Introduction
Evaluation and treatment of the long head of the biceps tendon (LHBT) continues to be a source of debate. This review is aimed at discussing the anatomy, biomechanics, pathology, diagnosis and management of the LHBT. An extensive literature review was performed to help guide in management of disorders of the LHBT.

Background
The LHBT has been a topic of discussion for many decades, and while it is becoming better understood it remains controversial with regards to function, diagnosis and treatment. The first known depictions of the biceps tendon date back to the early 1400s when it was noted in dissections performed by Vesalius. William Cowper reported one of the first pathologic etiologies of the LHBT in 1694 as a dislocation of the LHBT. It wasn’t until 1841 when this diagnosis was finally “proven” during an autopsy. Meyer subsequently discussed LHBT ruptures and dislocations in the 1920s where it was attributed to attrition. During the 1930s through 1950s the diagnosis of LHB tendinitis began appearing in the literature. Since these early discussions of the LHBT, a significant amount of energy has been focused on its significance with regards to anatomy, function, role in shoulder pain/pathology and management of various disorders. Although it was originally described with such phrases as “Proverbial stepchild of the shoulder,” “Appendix of the shoulder,” and “Somewhat of a maverick, easy to inculpate but difficult to condemn,” recent focus has turned to recognizing it as a source of pain and dysfunction in the shoulder that should be evaluated and treated appropriately. Evaluation and appropriate treatment remain controversial but trends in treatment are continuing to develop.

Anatomy
The LHBT originates at the supraglenoid tubercle and superior glenoid labrum. It then obliquely courses through the glenohumeral joint in an intra-articular but extra-synovial fashion before exiting the shoulder joint at a 30° to 40° angle via the biceps reflection pulley and the bicipital groove. The tendinous portion of the LHBT measures 9 to 10cm in length and joins with the short head inserting on the radial tuberosity. The long head of the biceps tendon is stabilized within the joint by the biceps reflection pulley and as it exits the joint by the osseous anatomy of the bicipital groove with the subscapularis forming its roof. The LHBT receives the majority of its vascularity from the anterior circumflex humeral artery, with a small portion coming from the suprascapular artery. There is a watershed area described residing between the biceps reflection pulley and the bicipital groove. Alpantaki performed immunohistochemical testing and found a high concentration of sensory nerve fibers in the proximal portion of the tendon.

Biomechanics
LHBT function remains controversial. Opinions vary widely, from no function in the shoulder to providing glenohumeral stability in all directions. There are also thoughts in between, including humeral head depressor and providing glenohumeral stability especially when rotator cuff or labral pathology is present. Pagnani in 1996 applied 55 N to LHBT in cadaveric specimens and demonstrated effect of humeral head depression. Kuhn demonstrated (2005) resistance to external rotation by LHBT in a cadaveric study with 44.5 N of force applied. Some authors have called into question these findings as it is felt LHBT cannot generate these amounts of force in vivo. EMG studies performed by Bassett and others call into question the amount of in vivo force the LHBT can generate, with their most recent findings suggesting the LHBT load is more reasonably estimated at 11 N. In 2 separate in vivo radiographic studies Warner and Kido demonstrated humeral head depressor function of the LHBT in shoulders with intact or disrupted rotator cuff tendons. Some authors have called these findings into question.

Pathology
Disorders of LHBT are associated with rotator cuff tears (RCT) in up to 90% of cases and also associated with glenohumeral arthritis. Isolated lesions on the LHBT are very rare and occur mostly in young overhead athletes. The main lesions found in the LHBT can be grouped into 3 broad categories: inflammation, instability, and ruptures. The first category, inflammation, is comprised of lesions varying in a spectrum of mild irritation to extensive fibrosis. Common etiologies leading to the spectrum of LHBT inflammation are related to anatomy of the bicipital groove and possibly overuse injuries with hypovascularity occasionally being implicated. The next category, instability, can lead to the disruption of the tendon’s mechanical properties and thereby decrease its function and/or cause pain. Lesions that may lead to instability include subscapularis tendon tear, RCT, impingement, pathology of biceps reflection pulley, and superior labral anterior to posterior tears. In
the final category, ruptures, these conditions are closely linked to the first 2 categories in that chronic inflammation or chronic instability can result in weakened mechanical properties of the tendon and eventual rupture. The other main cause of rupture of LHBT comes from trauma resulting in partial or complete tears. Partial ruptures may remain painful for extended periods of time whereas complete ruptures may be painful in the short term with pain subsiding in the long term.

**Diagnosis**

Patients typically present with anterior shoulder pain that may occur at rest, with lifting, with overhead activities or they may feel popping or catching. Patients may recall a specific injury or may state that symptoms began without a known inciting event. Standard shoulder radiographs should be obtained and often are normal in appearance.

Many physical exam tests have been described for evaluating the LHBT. The sensitivity and specificity of each test varies greatly. The commonly performed Speed’s test has a specificity of 81% but only has a sensitivity of 54%. The bear-hug test has a sensitivity of 79% but only 60% specificity, making it difficult to diagnosis LHBT pathology solely from physical exam. Magnetic resonance imaging (MRI) can be helpful in identifying inflammation, thickening, instability, and partial or full thickness ruptures. MRI has been shown to have 52% sensitivity and 27% specificity with a low interobserver reliability. However, MRI arthrogram may increase sensitivity to 90% and specificity to 95% and many feel that the 3 Tesla MRI magnet approaches these values, as well.

Ultrasound can also be used in diagnosis but it is highly operator dependent and therefore is not utilized on a regular basis by most clinicians. Diagnostic injections have also been utilized to help in diagnosis but a recent study by Hashiuchi suggests that ultrasound be used to insure the solution is injected around the biceps tendon, as he found a 27% injection accuracy without use of ultrasound and a much improved 87% with the ultrasound. Arthroscopy remains the gold standard for diagnosing pathology within the LHBT.

**Treatment**

Treatment generally starts with non-operative modalities, including activity modification, physical therapy, non-steroidal anti-inflammatory drugs (NSAID), and steroid injections. In general, non-operative treatments provide good results and patient satisfaction. When pain and disability continue despite non-operative management, surgical intervention can be discussed. There are a variety of surgical treatments to address LHBT pathological conditions but the mainstay of surgical treatment is tenodesis or tenotomy of the LHBT, with surgical repair of concomitant shoulder pathology, as isolated lesions of LHBT are rare. Current trends demonstrate tenotomy being recommended more in elderly patients while tenodesis is being recommended in young, active patients and especially patients involved in heavy labor. There is no clear evidence demonstrating that either tenotomy or tenodesis is superior, but patients should be informed of key aspects and outcomes of these 2 different procedures. In complete ruptures, patients should be counseled that non-surgical management may result in 21% loss of supination strength and 8% loss of elbow flexion strength. It is generally accepted that less strength will be compromised in patients undergoing tenodesis for complete ruptures. Patients should be counseled regarding the possible cosmetic outcome of rupture of the LHBT resulting in a Popeye deformity of the biceps muscle belly. Although complete rupture of the LHBT does not guarantee a biceps muscle deformity, the risk of experiencing a Popeye deformity is 33% higher in the non-operative group when compared with the tenodesis group. However, recent literature has shown that patients may be less worried about this deformity than their surgeons are. Various surgical techniques have been described for LHBT tenodesis, including soft tissue fixation, osseous fixation, and the level at which the tenodesis is performed. No variation has been shown to be superior and most current literature recommends using the technique with which the surgeon is most comfortable and efficient.

**Conclusions**

LHBT function remains not well understood. LHBT pathology is usually accompanied by other shoulder pathology and is rarely found in isolation. Diagnosing LHBT pathology can be challenging and surgeons are encouraged to use a variety of diagnostic avenues including history and physical exam, x-rays, MRI, ultrasound, and diagnostic injections. Arthroscopy remains the gold standard for assessing LHBT pathology and so surgeons should be prepared to address pathology intra-operatively even when lesions may not be suspected pre-operatively. Treatment with tenotomy or tenodesis is patient dependent. If using tenodesis, the technique the surgeon is most comfortable with should be used, as there are no obvious advantages of one over another. Patients should be counseled pre-operatively regarding possible loss of strength and possible deformity of the biceps muscle belly, which may occur with either tenotomy or tenodesis.

**References**


