Level V Evidence

Anatomy, Function, Injuries, and Treatment of the Long Head of the Biceps Brachii Tendon

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Abstract: Lesions of the long head biceps tendon (LHB) are frequent causes of shoulder pain and disability. Biceps tenotomy and tenodesis have gained widespread acceptance as effective procedures to manage both isolated LHB pathology and combined lesions of the rotator cuff and biceps-labral complex. The function of the LHB tendon and its role in glenohumeral kinematics presently remain only partially understood because of the difficulty of cadaveric and in vivo biomechanical studies. The purpose of this article is to offer an up-to-date review of the anatomy and biomechanical properties of the LHB and to provide an evidence-based approach to current treatment strategies for LHB disorders.

The long head of the biceps (LHB) tendon has long been considered a troublesome pain generator in the shoulder. Biceps tenotomy and tenodesis have gained widespread acceptance as quick, easy, and cost-effective procedures to manage both isolated LHB pathology and combined lesions of the rotator cuff and biceps-labral complex. Although these procedures have documented success in most outcomes studies, controversy persists as to the possible functional contributions of the LHB to shoulder stability and motion. This article offers an up-to-date review of the anatomy and biomechanical properties of the LHB and current strategies for successful treatment of LHB pathology (Tables 1-3).

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ANATOMY

The LHB originates from the supraglenoid tubercle of the scapula with an intra-articular portion that passes over the humeral head before exiting the glenohumeral joint through the bicipital groove.[1,2] The tendon is approximately 5 to 6 mm in diameter and approximately 9 cm in length. The size of the tendon varies, and the intra-articular portion is typically wide and flat whereas the extra-articular portion is both rounder and smaller.[3] The anterior circumflex humeral artery provides blood supply to the articular portion of the LHB. The more distal portion of the LHB is fibrocartilaginous and avascular to accommodate its sliding motion within its sheath in the groove, whereas the proximal tendon is more richly vascularized.

Alpantaki et al.[4] found a "net-like pattern" of rich sensory and sympathetic innervations of the LHB, which was concentrated at the biceps tendon anchor and became attenuated at the distal musculotendinous junction.

A soft-tissue sling stabilizes the extra-articular LHB as it enters the bicipital groove (Figs 1 and 2). This

TABLE 1. *Overall Key Points*

Cadaveric biomechanical studies suggest that the LHB has stabilizing effects on the glenohumeral joint in all directions.
In vivo studies have yet to establish this stabilizing effect.
EMG studies have further questioned the role of the LHB in shoulder kinematics, showing little or no activation when the elbow is immobilized.
Further in vivo investigations using advanced imaging for increased precision are needed to better define the role of the LHB in glenohumeral kinematics.

biceps reflection pulley (BRP) is built by fibers of the coracohumeral ligament, superior glenohumeral ligament (SGHL), and parts of the subscapularis tendon, as shown by anatomic dissection and histology.[5] The LHB is subject to mechanical stresses in the groove, at the pulley, and by pathology of the rotator cuff and subacromial space. Braun et al.[6] have shown in a biomechanical study that the tendon slides up to 18 mm in and out of the glenohumeral joint in forward flexion and internal rotation compared with a reference of neutral arm position and neutral rotation. Habermeyer et al.[7] described a 30° to 40° turn of the biceps tendon as it exits the joint and stabilization of the tendon by a pulley sling.

The depth and morphology of the bicipital groove may also play a role in function, stability, and not least of all, pathology of the LHB. Pfahler et al.[8] described the bicipital groove anatomy in a radiographic study. The medial wall of the bicipital groove was higher, with an opening angle from 30° to 40° in most patients without LHB pathology. The total opening angle between the lateral and medial wall was found to be 101° to 120° in most asymptomatic shoulders.

FUNCTION OF LHB TENDON

A majority of biomechanical studies investigating the role of the LHB have focused on its contributions to glenohumeral stability, restraining abnormal translations. With few exceptions,[9,10] these studies have relied on cadaveric models to examine this interaction.

TABLE 2. *Key Points of LHB Pathologies*

Instability of the LHB varies from subluxation to dislocation and is usually associated with rotator cuff tears, especially subscapularis tendon tears.
High shear forces to the biceps reflection pulley occur in forward flexion and internal rotation and may result in "hidden" pulley lesions.
Proximal biceps tenodesis may result in residual pain in the bicipital groove.
Coracoid impingement is an often overlooked cause of anterior shoulder pain.

TABLE 3. *Key Points of Current Treatment Strategies*

Although LHB tenotomy has shown excellent results for pain relief, tenodesis has been shown to better restore supination strength and endurance, in addition to maintaining normal biceps contour.
In patients with coracoid impingement and limited CHI, a coracoidplasty may be considered.
Subpectoral biceps tenodesis yields excellent outcomes with a low complication rate.
There is evidence to suggest that functional outcomes and return to sport may be improved with biceps tenodesis compared with SLAP repair, but further studies are needed.

Cadaveric Biomechanical Studies

Pagnani et al.[11] tested the effect of simulated contraction of the LHB (55 N) in 10 cadaveric shoulders and showed significantly decreased humeral head translations anteriorly, superiorly, and inferiorly when load was applied to the biceps, especially in lower angles of elevation.

Itoi et al.[12] concluded from their biomechanical studies that both the LHB and the short head of the biceps brachii are anterior stabilizers to the glenohumeral joint in abduction and external rotation when loaded with 1.5 kg and 3 kg. According to their work,

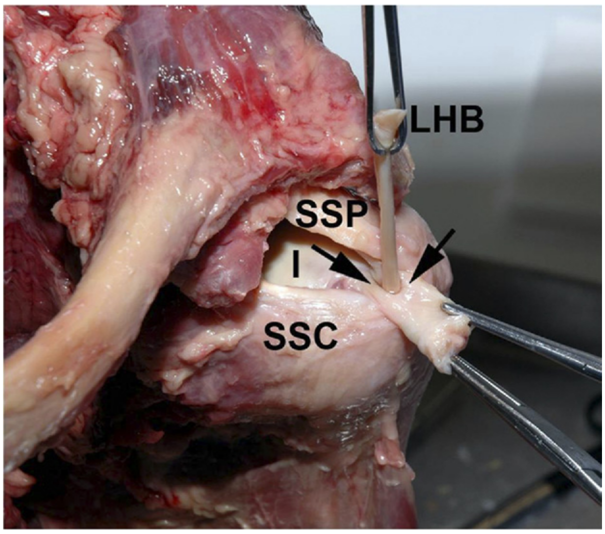


FIGURE 1. Cadaver (left shoulder) showing pulley sling (arrows). The rotator interval (I) has been dissected from the supraspinatus (SSP) and subscapularis (SSC) tendons. The LHB has been dissected from its origin at the glenoid.

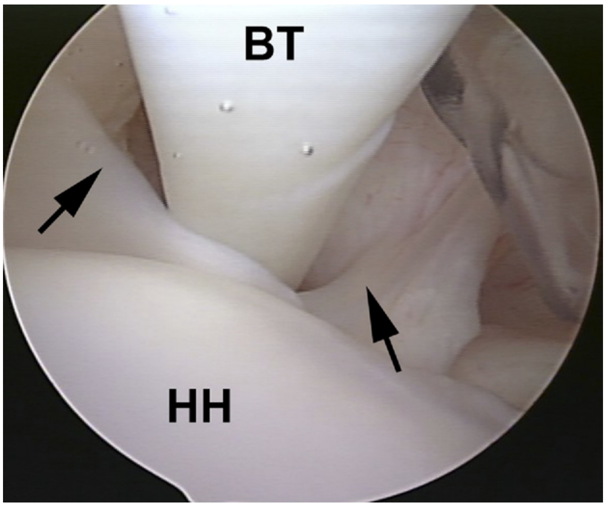


FIGURE 2. Arthroscopic view of a left shoulder from posterior showing intact biceps pulley (arrows). (BT, biceps tendon; HH, humeral head.)

the role of both tendons increases in a setting of instability.

Rodosky et al.[13] performed a study using a dynamic cadaveric shoulder model that simulated the forces of the rotator cuff and LHB muscles. Their data suggest that the long head of the biceps muscle contributes to anterior stability of the glenohumeral joint by increasing the shoulder's resistance to torsional forces in the vulnerable abducted and externally rotated position. Furthermore, the authors found significantly less torsional rigidity and significantly increased strain to the inferior glenohumeral ligament in a setting of detachment of the biceps-labral complex at the superior glenoid.

Payne et al.[14] applied a 40-N load to the biceps tendon and found a significant decrease in anterolateral contact pressure in 6 shoulders whereas the contact pressures in 3 shoulders with type III acromions were unchanged. Kumar et al.[15] showed that tensioning of the short head of the biceps alone caused significant upward migration of the humeral head whereas tensioning of the LHB alone or of both heads did not cause any difference in a setting of simulated powerful elbow flexion and supination. When the LHB was cut, there was also a significant upward migration noted. The authors concluded that the LHB therefore plays a stabilizing role in the glenohumeral joint in powerful elbow flexion and supination.

Youm et al.[16] showed in a recently published biomechanical study that the loaded LHB (22 N) significantly affects glenohumeral translation (anterior, posterior, superior, and inferior), kinematics, and rotational range of motion in a simulated position of 90° of arm abduction and different angles of internal and external rotation.

Su et al.[17] applied 55 N of load to the LHB in differently sized rotator cuff tears. They found significantly decreased anterosuperior and superior glenohumeral translation when loading the LHB for all sizes of rotator cuff tears.

The conclusion is that biomechanical studies indicate that the LHB contributes to stability of the glenohumeral joint in all directions. However, considerable variability exists with regard to the load applied to the tendon (11 to 55 N). The maximum load of 55 N has been predicted by multiplying the physiologic cross-sectional area of the LHB by an accepted conversion factor.[18] Closer examination of this report shows that the mean potential moment generated by LHB was 16.8 Ncm^{-1} and the mean moment arm was 2.4 cm. This produces a load of 40.32 N and not 55 N. Some authors found this load to be extremely high[16] and therefore incorporated electromyographic (EMG) data showing the percentage of maximum voluntary contractions for the positions or motions tested to calculate the proper biceps load. Interestingly, Youm et al.[16] calculated a biceps load of 11 N for their model but did not find significant changes with that load. The conclusion is that we do not know to date how much load is physiologic for the LHB tendon. However, the amount of load is critical for all biomechanical studies. It is therefore possible that some studies that applied higher loads showed significant changes because of nonphysiologic high loads.

EMG Studies

There are many studies in the literature that document activity of the biceps brachii during shoulder motion.[19-24] The important question for all EMG studies is how the recorded biceps activity affects glenohumeral joint kinematics.

There are 2 EMG studies that examined the effects of the biceps on the shoulder. In both studies the elbow joint was immobilized with a brace to minimize elbow-related biceps activity. Interestingly, the findings of these studies appear to be contradictory. Sakurai et al.[22] found that LHB activity stabilized the humeral head, whereas Levy et al.[21] found that the LHB either had a passive role or served as a functional stabilizer only when tensioned in association with elbow and forearm activity.

Jobe et al.[25] evaluated pitching biomechanics and showed that the biceps predominantly activates during cocking to accomplish elbow flexion and then reactivates during follow-through to decelerate the forearm. In a recent study Rojas et al.[26] found that biceps activity was higher during windmill pitch than during overhead throw, especially before and after ball release between 9 o'clock and the follow-through phase. In this position it is likely that most of the biceps activity is attributed to elbow and not shoulder function.

These are important findings for interpretation not only of EMG but also of biomechanical cadaveric studies that assume that there is bicipital activity associated with shoulder activities.

The conclusion from EMG data on LHB function remains controversial. On the basis of the current literature, it is not clear whether the biceps activity during shoulder movements is partly, mainly, or completely from the activation at the elbow joint. However, these data, as stated previously, are critical for all biomechanical studies, because they form the foundation for the amount to which the LHB should be loaded.

In Vivo Biomechanical Studies

Warner and McMahon[10] performed a radiographic study on 7 patients with loss of the proximal attachment of the LHB compared with the healthy contralateral control. In this study true anteroposterior radiographs were made in 0°, 45°, 90°, and 120° of abduction in the scapular plane. The authors found a significant superior translation of the humeral head at all degrees of abduction in patients with rupture of the LHB.

Intraoperative electrical stimulation of the biceps muscle during arthroscopy in 5 patients showed a compression of the glenohumeral joint.[9] Kido et al.[27] documented higher humeral head positions in patients with rotator cuff tears without contraction of the biceps. In this study the humeral head depressed significantly at different angles of abduction when the biceps muscle was activated in a radiographic model. The authors concluded that the LHB has an active depressor function of the humeral head.

The conclusion from in vivo studies is that because of a lack of applicable methods, there is almost no evidence about the function of the LHB in vivo. Both the studies of Warner and McMahon[10] and Kido et al.[27] are based on a radiographic model with true anteroposterior radiographs. There are concerns about the accuracy of these models for several reasons. First, in different angles of scaption, the scapula moves around the thorax and the orientation of the glenoid changes with that movement. It is therefore difficult to generate true anteroposterior radiographs with any consistency. Second, these methods fail to capture 3-dimensional movements. Finally, the accuracy of such measurements remains quite limited.

Although the existing body of biomechanical work suggests that the loaded LHB may restrain the shoulder from abnormal translations, many researchers have admitted the limitations of cadaveric testing in re-creating the dynamic interplay of anatomy that occurs in vivo. Further in vivo investigations must be undertaken with advanced imaging technology to elucidate the biomechanical role of the LHB.

We have recently performed a biplane fluoroscopy in vivo study on LHB function in 5 patients (10 shoulders) to assess LHB function during various arm movements. In contrast to the findings in previous studies,[10,27] we did not find an increase in superior migration for shoulders when the LHB was absent (subjects with isolated subpectoral tenodesis) when compared with their healthy contralateral controls. We did find that the shoulders that had undergone tenodesis tended to be more anteriorly positioned (P = .003), but the difference was only 0.7 mm, which does not appear to be clinically significant (unpublished data, J.E.G., November 2010).

LHB PATHOLOGIES

The LHB can be a source of shoulder pain or diminished function for various reasons. LHB pathologies include tendinitis, rupture, subluxation or instability, pulley lesions, and SLAP lesions.

Tendon Rupture

The most common sites of tendon rupture are at the tendon's origin and at the exit of the bicipital groove near the musculotendinous junction.[28] When ruptures of the long head occur, the muscle mass moves distally, often resulting in a characteristic Popeye deformity. Ruptures of the long head are most common in patients aged over 50 years, and they occur more frequently than ruptures of the short head or the distal tendon, accounting for 96% of all biceps brachii injuries.[1] Often, they are associated with biceps tendinitis,[29] which may lead to degeneration of the biceps tendon and a resulting rupture with little or no trauma.[28]

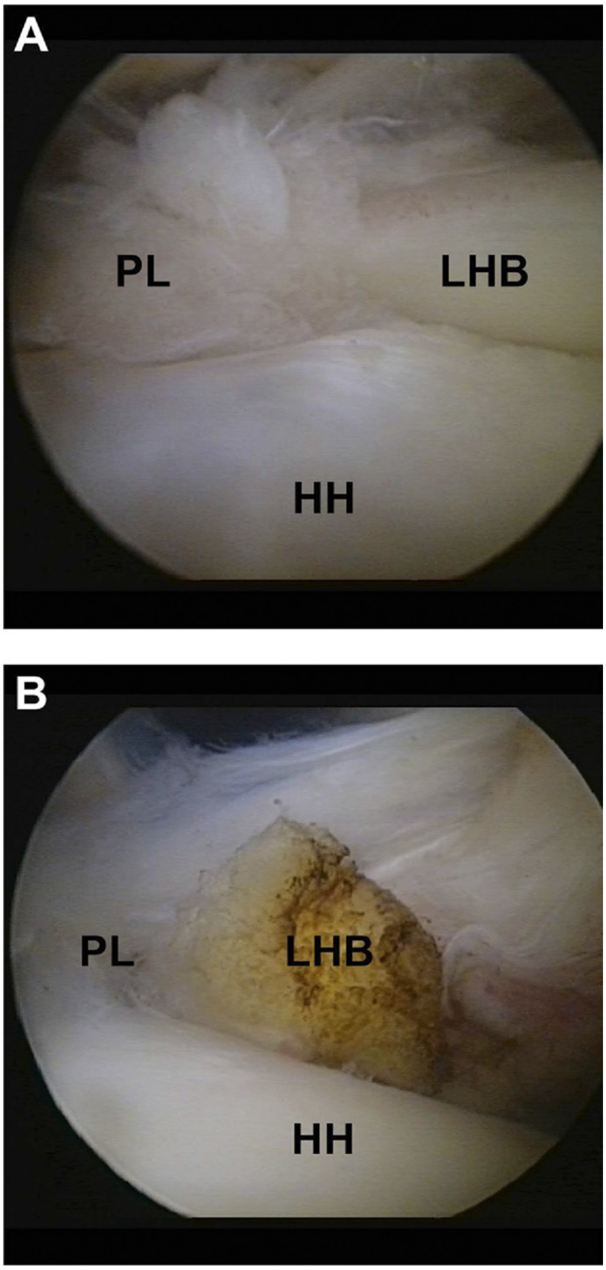


FIGURE 3. Arthroscopic views of a right shoulder from posterior. (A) Pulley lesion with dislocation of LHB into subscapularis tendon. (B) Damage to subscapularis tendon after tenotomy of LHB. (HH, humeral head.)

Biceps Instability

LHB instability and BRP tears,[30-32] so-called pulley or biceps reflection pulley lesions (Fig 3), are well described. Instability of the LHB varies from subluxation to dislocation and is usually associated with rotator cuff tears, especially subscapularis tendon tears.[33] Different classification systems for biceps instability have been described.[7,30] Habermeyer et al.[7] defined 4 different arthroscopically observed types, with isolated lesions of the SGHL (type I), SGHL lesion and a partial articular-sided supraspinatus tendon tear (type II), SGHL lesion and a deep surface tear of the subscapularis tendon (type III), and lesion of the SGHL combined with a partial articular-sided supraspinatus and subscapularis tendon tear (type IV).

We have performed a prospective study to look at the incidence of injury to the BRP in a group of 229 consecutive patients undergoing shoulder arthroscopy. The incidence of BRP or pulley lesions was 32.4%. As stated initially, there is a significant correlation between pulley lesions and SLAP tears ($P = .003$), rotator cuff pathology ($P = .001$), and LHB pathologies ($P < .05$).[34]

There is speculation that loading the tendon in external rotation–abduction positions of the arm is the pathomechanism of these pulley lesions.[7,32] Our findings in a biplane fluoroscopy cadaveric study showed high shear forces on the BRP in the following shoulder positions[6]:

- forward flexion and internal rotation
- neutral position
- neutral position and internal rotation

Coracoid Impingement

Coracoid impingement can be another cause for anterior shoulder pain. It is defined as the impingement of the subcoracoid bursa and subscapularis tendon between the coracoid and lesser tuberosity. It has been described as a potential cause of degenerative wear of the pulley sling and subscapularis tendon insertion,[35] but not mechanical wear of the LHB, as it slides up and down in the bicipital groove.

The coracohumeral interval (CHI) can be measured on axial cuts of cross-sectional images and is defined as the shortest distance between the humeral head and the coracoid tip (Fig 4). There is no consistency in the literature regarding reference values for the CHI. Gerber et al.[36] found a mean distance of 8.7 mm on computed tomography scans in healthy subjects with the shoulder in adduction, whereas Giaroli et al.[37] found a sex-adjusted CHI of 10.5 to 11.5 mm in patients with coracoid impingement on magnetic resonance imaging. Our own data suggest that narrowing of the CHI distance on magnetic resonance imaging is related to pathologies of the LHB and rotator cuff. We found a mean distance of 10.2 mm for subjects with

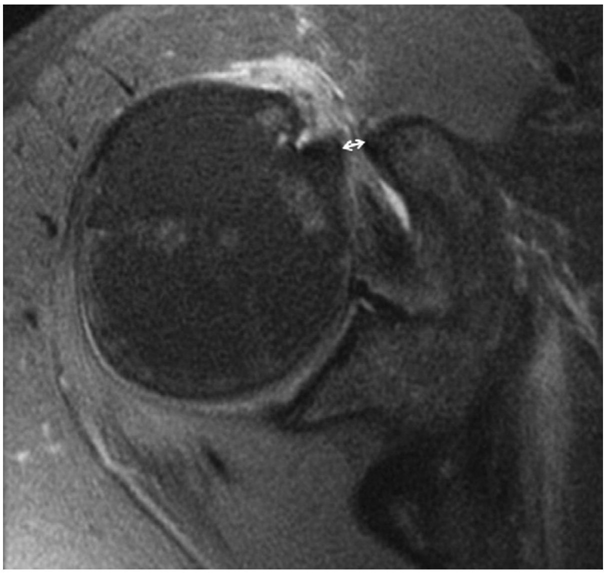


FIGURE 4. Cross-sectional magnetic resonance image of a patient with narrowed CHI (i.e., shortest distance between humeral head and tip of coracoid) (arrow).

anterior shoulder pathology and 12.3 mm without anterior shoulder pathology (unpublished data, S.B., November 2010).

SLAP Lesions

The pathology of the superior labrum was first described by Andrews et al.[9] in 1985. It was subsequently described by Snyder et al.,[38] who labeled the pathology "SLAP lesion," because of the location at the superior labrum extending from anterior to posterior (Figs 5 and 6). Snyder et al. also defined 4 different types of SLAP lesions (types I to IV), which were later supplemented by 3 further types (types V to VII).[38] The incidence according to Maffet et al.[39] is 11.8% (84 of 712 patients), but there is substantial interobserver and intraobserver variability even among experienced shoulder arthroscopic specialists with regard to diagnosis and treatment of SLAP tears.[40] SLAP lesions can be caused by recurrent micro-traumatic impairment, mainly in overhead athletes, or by single traumatic events.[41] The type of SLAP lesion typically dictates treatment.

These injuries are not limited to young throwing athletes as initially described. They are certainly more ubiquitous and may be seen in varying patient populations. Studies have shown that rotator cuff tears are frequently associated with concomitant labral lesions.[42] In a study performed by Miller and Savoie,[43] 74% of individuals with full-thickness rotator cuff tears had associated intra-articular lesions, with labral tears being the most commonly associated disorder. Snyder et al.[44] showed that 40% of 140 arthroscopically examined superior labral lesions were associated with full- or partial-thickness rotator cuff tears.

Biceps Tendinitis

Slatis and Aalto[45] have classified biceps lesions into 3 categories: impingement tendinitis, subluxation of the biceps tendon, and attritional tendinitis. Biceps tendinitis is inflammation of the LHB (Fig 7) and most often attributed to surrounding shoulder pathology such as degenerative rotator cuff lesions and impingement syndrome, and it has typically been characterized as a secondary process.[46] Primary tendinitis is rare and has been estimated to represent about 5% of the cases.[47] There is a general consensus, however, that tendinitis is the common pathologic process in both primary and secondary degeneration of the tendon.[48]

Treatment of biceps tendinitis has included open decompression of the transverse humeral ligament, which

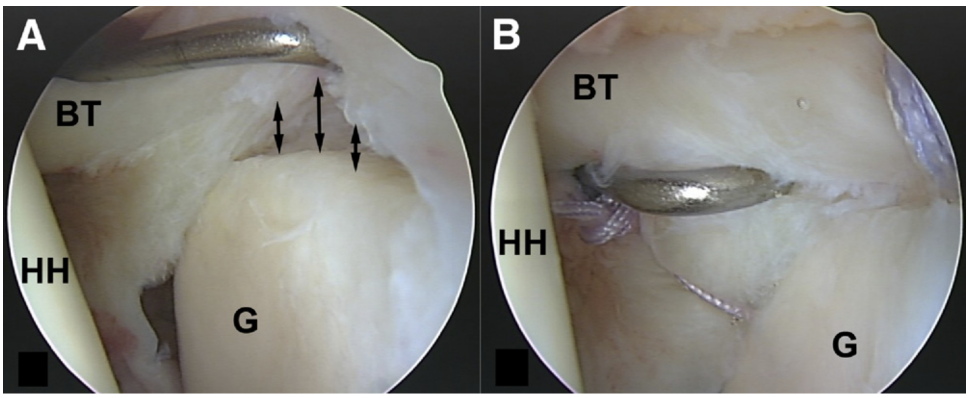


FIGURE 5. Arthroscopic view of a left shoulder from posterior showing SLAP lesion (arrows) (A) before and (B) after repair. (BT, biceps tendon; G, glenoid; HH, humeral head.)

FIGURE 6. Coronal magnetic resonance image of a left shoulder showing SLAP lesion (arrow). (HH, humeral head; G, glenoid; Acr, acromion.)

was initially proposed by Neer,[49] who released the transverse humeral ligament and decompressed the biceps tendon sheath in an effort to address secondary biceps pathology. Transverse humeral ligament decompression along with synovectomy of the inflamed biceps tendon as an arthroscopic procedure has been advocated for the treatment of isolated biceps tendinitis.[50-52]

A specific subtype of bicipital tendinitis has been attributed to the hourglass-shaped LHB tendon, as visualized by magnetic resonance arthrography.[53] The mechanical symptoms are attributed to a thickened, inflamed intra-articular LHB that engages the superior aspect of the bicipital groove during shoulder motion. The hourglass lesion has been likened to a trigger finger of the shoulder in that it prevents the normal excursion that occurs with abduction. This type of pathologic variant is best addressed by subpectoral biceps tenodesis (Figs 8 and 9).[54]

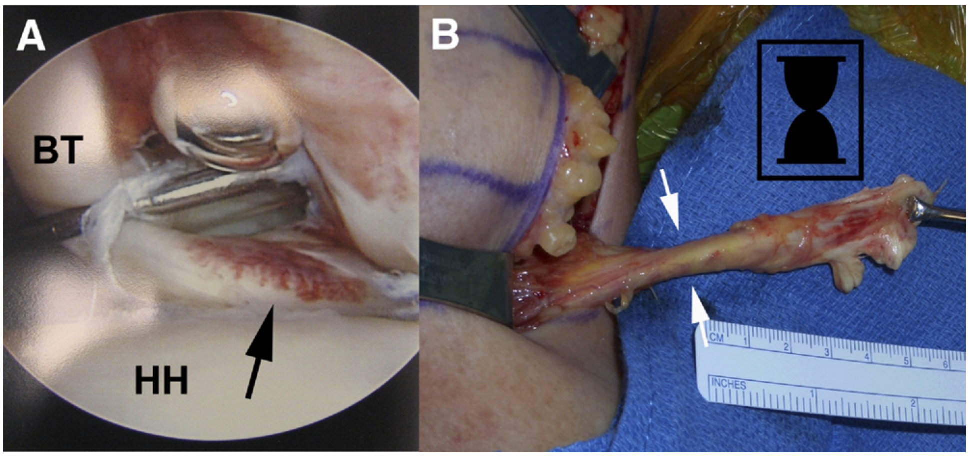
CURRENT TREATMENT STRATEGIES

LHB pathologies can be addressed by nonoperative treatment, reconstructive techniques, and tenodesis/tenotomy.

Conservative treatment of biceps rupture usually results in relatively little functional impairment of the shoulder.[28] Research at our institution found no statistical difference at the elbow joint in forearm supination or elbow flexion strength when comparing tenotomy, tenodesis, and control groups.[55] Because of the minimal functional sequelae of biceps ruptures in middle-aged and older patients, surgical repair is indicated only in those with persistent spasm or in those whose occupations require significant supination strength. Surgical repair is also indicated in younger, more physically active patients or in those in whom the minor strength or cosmetic effects of conservative treatment are unacceptable.[2,29]

In the case of a tenodesis, the intra-articular portion of the tendon is resected and the proximal portion of the remaining tendon is fixed to the proximal humerus (Fig 8).[2] In some cases a simple arthroscopic release of the tendon may be performed.[2,56] There is a lack of quality evidence to advocate tenodesis versus tenotomy.[57] It has been suggested that tenodesis results in less strength loss compared with conservative treatment for tendon

FIGURE 7. (A) Arthroscopic view of a right shoulder from posterior showing biceps tendinitis (black arrow). (B) Intraoperative view of LHB after tenotomy with an hourglass lesion, with white arrows pointing to narrow part of tendon (right shoulder). (BT, biceps tendon.)



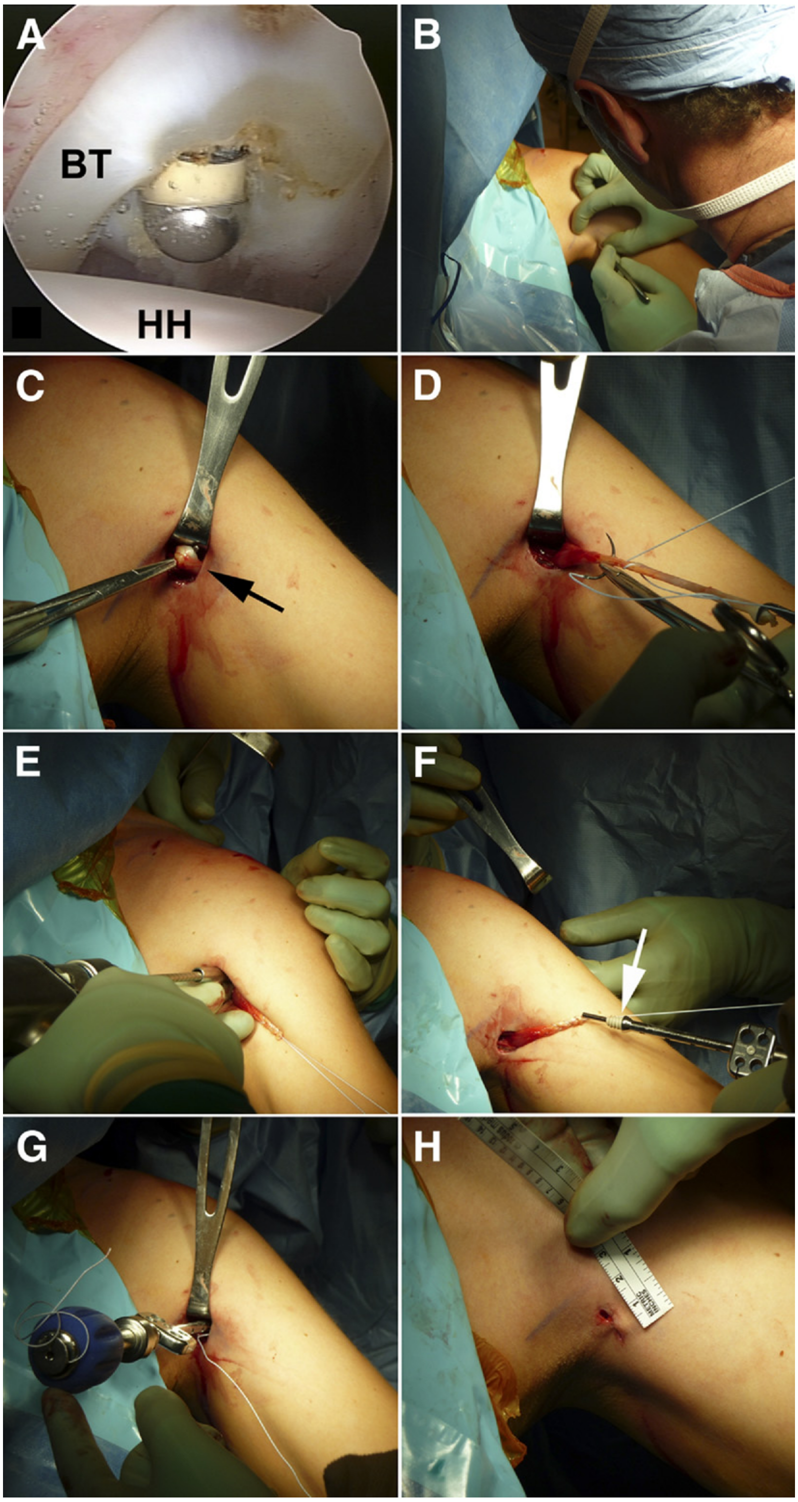


FIGURE 8. Intraoperative images of subpectoral biceps tenodesis of a left shoulder. (A) Arthroscopic view of left shoulder showing tenotomy of biceps tendon (BT) with radiofrequency device. (HH, humeral head.) (B) Subpectoral skin incision. (C) Finding of BT after tenotomy. (D) Preparation of BT for fixation, with stitches for secure bite of interference screw (arrow in F). (E) Drilling of monocortical hole for BT and interference screw. (F and G) Preparation and insertion/fixation of BT with interference screw (arrow). (H) After placement of 1 subcutaneous stitch, the incision measures only 1.5 cm in length.

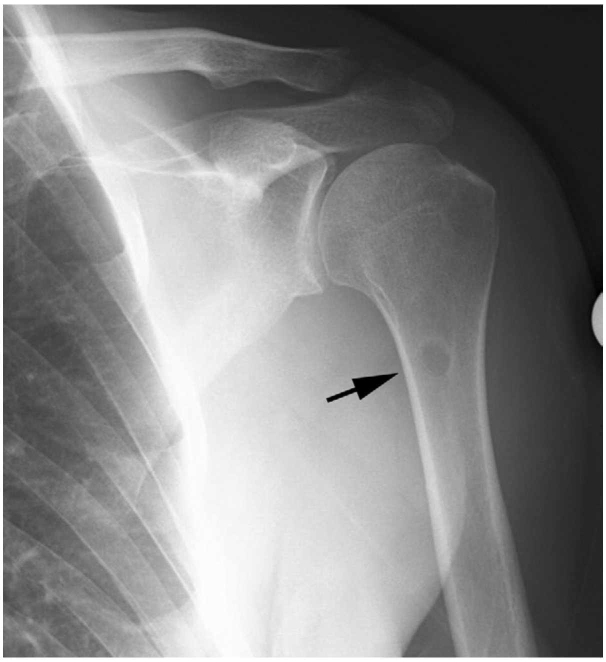


FIGURE 9. Postoperative anteroposterior radiograph of a left shoulder after subpectoral biceps tenodesis (arrow).

rupture[58] and less risk of postoperative cramping and improved cosmetic results.[59,60] Multiple techniques for LHB tenodesis have been described. Tenodesis of the LHB may be performed arthroscopically or in an open manner, either above the bicipital groove or through a subpectoral approach. Fixation techniques include suture anchor fixation, suture–to–adjacent tissue fixation, keyhole-to-bone fixation, and interference screw fixation.[61-69]

Biomechanics

Mazzocca et al.[70] compared cyclic displacement and ultimate failure strength of open subpectoral bone tunnel biceps tenodesis, arthroscopic suture anchor tenodesis, an open subpectoral interference screw fixation technique, and an arthroscopic interference screw technique. They did not find statistically significant differences in ultimate failure strength among any of the methods tested. Other investigators have found superior biomechanical properties for bioabsorbable interference screw fixation compared with suture anchor fixation.[71,72] However, Millett et al.[73] found no statistical difference between interference screw and suture anchor fixation for mini-open subpectoral biceps tenodesis.

Clinical Results

Numerous studies have shown good results after proximal biceps tenodesis.[65,74-76] However, persistent tenosynovitis or stenosis after arthroscopic or proximal groove tenodesis may cause residual pain in the bicipital groove[77] and higher failure rates.[59] Becker and Cofield[78] found unsatisfactory long-term outcomes after proximal biceps tenodesis in approximately 50% of cases, with a reoperation rate of 15%. They concluded that failures were likely to exhibit subacromial impingement with a rotator cuff tear. According to recent studies, it is more likely that high failure rates after proximal tenodesis are caused by persistent tenosynovitis and pain.[59] Authors of one study found significantly decreased revision rates after subpectoral tenodesis.[59] Another study on the incidence and types of complications after an open subpectoral tenodesis procedure found a low complication rate of only 2% in a population of 353 patients over the course of 3 years.[79]

Nonetheless, tenodesis, tenotomy, and conservative treatment of LHB pathology all fail to address the potential loss of superior[15] and anterior[12] stability of the glenohumeral joint. The absence of an LHB tendon may have particular implications for throwers and very young patients in whom instability may lead to long-term functional deficits.[2]

Symptomatic pulley lesions can also be treated by tenodesis or tenotomy. Some authors perform bicipital pulley repair,[80] although there are no prospective studies comparing the different treatment options. Depending on the type of SLAP lesion, arthroscopic repair or debridement of the torn tissue can be performed with predictably good results. In more severe cases, biceps tenodesis may be preferable. There is evidence to suggest that return to sport may be improved with biceps tenodesis compared with SLAP repair.[53,62]

Little evidence exists in the literature about treatment of superior labral lesions in patient populations aged over 45 years with concomitant rotator cuff tears. A recent study published by Abbot et al.[81] showed that patients with debridement and rotator cuff repair had better results than patients with SLAP repair and rotator cuff repair. Studies also indicate that patients with tenodesis or tenotomy have less pain after rotator cuff repair compared with cases where the LHB has been preserved. A recently published study on tenodesis versus repair of type II SLAP lesions suggests that patients with tenodesis have a higher satisfaction rate and are able to return to their previous level of sports more frequently.[82] These results suggest that LHB tenodesis may yield superior results over SLAP repair for certain patient groups.

CONCLUSIONS

Biomechanical studies in cadaveric models fail to "recreate the myriad factors that act in synergy to provide glenohumeral stability in vivo."[16] A consistent limitation of cadaveric biomechanical studies is their failure to apply physiologic loads to the LHB. There is no consensus in the literature, and values range from 11 to 55 N. In future studies LHB loading conditions should reflect in vivo muscle activation levels.

The function of the LHB tendon and its role in glenohumeral kinematics remain poorly understood because of the paucity of literature and the difficulty of performing biomechanical cadaveric and in vivo studies. LHB tenodesis has become a popular surgical treatment for managing isolated and combined pathology of the LHB. Our own research with biplane fluoroscopy in vivo testing indicates that the role of the LHB in glenohumeral kinematics may have been overestimated. Future biomechanical research should focus on in vivo studies to investigate any adverse effects of removing the intra-articular portion of the LHB on glenohumeral kinematics.

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