspinatus tendon and the CHL (14). Similarly, in a study of 85 cadaveric shoulders, MacDonald et al (15) found no distinct transverse humeral ligament. They stated that in only 8% of cases did the subscapularis tendon...

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**Figure 7.** Biceps pulley. C = subscapularis tendon, D = middle GHL. (a) Cadaveric photograph (superoposterior view) shows that the rotator cuff has been cut between the supraspinatus tendon (A) and infraspinatus tendon and has been lifted. Note how the inferior wall of the biceps pulley is formed by the subscapularis tendon, the anterior wall by the superior GHL (B), and the superior wall by the CHL. Dotted line indicates the rotator interval. E = infraspinatus tendon, F = posterior band of the inferior GHL. (b) Cadaveric photograph (superoposterior view) shows the distal insertion of the superior GHL to the humeral head (arrow).

**Figure 8.** Cadaveric photographs (superolateral view) show the floor of the biceps pulley. The distal insertion of the superior GHL to the humeral head (arrow) is seen from a different angle than in Figure 4c. The LBT (*) has been pulled slightly anterior to allow a better view. The distal insertion of the supraspinatus tendon (Ssp) is also depicted (arrowhead in b).
insert exclusively onto the lesser tuberosity. They suggested that what was once thought to be a transverse humeral ligament is actually combined fibers from the subscapularis tendon and posterior lamina of the tendon of the pectoralis major muscle (15). The medial band of the CHL and the superior GHL inserting on the lesser tuberosity, together with the superior fibers from the subscapularis tendon, act as a pulley to stabilize the biceps tendon in the bicipital groove. This entity is also referred to as the biceps reflective pulley or sling (7,9,10,12).

**MR Imaging Assessment**

The biceps pulley is composed of several small anatomic structures that lie very close to one another and actually blend together at their distal attachment sites. Consequently, these structures can be difficult to evaluate using conventional MR imaging. Direct MR arthrography has been shown to be the imaging modality of choice for identifying the normal anatomy and depicting lesions of the pulley mechanism. Chung et al (16) conducted a study of pre- and postarthrographic MR images obtained in 32 cadaveric shoulders to better define the anatomy and MR findings of this area (16). They found that the rotator interval and its capsular structures were better depicted using direct MR arthrography. In their study, only the extraarticular biceps tendon and some parts of its intraarticular portion were seen at routine MR imaging in all cases, whereas MR arthrography demonstrated the entire biceps tendon, including the intraarticular portion, in all cases. The authors also stated that the CHL was seen in only 60% of cases, and that in no case was the superior GHL well delineated with routine MR imaging (16). Both ligaments are identified at MR arthrography in all cases (Figs 9–11). Because of its variety of
Figure 12. Biceps pulley lesion with a rotator cuff tear in a 34-year-old man who suffered an acute injury from a fall. (a, b) Axial fat-saturated T1-weighted MR arthrographic images demonstrate absence of the superior GHL (arrow in a) and a swollen subscapularis tendon, along with contrast material insinuation indicating a partial tear of the subscapularis tendon (arrow in b). (c) Oblique sagittal fat-saturated T1-weighted MR arthrographic image also shows absence of the (torn) superior GHL (black arrow) and a frayed and irregular superior margin of the subscapularis tendon (white arrow). (d) Oblique coronal T2-weighted MR arthrographic image shows a tear of the supraspinatus tendon (arrow). Subacromial impingement and rupture of the supraspinatus tendon were also confirmed with arthroscopy. (e) Arthroscopic photograph shows a rotator interval lesion (arrow). Fraying of the subscapularis tendon (not shown) was also confirmed. $H =$ humeral head.
insertion sites, the CHL may be difficult to visualize as a separate structure at MR imaging, especially at the insertion site.

Optimal standard MR imaging should include images obtained in all three planes and aligned with the glenohumeral joint. Oblique coronal images are suitable for evaluation of the rotator cuff and labrum, but it is difficult to evaluate the rotator interval on these images. Oblique sagittal images obtained parallel to the plane of the glenoid fossa and orthogonal to the long axis of the rotator cuff are thought to be the best for evaluating the rotator interval and its contents (Fig 9) (7). Axial images are also valuable for identifying the biceps pulley complex, along with the biceps tendon within the proximal bicipital groove. The ligamentous pulley can be identified on cranial images upon close examination (Fig 10). High-resolution (<3 mm) sequences performed with a high image matrix are recommended to optimize evaluation of the individual structures of the biceps pulley complex (9). Some authors have reported three-dimensional fat-suppressed gradient-echo MR imaging to be useful (17).

At our institution, intraarticular gadolinium-based contrast material diluted with saline solution is injected under fluoroscopic guidance with an anterior approach before MR imaging is performed. Our standard shoulder imaging protocol includes axial, oblique coronal, and oblique sagittal fat-saturated T1-weighted imaging (repetition time msec/echo time msec = 660/11), axial and oblique coronal fast spin-echo T2-weighted imaging (3360/82), and axial gradient-echo T2*-weighted imaging (550/15). Other imaging parameters include a 15 × 15-cm field of view, 3-mm section thickness, 0.6-mm intersection gap, and 215 × 300 matrix. A 1.5-T MR imager and a dedicated shoulder surface coil are used, and patients are positioned supine with the shoulder in neutral position.

**Pathologic Processes of the Anterosuperior Shoulder**

**Rotator Interval Lesions**
The structures that make up the rotator interval, including the LBT, labral-biceps anchor, superior GHL, CHL, anterior margin of the supraspinatus tendon, superior margin of the subscapularis tendon, and joint capsule, are closely associated with each other. Therefore, disease affecting any of the rotator interval components may be part of a complex spectrum of pathologic conditions. Injury to any of these components requires that the entire rotator interval system be evaluated. Clinical manifestations of injury to this area may be nonspecific, and some of the lesions may be missed at arthroscopy (18). Persistent shoulder pain may result if these “hidden lesions” are not identified preoperatively and addressed at the time of surgery (19).

Isolated rotator interval lesions have rarely been described in the literature. Nobuhara and Ikeda (20) observed rotator interval defects with subsequent inflammatory changes causing inferior instability. They identified two types of lesions: type 1, consisting of inflammation of the superficial bursal area without instability; and type 2, consisting of extensive inflammation of deeper tissues in the rotator interval with anterior instability. An inflamed synovium, hypertrophy and elongation of the middle GHL, possible tear of the ligament, and granulation tissue over the biceps tendon as well as on the undersurface of the superior aspect of the subscapularis tendon were associated surgical findings in the cases they described (20). Some authors have described rotator interval tears in association with shoulder instability, with secondary impingement by the coracoacromial and coracoid processes due to anterior subluxation of the joint being the suggested cause (21). Le Huec et al (22) described a traumatic tear of the rotator interval in 10 young patients. An associated partial tear of the supraspinatus tendon was seen in only one patient. All lesions were seen in the upper part of the subscapularis tendon near the lesser tuberosity.

Because the biceps pulley lies in the rotator interval, there may be no substantial difference between reported rotator interval lesions and biceps pulley lesions.

**Biceps Pulley Lesions**
Instability of the LBT is closely related to biceps pulley lesions, with the specific pattern of instability depending on the injured supporting structures. A pulley lesion can be caused by degenerative changes, acute trauma, repetitive microtrauma, or injury associated with a rotator cuff tear (Figs 12, 13).
Baumann et al (18) performed a retrospective review of 1007 arthroscopies and found isolated pulley lesions in 7.1% of cases. The authors (as have other investigators) described different mechanisms of pathogenesis that led to the pulley lesions. Traumatic injuries resulting from a fall on an outstretched arm in combination with full external or internal rotation, a fall backward on the hand or elbow, or direct anterior impact may cause disruption of the pulley. The capsuloligamentous complex may detach from the lesser tuberosity, leading to anteromedial subluxation of the LBT and, over time, injury to the biceps tendon itself. A subscapularis tendon tear may also result from these injuries (7,18,19,22). Chronic and repetitive stress may also cause degenerative changes to the pulley. This pattern of injury is seen in patients who engage in overhead activity either occupationally or in association with sports. Large forces of acceleration and deceleration in the throwing motion are considered to cause damage. In this type of injury, the constituents of the superficial layer of the bicipital groove may be disturbed first by a transverse humeral ligament tear or distal subscapularis tendon tear. Biceps tendon laxity in the bicipital groove may lead to superior extension of degenerative changes in the rotator interval (7,18,20). Potential causes of pulley lesions include a congenital rotator interval defect (23), a supratubercular ridge as an osseous protrusion of the lesser tuberosity (24), or a shallow groove (5).

**Anterosuperior Impingement**
Gerber and Sebesta (25) first described anterosuperior impingement (ASI) as a form of intraarticular impingement responsible for painful structural diseases of the shoulder. They described impingement of the deep surface of the subscapularis tendon and the pulley against the anterosuperior glenoid rim in a position of horizontal adduction and internal rotation of the arm.

Figure 13. Bankart lesion with a superior GHL tear in a 35-year-old man who suffered recurrent shoulder dislocations from skiing. (a) Axial fat-saturated T1-weighted MR arthrographic image shows a classic Bankart lesion, with avulsion of the anteroinferior glenoid labrum (arrow). (b) Arthroscopic photograph shows avulsion of the anterior glenoid labrum (arrow). A superior GHL tear was also confirmed with arthroscopy. (c, d) Oblique sagittal (c) and axial (d) fat-saturated T1-weighted MR arthrographic images show the superior GHL tear (arrow).
Figure 14. Drawings (axial view) illustrate pulley lesions as defined by the Habermeyer classification system. (a) Normal anatomy. \(G\) = glenoid, \(H\) = humeral head, \(Ssc\) = subscapularis tendon, \(Ssp\) = supraspinatus tendon, \(1\) = CHL, \(2\) = LBT, \(3\) = superior GHL, \(4\) = lesser tuberosity, \(5\) = greater tuberosity, \(6\) = anterior glenoid labrum. (b) Isolated superior GHL lesion (group 1). (c) Superior GHL lesion with a partial articular-side supraspinatus tendon tear (group 2). The biceps tendon is slightly dislocated anteriorly. (d) Superior GHL lesion with a partial articular-side subscapularis tendon tear (group 3). The biceps tendon is dislocated into the torn subscapularis tendon. (e) Superior GHL lesion with partial articular-side supraspinatus and subscapularis tendon tears (group 4). The biceps tendon is dislocated completely outside the biceps pulley and is located in the torn subscapularis tendon. (Fig 14 adapted from reference 12.)

Figure 15. (a–c) Habermeyer group 1 lesion in a 53-year-old man who suffered recurrent shoulder dislocations from surfing. Axial (a) and oblique sagittal (b) fat-saturated T1-weighted MR arthrographic images show an isolated superior GHL tear (arrow). The superior GHL is indistinct (b) compared with a normal superior GHL (cf d). (Fig 15a and 15b reprinted, with permission, from reference 26.) (c) Arthroscopic photograph demonstrates fraying of the superior GHL at its origin (arrow). \(H\) = humeral head. (d) Oblique sagittal fat-saturated T1-weighted MR arthrographic image obtained in a healthy 15-year-old girl shows the superior GHL (arrowhead) with a normal smooth low-signal-intensity appearance. (Reprinted, with permission, from reference 26.)

Habermeyer et al (12) studied the factors influencing the development of ASI and subdivided pulley lesions into four different patterns (Fig 14). Group 1 lesions were defined as isolated superior GHL lesions (Fig 15); group 2, as superior GHL lesions with a partial articular-side supraspinatus tendon tear (Fig 16); group 3, as
significantly more often in patients with ASI than in those without ASI, but its association with the development of ASI remains uncertain (12,18). None of the patients described in these reports had a bursal-side rotator cuff tear.

superior GHL lesions with a partial articular-side subscapularis tendon tear (Figs 17, 18); and group 4, as superior GHL lesions with partial articular-side tears of both the supraspinatus and subscapularis tendons (Fig 19). ASI was seen more often in patients with an additional articular-side tear of the supraspinatus tendon (group 4) or an additional partial articular-side tear of the subscapularis tendon (group 3) (12). Acromioclavicular arthritis was also observed.
**Figure 18.** Habermeyer group 3 lesion in a 42-year-old man who sustained a weightlifting injury. (a) Axial fat-saturated T1-weighted MR arthrographic image demonstrates an arthroscopically confirmed partial articular-side subscapularis tendon tear (arrow). (b, c) Oblique sagittal fat-saturated T1-weighted MR arthrographic image (b) and corresponding arthroscopic image (c) show a superior GHL tear (arrow). $H =$ humeral head. (Fig 18b reprinted, with permission, from reference 26.)

**Figure 19.** Habermeyer group 4 lesion in a 56-year-old man. (a, b) Axial fat-saturated T1-weighted MR arthrographic images demonstrate an extensive subscapularis tendon tear with medial dislocation of the LBT (arrow in a) and a superior GHL tear (arrowhead in b). (Fig 19b reprinted, with permission, from reference 26.) (c, d) Oblique coronal fat-saturated T1-weighted MR arthrographic image (c) and corresponding arthroscopic image (d) show a tear (arrow in c) of the undersurface of the supraspinatus tendon ($Ssp$). $H =$ humeral head.
The biceps tendon lies within a reflection of the synovial membrane as it courses down the bicipital groove; thus, it is intraarticular but extrasynovial (5, 13). Given the communication of the tendon sheath with the glenohumeral joint and its anatomic association with the rotator cuff and rotator interval, any pathologic process involving one of these components may also affect other components.

Pathologic Conditions of the Biceps Tendon

The biceps tendon lies within a reflection of the synovial membrane as it courses down the bicipital groove; thus, it is intraarticular but extrasynovial (5, 13). Given the communication of the tendon sheath with the glenohumeral joint and its anatomic association with the rotator cuff and rotator interval, any pathologic process involving one of these components may also affect other components.

In contradistinction to the function of the biceps tendon at the elbow, its role at the shoulder is still controversial, and its previously accepted role as a stabilizer of the humeral head has been reconsidered (10, 13, 29). It is important to note that pathologic conditions of the biceps tendon have been widely accepted as a source of anterior shoulder pain. These pathologic conditions include instability, tendinopathy, tendinosis, and partial or complete biceps tendon rupture (10, 29). Several pathologic processes may coexist, some of which are a consequence of others.

Subluxation and Dislocation.—The association of dislocation of the biceps tendon with rotator cuff tears is generally accepted. However, isolated dislocations of the biceps tendon without rotator cuff injuries (although rare) have also been reported.

In their retrospective review of 445 surgical cases, Walch et al (19) defined biceps tendon dislocation as the total and permanent loss of contact between the tendon and the bicipital groove. They described four types of dislocation: (a) dislocation inside the subscapularis tendon, leaving the anterior fascia intact; (b) intraarticular dislocation with complete tear of all the insertions on the lesser tuberosity; (c) intraarticular dislocation with complete tear of all the insertions on the lesser tuberosity but intact anterior fascia, so that the lesion is hidden in the joint space; (d) subscapularis tendon (rupture of the supraspinatus tendon and CHL) (19). All dislocations are associated with tears of the ligamentous pulley. The authors found subluxation more difficult to define because partial or transitional loss of contact between the biceps tendon and the bicipital groove was not easy to define during surgery. The best arthroscopic sign of subluxation is fraying of the deep layer of the biceps tendon. The authors defined subluxation at arthroscopic computed tomography as occurring when the biceps tendon appeared fixed over the medial rim at the superior part of the groove or recentered into the groove before disappearance of the groove. In the cases of subluxation in their study, the ligamentous pulley was noted to be intact, attenuated, or torn. Tear of the supraspinatus tendon was almost always observed, and a lesion of the subscapularis tendon was always associated with subluxation (19). In a retrospective review of arthroscopic reports, Bennett (1) classified subluxation into four types according to the direction of the subluxation: (a) intraarticular,
between the subscapularis tendon and CHL, (c) external to the CHL, and (d) intrasheath. The author stated that the pattern is dependent on which underlying supporting structures are injured. Castagna et al (30) reported nine cases in which pulley system lesions were unclear but the diagnosis of an unstable biceps tendon was associated with a “chondral print,” seen at arthroscopy as a line on the anterior part of the humeral head representing a subluxated biceps tendon. In chronic subluxation, minimal dynamic medial displacement of the biceps tendon was difficult to detect with arthroscopy because of the minimal abnormality in the pulley system. The authors found the chondral print on the humeral head helpful in making the correct diagnosis when an associated pulley lesion was not clearly depicted (30).

At MR imaging, the dislocated biceps tendon can be identified medial to the empty bicipital groove, most clearly on axial images. Oblique coronal and oblique sagittal images are also useful. As mentioned earlier, the displaced biceps tendon can be identified as either extraarticular or intraarticular, with a variable degree of injury to the surrounding structures. At conventional MR imaging, associated abnormalities of the biceps tendon may manifest as variable degrees of increased signal intensity, changes in the shape of the tendon (thickening, flattening, broadening), and fluid around the displaced biceps tendon. Other abnormalities associated with dislocation of the biceps tendon include an abnormal shape of the bicipital groove, abnormalities of the rotator cuff, disruption of the CHL, disruption and thinning of the subscapularis tendon, and supraspinatus tendon tear (31,32).

**Tendinopathy or Tendinosis.**—Secondary biceps tendinopathy is seen in 95% of patients with bicipital tendinopathy and is usually associated with disease of the rotator cuff and impingement syndrome (13,33). Primary biceps tendinopathy is diagnosed when other associated pathologic processes are excluded. Structural anomalies of the bicipital groove and repeated trauma are described as causes in young patients, with degenerative changes being implicated in older patients (34).

On MR images, tendon diameter changes, abnormal signal intensity, and associated fluid collections should be assessed (Fig 20). Thorough observation of the surrounding structures is also essential for making the correct diagnosis. In their study of cadaveric shoulders, Buck et al (35) suggested diameter change as a primary criterion for the diagnosis of tendon degeneration, although absence of a diameter change does not mean that there is no abnormality. A change in signal intensity in the biceps tendon likely indicates degeneration but is not sufficient to help differentiate between the various types of degeneration (eg, mucoid degeneration, lipoid degeneration). Fluid along the LBT sheath is not...
Figures 21, 22. (21) Type II SLAP lesion in a 23-year-old male baseball player. (a) Oblique coronal fat-saturated T1-weighted MR arthrographic image shows a superior labral tear (arrow) and a partial tear of the undersurface of the supraspinatus tendon (arrowhead). (b) Axial fat-saturated T1-weighted MR arthrographic image shows an attenuated and irregular superior GHL (arrow). (c) Arthroscopic photograph shows synovitis in the rotator interval. H = humeral head. (22) Type III SLAP lesion in a 19-year-old male baseball player. (a) Oblique coronal fat-saturated T1-weighted MR arthrographic image shows a superior labral tear (arrow). (b) Corresponding arthroscopic photograph shows an avulsed superoanterior glenoid labrum (arrow). H = humeral head. (c) Axial fat-saturated T1-weighted MR arthrographic image shows an associated biceps pulley lesion. The superior GHL is irregular (arrowhead).
Figure 23. Adhesive capsulitis in a 45-year-old man. Only 8 mL of contrast material was injected secondary to pain and elevated intraarticular pressure. Oblique sagittal T1-weighted MR arthographic image shows the subcoracoid fat triangle (arrowheads), which is partially obliterated (black arrow). The borders of the triangle are defined anterosuperiorly by the coracoid process, superiorly by the CHL (white arrow), and posteroinferiorly by the joint capsule.

necessarily abnormal, since the tendon sheath and glenohumeral joint are in direct communication. However, fluid completely surrounding the tendon may raise suspicion for tenosynovitis, although anterior circumflex humeral vessels may mimic fluid in the sheath (36).

Superior Labrum
Anterior and Posterior Lesions
With injuries to the rotator interval and LBT, superior labrum anterior and posterior (SLAP) lesions should be suspected. The converse is also true: The presence of a SLAP lesion suggests injuries to the rotator interval and LBT. SLAP lesions were initially reported as clinically significant because the superior labrum serves as an anchor for the biceps tendon. Thus, it is understandable that the cause of SLAP lesions is similar to those of isolated pulley lesions.

Four types of SLAP lesions were originally described by Snyder et al (37) on the basis of arthroscopic findings. A type I lesion is characterized by marked fraying of the superior glenoid labrum without labral detachment or biceps tendon injury. A type II lesion consists of labral fraying with stripping of the superior labrum and the attachment of the biceps tendon from the underlying glenoid fossa. A type III lesion is a “bucket-handle” tear of the superior labrum; the peripheral portion of the labrum remains firmly attached to the underlying glenoid fossa and the biceps tendon remains intact. A type IV lesion is a bucket-handle tear of the superior labrum that extends into the biceps tendon. Six additional classifications have been developed, mainly representing a combination of superior labral tears with extension into different areas of the labrum or other adjacent capsuloligamentous components. A type X lesion consists of a superior labral tear with extension to the rotator interval; some of the other types involve the biceps anchor or biceps tendon. It is unclear whether these ten types of SLAP lesions can be differentiated with MR imaging, and the mechanisms of injury have not yet been fully elucidated (38). The original classification scheme developed by Snyder et al (37) is still the most widely accepted.

It is important to evaluate the contour and signal intensity of the superior labrum on oblique coronal and axial MR images; the superior labrum should be smooth with uniform low signal intensity. Normal variants can be seen in an area where disease is also common. Variations in signal intensity, morphologic features, attachment, and presence or absence of the labrum may be seen. For example, transitional zones of the labrum may have high signal intensity, a Buford complex can be confused with pathologic detachment, or a sublabral sulcus-recess may be difficult to differentiate from SLAP lesions (39). Normal variants and pathologic lesions may coexist. Increased awareness of these variants may lead to correct interpretation of the images. A complete review of SLAP lesions and labral variants is beyond the scope of this article, but the reader should be familiar with them, especially since SLAP tears may be associated with lesions of the biceps pulley and rotator interval (Figs 21, 22).

Adhesive Capsulitis
Idiopathic adhesive capsulitis (Fig 23) is a self-limiting disease that is characterized clinically by the gradual onset of severe shoulder pain with restricted shoulder motion of unknown cause. Many terms have been applied to this specific clinical entity, including frozen shoulder, adhesive capsulitis, stiff and painful shoulder, periarthritis, periarticular adhesions, and pericapsulitis. There is no single agreed-upon criterion for establishing the diagnosis of this condition; instead, the diagnosis is made on the basis of clinical history and physical examination findings. Synovial inflammation with
surgical treatment. Adhesive capsulitis may be associated with (a) diabetes mellitus; (b) conditions such as Dupuytren disease; (c) hyper- or hypothyroidism; (d) cerebral, cardiac, and respiratory conditions; and (e) surgical procedures that do not directly affect the shoulder, such as cardiac surgery (42,43). Any other pathologic conditions of the shoulder that could cause secondary capsular adhesions and contracture should be excluded. Such conditions include rotator cuff disease, calcific deposits, biceps tendinopathy or tear, rheumatoid arthritis, hemiplegia, postsurgical scarring, and posttraumatic stiffness with or without a fracture (40,42).

Conventional shoulder arthrography has long been considered the standard imaging study for establishing the diagnosis of adhesive capsulitis. Decreased joint volume of less than 10 mL, pain after the injection of less than 10 mL of contrast material, and marked loss of the normal axillary fold are seen in patients with adhesive capsulitis (40,44).

Recent studies have shown the usefulness of other imaging modalities such as conventional MR imaging, ultrasonography, and nuclear medicine imaging (45,46). There have been reports establishing the criteria for determining the diagnosis of adhesive capsulitis at conventional MR imaging (47) as well as at direct and indirect MR arthrography (43,44,48). Thickening of the joint capsule in the axillary recess has been described as a characteristic sign of adhesive capsulitis (48), but not all investigators agree that it is an accurate sign (43,47). There have been many arthroscopic studies showing inflammation in the rotator interval, synovitis at the anterosuperior glenohumeral joint, and thickening of the CHL as more definitive for the diagnosis (41). Mengiardi et al (43) described (a) thickening of the CHL and the capsule at the rotator interval and (b) complete obliteration of the fat triangle under the coracoid process as the most characteristic MR imaging findings in adhesive capsulitis. Some researchers have found that the coefficient of enhancement (ie, the rate of increase in signal intensity on dynamic contrast material–enhanced MR images) correlates with the level of inflammatory activity of the synovium (49).

Making a diagnosis of adhesive capsulitis is difficult with MR imaging performed to evaluate a painful shoulder, but recognition of this disorder may have an important effect on medical and surgical treatment.

Subcoracoid Bursa

The capsule of the glenohumeral joint usually has at least two openings: (a) below the coracoid process, connecting the joint to a bursa behind the subscapularis tendon (anterior); and (b) between the humeral tubercles, transmitting the long tendon of the biceps muscle and its synovial sheath, connecting the joint to a bursa under the infraspinatus tendon (posterior and inconstant) (Fig 4) (3).

The recess that projects anteriorly between the superior and middle GHLs is called the superior subscapularis recess or subscapularis bursa. This recess lies between the subscapularis muscle and the anterior surface of the scapula and extends above the superior margin of the subscapularis tendon. Because this is not a separate bursa, fluid within this recess may be simply physiologic. On the other hand, fluid in the subcoracoid bursa may represent a pathologic process. The subcoracoid bursa lies between the anterior surface of the subscapularis muscle and the coracoid process. It extends along the tendon formed by the merger of the coracobrachialis tendon and the short head of the biceps tendon. This bursa normally communicates, not with the glenohumeral joint, but with the subacromial-subdeltoid bursa (50,51).

Grainger et al (50) retrospectively reviewed imaging reports from 1831 shoulder MR imaging examinations and identified 16 patients who were reported to have subcoracoid bursa effusions. Rereview of the MR images revealed that 13 of these patients actually had fluid collections in the subcoracoid bursa. A rotator cuff tear was seen in all cases, and a rotator interval tear was seen in 11 patients. Although the superior subscapularis recess and the subcoracoid bursa may be difficult to visualize on axial images, the authors found oblique sagittal images to be useful in differentiating between the two (Fig 24) (50).

The clinical significance of fluid in the subcoracoid bursa is uncertain. However, there have been some reports on the association of such fluid with tears of the rotator cuff and rotator interval (50,51). The effusion may represent isolated subcoracoid bursitis, inadvertent injection of contrast material into the bursa (which, if not recognized, could result in an erroneous diagnosis because of the communication between the bursa and the joint with a complete rotator cuff tear), or a posttraumatic inflammatory response. When an effusion is identified, the rotator cuff and rotator interval should be carefully evaluated for possible tears.
Figure 24. Subcoracoid bursa. Oblique sagittal T2-weighted MR arthrographic image shows a superior subscapularis recess (arrow) and subcoracoid bursa (s) located anterior to the subscapularis muscle (S). Note the caudal extent of the subcoracoid bursa and the fibrous septum (arrowhead) separating the two fluid-filled spaces. T = coracobrachialis tendon and the short head of the biceps tendon.

Conclusions

The biceps pulley consists of the CHL, the superior GHL, and fibers from the subscapularis tendon. These constituents are distinct anatomic structures, yet their fibers merge to form a functional unit that stabilizes the LBT and the glenohumeral joint. Not only the biceps pulley, but also the surrounding musculotendinous structures (LBT, glenoid labrum, biceps anchor, rotator cuff) should be considered as constituting a functional and anatomic unit. MR arthrography effectively demonstrates the normal anatomy and pathologic conditions of this area, as well as associated findings. Oblique sagittal and axial images should be obtained for the evaluation of pulley lesions.

Injury to the anterosuperior aspect of the shoulder is better understood as part of a spectrum of processes than as an isolated lesion. When an abnormality of one of the system components is suspected, thorough radiologic assessment of the entire system is especially important. Familiarity with the cause, classification, and direction of the injury may be useful for identifying and characterizing such abnormalities.

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References

Biceps Pulley: Normal Anatomy and Associated Lesions at MR Arthrography

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The medial band of the CHL and the superior GHL inserting on the lesser tuberosity, together with the superior fibers from the subscapularis tendon, act as a pulley to stabilize the biceps tendon in the bicipital groove. This entity is also referred to as the biceps reflective pulley or sling (7,9,10,12).

Page 799 (Figure on page 797)
Oblique sagittal images obtained parallel to the plane of the glenoid fossa and orthogonal to the long axis of the rotator cuff are thought to be the best for evaluating the rotator interval and its contents (Fig 9) (7). Axial images are also valuable for identifying the biceps pulley complex, along with the biceps tendon within the proximal bicipital groove.

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Disease affecting any of the rotator interval components may be part of a complex spectrum of pathologic conditions. Injury to any of these components requires that the entire rotator interval system be evaluated.

Page 799 (Figure 12 on page 798, figure 13 on page 800)
A pulley lesion can be caused by degenerative changes, acute trauma, repetitive microtrauma, or injury associated with a rotator cuff tear (Figs 12, 13).

Page 804
A pulley lesion leads to instability of the LBT, which causes partial articular-side tears of the subscapularis and supraspinatus tendons (12,18). The medially dislocated biceps tendon further reinforces anterior and upward translation of the humeral head, thus resulting in ASI.