# Long head of biceps: from anatomy to treatment

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ACTA REUMATOL PORT. 2015;40:26-33


## ABSTRACT/RESUMO

The long head of the biceps (LHB), the tendinous structure of the proximal brachial biceps, has its well-known anatomy, which contrasts with its limited current functional characterization. Various forms of proximal anchor and intra-articular route, important for the correct interpretation of its contribution to the pathology of the shoulder as well as the treatment methodology, are described. Knowledge of its biomechanics results mainly from cadaveric studies that contradict each other. Already the few studies in vivo indicate a depressant and stabili-zing action, anterior, for the humeral head. Its pathology is rarely isolated because it is almost always correlated with rotator cuff or labrum pathology. It can be divided into 3 major groups (inflammatory, instability and traumatic) and subdivided according to its location. The anterior shoulder pain is the initial symptom of pathology of LHB Its perfect characterization is dependent on the associated injuries. Clinical tests are multiple and only their combination allows better sensitivity and specificity for LHB pathology. The arthro-MRI and dynamic ultrasound are able to increase proper diagnostic of the pathology of LHB. Treatment ranges from conservative and surgical. The latter includes the repair, tenotomy and tenodesis of LHB, which can be performed by open or arthroscopic methodology. The author intends to review existing literature on all aspects related to the long head of the biceps from anatomy to treatment, presenting the latest results.


**Keywords:** Long head of the biceps; Anatomical variants; Rotator cuff; Labrum; Tenodesis; Tenotomy

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## ANATOMY

The long head of the biceps (LHB) originates from the supraglenoid tubercle of the scapula in continuity with the glenoid labrum[1,2]. The insertion is located medial to the glenoid articular rim, creating a subsynovial recess, which may be posterior, predominantly posterior, anterior and posterior, and also predominantly anterior[2].

In the anterosuperior part of its insertion and in its continuity with the labrum, three anatomical variants have been described. In the study by Rao *et al*. normal insertion was found in 86 % of patients and in the remaining 14 % there was an isolated sublabral foramen (3.3%), one sublabral foramen associated with a middle cord-like glenohumeral ligament (8.6%) and complete absence of anterior and superior labrum associated with a cord-like of middle glenohumeral ligament (1.5 %)[3].

In its intraarticular portion of the LHB takes several variants (12), dependent on anatomic criteria and its mechanic behavior to arthroscopic mobilization[4]. These are grouped into 4 large families and are dependent on the migration of LHB during embryonic growth, passing for an extra-articular structure to an intrarticular one through the joint capsule[5]:

1. "meso" family: in this group LHB has a free movement beneath the rotator cuff;
2. "adherent" family: LHB is very adherent to the rotator cuff;
3. "split" family: the LHB is divided intra - articularly;
4. absence of LHB.

In the "meso" family there are 5 types. In the first there is a small cord, vascularized, between LHB and the rotator cuff. In another there is a synovial band from medial to lateral that is never in stress between the LHB and the rotator cuff. In a third type, the presence of a pulley based in the rotator cuff involves the LHB without trapping it and allowing it to slide freely. When this

pulley allows movement without letting it slide, we are in the presence of the fourth kind. Finally, there is the presence of a vascular synovial sheath and not just a pulley with freedom of movement without sliding of the LHB.

In the "adherent" family, and its first type, there is a strong and partial grip, from the medial part of the LHB extending down to the lateral part of the rotator cuff and that is in tension when placing the limb in abduction, pulling the supraspinatus inferiorly. When this adherence is essentially lateral, leaving the medial part of the LHB free, relaxing when the limb in abduction, we are in the presence of the second type. In the third type there is no mesotenon but a thick synovial covering both the LHB in front and behind in continuity with the synovial capsule and not allowing any mobility. Finally, rare, the LHB is completely compliant in the thickness of the supraspinatus without insertion in the medial glenoid tubercle.

In the "split" family we have 2 types: in the first, the LHB originates partially on the surface of the supraspinatus and partially in the glenoid, joining before emerging the groove and in the second the source is single but in emergency it is divided with a part out to the groove and another to join the most lateral part of the capsule, relaxing with adduction without limitation of the LHB slipping (Figure 1).

Finally we have a lack of embryological LHB, the fourth family[4].

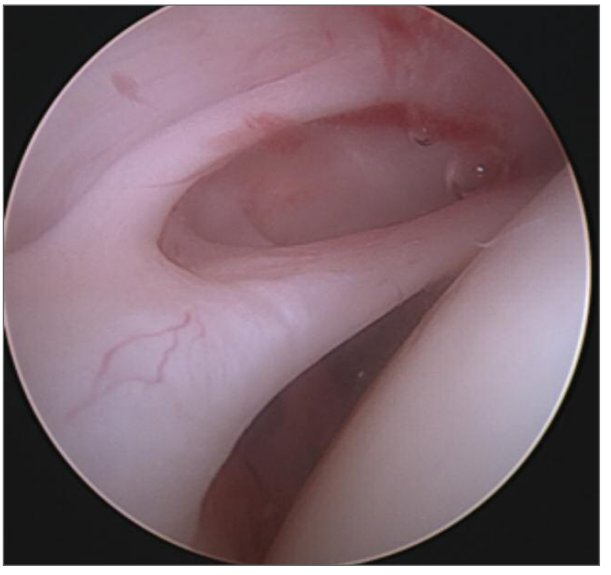

**FIGURE 1.** Split LHB, with a unique origin at the supraglenoid tubercle and divided at the emergence of the groove

The vascularization of the most proximal part of the LHB is made from ascending vessels of the anterior humeral circumflex artery while the distal part is irrigated by branches of the brachial and deep brachial arteries[6]. There is a hypovascular area from 1.2 to 3 cm from the origin, which corresponds to the sliding part in its groove[7].

The network of innervation, sensitive and sympathetic, it is also more exuberant in their anchor insertion than distally in the muscle-tendon junction[8].

Intra-articular tendon LHB has a diameter of 5--6mm and a length of 9 cm[9] and slides, on average, 18 mm inside the joint in anterior flexion and internal rotation movement as compared with the neutral position[10].

Upon entering the bicipital groove, the LHB undergoes a twist of 30°-40°[11] and is stabilized by the morphology of the groove that has a depth of 4 mm and an opening angle of the medial wall which can reach 56°[12]. The remaining stability is conferred to it by the roof of the biceps pulley, which consists of fibbers of superior glenohumeral ligament, coracohumeral ligament, supraspinatus and subscapularis, insertion of the pectoralis major tendon and falciform ligament[13,14].

The short head of the biceps originates from the coracoid apophysis in the most lateral part of the conjoined tendon. It forms the medial part of the biceps mass and in its distal insertion, in the proximal bicipital tuberosity of the radius bone, both suffer an external rotation of 90°. So the LHB has a more proximal insertion conferring supinator function while the short head, more distal, has essentially flexion function of the elbow[15,16].

## FUNCTION

Most biomechanical studies on the function of the LHB were performed on cadavers and focused on its effect on the stability of the glenohumeral joint, with controversial results[17].

It is relatively consensual its stabilizing action of the glenohumeral joint when the limb is in abduction and external rotation[18,19].

*In vivo* biomechanical studies shown a proximal migration of the humeral head when the LHB was absent or when unstimulated, so it could be concluded that it acts as a humeral head depressor[20,21]. There has also been an anterior translation of the shoulders when compared to the contralateral[22].

## PATHOLOGY

Diseases of the LHB can be classified into 3 broad groups: (1) inflammatory, (2) instability or (3) traumatic. Meanwhile we can subdivide each group by anatomical location, the pathophysiological process and the state of the LHB[23,24].

## LHB TENDINITIS

The inflammatory process of LHB is associated with pathology related to surrounding tissues like the rotator cuff (90%), the subacromial impingement or the glenohumeral arthritis. Its isolated primary inflammatory process is rare (5% of cases)[25,26] (Figure 2).

This tendinopathy is characterized by chronic inflammatory process, fibrotic degeneration and decreased tenoblastic capacity associated with increasing release of neurotransmitters such as CGRP (calcitonin gene related peptide) and P substance[27].

If this process becomes hypertrophic might cause a blocking of the tendon in its intra-articular sliding portion or in the groove by narrowing at this level - " hour--glass biceps " - which acts as trigger lesions in the finger flexors of the hand[28].

## LHB RUPTURE

The ongoing process of the inflamed tendon friction with the movements of the shoulder can lead to a process of macroscopic delamination of the tendon (Figure 3), with partial and subsequently complete rupture.

The ruptures are mainly located at the origin of the tendon or at the emergence of groove, and this corresponds to the hypovascular zone[7,17]. When they occur they are generally associated with a symptomatic relief and a deformity of the distal biceps mass migration called the Popeye signal[17,22]. These ruptures are more common over the 50's years, and with a higher incidence (96%) than the short head portion or the distal biceps[29].

## INSTABILITY

LHB becomes very unstable due to the non-integrity of its emergence in its groove. This instability can range from subluxation to complete dislocation both to the medial or posterior lateral side. In the first case, it is associated with rupture of the subscapularis tendon (Figure 4) and the second with previous rupture of the supraspinatus[30]. Habermeyer *et al.* divided these instabilities into 4 groups after arthroscopic visualization: type I - isolated lesions of the uppermost part of the superior glenohumeral ligament (SGHL), type II - SGHL injury associated with partial articular supraspinatus rupture, type III – SGHL injury associated with rupture of the articular part of the subscapularis and type - IV SGHL injury combined with partial rupture of the supraspinatus and subscapularis[11].

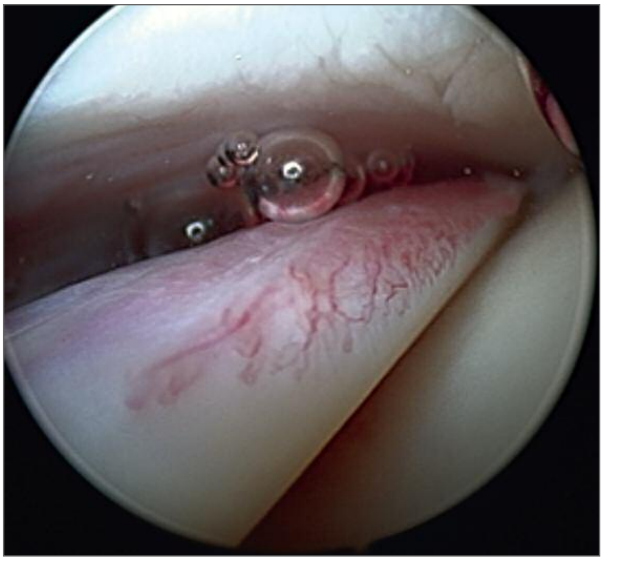

**FIGURE 2.** Inflammatory signals in the intraarticular portion of the LHB

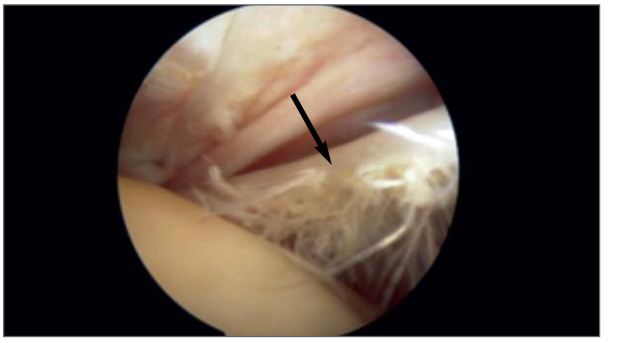

**FIGURE 3.** LHB delamination that affects 50% of its diameter

## SLAP LESION

This entity refers to the insertional lesions of the LHB

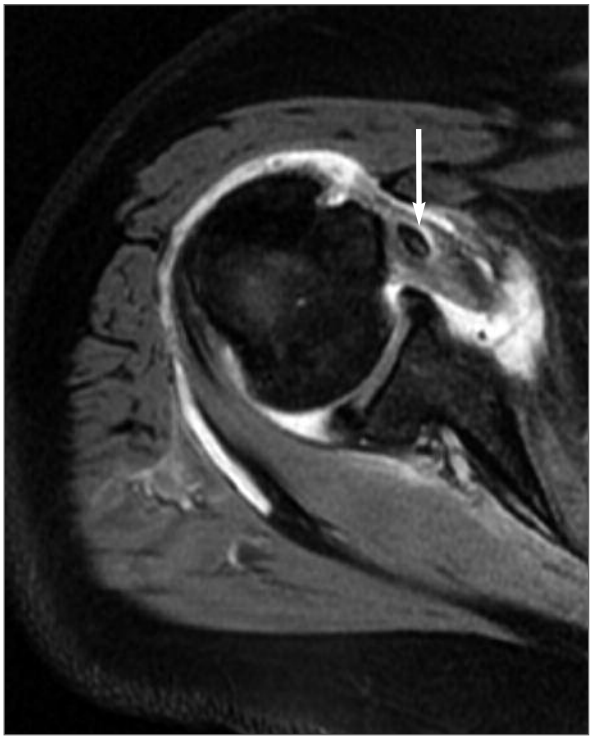

**FIGURE 4.** LHB instability, medialized because a concomitant subscapularis tendon tear

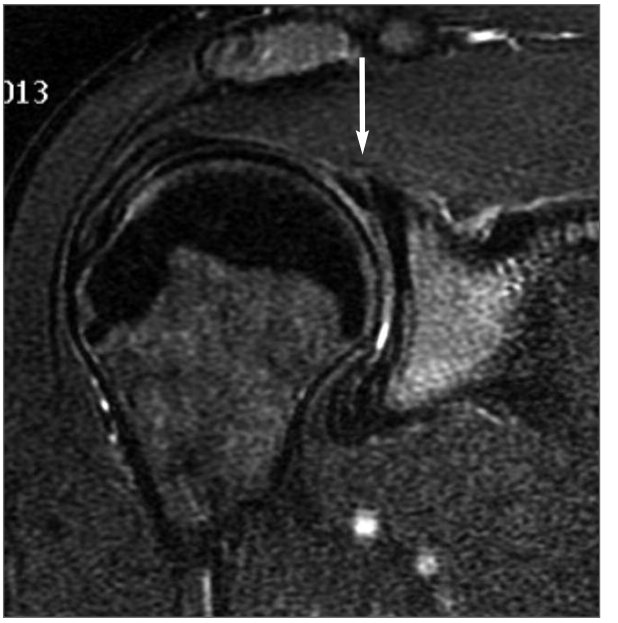

**FIGURE 5.** SLAP lesion with a well-defined plane between the supraglenoid tubercle and the LHB tendon origin

at labrum level, normally starting at a posterior level and subsequently extend to anteriorly (Superior Labrum Anterior and Posterior), beginning by peeling or by grinding, with or without involvement of the LHB tendon itself (Figure 5).

The classic description of this injury made by Snyder *et al.* in 1990 has 4 types and is still the most widely used[31]. Later, more types were added (type V to X), despite its limited clinical applicability and generating therapeutic guidelines[32,33]. In type I there is a fraying of the labrum and integrity at the biceps anchor. In type II, the LHB anchor is detached from its bed. In type III there is a basket handle rupture of the anchor that falls into the glenohumeral joint and type IV this basket handle is associated with rupture of the tendon extending through and along the LHB[31].

## CLINICAL PRESENTATION

The typical history of LHB is the appearance of pain in the anterior aspect of the shoulder, of insidious onset that worsens over time associated with signs/symptoms of rotator cuff or subacromial impingement. Sporadically feeling/observation of instability of the tendon at the level of the groove and distal migration of the biceps mass when complete rupture may occur – Popeye's sign[34].

The awakening or worsening of pain on palpation on the path of LHB on the groove and at the level of pectoralis major insertion is very suggestive and should be supplemented with oriented clinical test for LHB[35,36].

In the broad spectrum of clinical test to assess the integrity of the LHB, the bear hug and upper cut were the most sensitive (0.79 and 0.73, respectively) while the belly press and Speed's test the most specific ( 0.85 and 0.81 respectively). The association of upper cut and Speed's test seems to be the best positive predictor group of tests we have to identify pathology of LHB[36].

We should always evaluate for the possibility of pathology of the cervical spine, shoulder girdle and scapular- thoracic joint with which it makes differential diagnosis[34].

## COMPLEMENTARY DIAGNOSTIC BY IMAGIOLOGY

Conventional radiographs and MRI may allow to con-

firm the diagnosis but mostly can determine associated lesions of the rotator cuff[34]. Conventional radiology at AP, lateral and axillary views, allows to exclude degenerative pathology of the glenohumeral and acromioclavicular joints, and the presence of bone disease causing impingement[37]. MRI nevertheless has little diagnostic capacity for intra -articular lesions of LHP, LHP, and the sensitivity and specificity may slightly increase with arthro-MRI[38]. Ultrasonography (US) is useful in confirming tears or dislocations but especially in dynamic instabilities[39]. Regarding the latter, comparing its diagnostic capacity prior to surgery and intraoperative findings, US identifies 90 % of normal LHB and 88 % of full-thickness tears of the LHB but its diagnostic ability of intermediate lesions, as partial tears or inflammatory lesions drops to 27 and 22 %, respectively[40-42]. It has the advantage of being an inexpensive examination and easier to access, but very operator dependent[41,42]. Although no study with the same characteristics for MRI exist, this demonstrated a capacity of overdiagnosis for partial tears of the LHB and underdiagnosis for inflammatory disease[43].

## TREATMENT

Diseases of the LHB can be treated conservatively for technical reconstruction or tenodesis/tenotomy. We cannot ignore, as we have previously reported, that isolated lesions of LHB are rare and therefore its treatment methodology is dependent on the associated and concomitant lesions[22].

## CONSERVATIVE

It includes changes in daily activities, anti - inflammatory and analgesic medications, cryotherapy and physiotherapy treatments for associated pathology[17]. A corticosteroid injection into the sheath of the LHB, the subacromial space or intra-articular, with or without ultrasound support, can result in symptomatic relief, which is oriented to the associated lesions[17]. The injection under ultrasound support is demonstrably more accurate: 86.7 % of localization in tendon sheath versus 26.7 % when the injection is made in a non-guided manner[44].

As isolated pathology of the LHB is rare, also isolated injection to the sheath of the LHB is not very common and it is usually associated with subacromial bursa and/ or intra-articular treatments. Injection of the subacromial bursa or of the gleno-umeral joint, when effective, turns out to have a direct action on the primary causal mechanism of the pathology and secondarily in LHB. The injection to the LHB sheath is usually done with the patient in a sitting position after identifying the bicipital groove by palpation and tenderness[45].

Local anesthesia is most commonly made with lidocaine or bupivacaine[46]. Regarding the choice of corticosteroids, it can be divided into: 1) acetates (methylprednisolone acetate, betamethasone acetate, hydrocortisone acetate and dexamethasone acetate) versus phosphates (prednisolone sodium phosphate, betamethasone sodium phosphate), 2) fluorinated (betamethasone sodium phosphate, dexamethasone sodium phosphate, triamcinolone hexacetonide and triamcinolone acetonide) versus non-fluorinated (prednisolone, methylprednisolone, hydrocortisone). In the first group, acetates are less soluble and therefore of major indication for chronic conditions compared with phosphates, more soluble, so more prone to be used in acute situations. Fluorinated corticosteroids, in the extraarticular soft tissues, are associated with much higher rate of tendon ruptures and skin and subcutaneous atrophy, so their use should be avoided in this location/pathology if the treatment is not guided by image[46].

## SURGICAL PROCEDURES

The ideal type of surgery to deal with the LHB remains unclear and continues to be a source of controversy[14,47,48]. Many forms of tenotomy and tenodesis are described either arthroscopically or in open surgery. More consensual criteria than what surgical treatment perform in the LHB are when to act on it: partial rupture of the LHB, involvement of more than 25-50 % of its diameter, longitudinal delamination of the tendon that interferes with their ability to migrate in the groove, medial subluxation or pulley ruptures associated subscapularis injuries[14,49].

There have been many attempts to answer which of the treatment methodologies gets the best results. In a systematic review[48] in which were considered 5 studies of tenotomies, 8 studies of tenodesis and 8 which compared the two techniques, no significant differences in clinical outcomes were observed, both in percentage of success as in failure, except for the presence

of Popeye's sign (more prevalent in patients undergoing tenotomies). However it was suggested a correct design of a prospective and randomized study to be able to achieve results with greater clinical evidence.

In the decision on tenotomy/tenodesis algorithm it must also be considered the fact that the tenotomy has a simpler, faster and shorter post-operative recovery procedure while tenodesis consumes more surgical time, may have an associated cost of implant and there is greater restriction in the initial stage of rehabilitation[34].

While tenotomy is a consensual technical procedure, performed by arthroscopy at the LHB origin in the supraglenoid tubercle, and it should be verified its retraction into the groove and if it does not, remove the intra-articular stump[50]. The same consensus cannot be said for tenodesis.

Remains relevant the discussion of open or arthroscopic fixation technique approach, location and anchor point. The open approach is more appropriate at lower risk of recurrence in patients whose major concern is cosmetic, and also the most preferred in younger patients, athletes and heavier workers[17]. It is difficult to determine the correct length-tension relationship of the biceps mass and hence the numerous anchor points already attempted: lesser tuberosity[51], coracoid process[51,52], bicipital groove[53], the transverse humeral ligament, short head of the biceps[6], the pectoralis major tendon[6,9] or subpectoral bone tunnel[35,53]. Lately the controversy is centred on the location in the proximal half or the distal half of the bicipital groove/subpectoral. Lutton *et al.* in a retrospective case control study concluded that the most distal location favors the lower incidence of residual pain[54]. Furthermore, the revision rate is much lower (8 %) when the fixation is subpectoral, compared to arthroscopic proximal tenodesis (45%)[55].

The method of attachment may be performed by the use of anchor sutures, soft tissues sutures, bone tunneling or interference screw[56-58].

The technical reconstruction/repair is particularly useful at the treatment of SLAP lesions. The SLAP I, more frequent in elderly patients, are rarely an isolated source of pain, so the mechanical debridement is the most appropriate treatment[32]. The lesions of SLAP II, typical in the active young people, benefits in its anchoring fixation, which may be achieved with a single double wire anchor or with two anchors with a single wire for stabilizing the anterior and posterior biceps insertion pillars[32,59]. Type III lesions are usually treated with removal of the basket handle lesion and type IV depend on the degree of involvement of the tendinous portion of the LHB: greater involvement of the tendon requires tenotomy of the LHB with or without tenodesis; small involvements only debridement[60].

## CONCLUSION

The long head of biceps remains a little-known structure of a functional standpoint, which contrasts with its anatomy, where it is known a huge number of intra-articular variants and their relationship to the anterior and superior labrum.

The clinic manifestations are the most consistent criteria in decision making since the amount of provocative tests alone do not combine good specificity and sensitivity, allied by imagiological diagnostic procedures.

The determination of treatment is very dependent on the associated and concomitant diseases, since the pathology of isolated LHB is uncommon. The choice of treatment is not easier because of the lack of clinical studies that support accurate and clear guidelines.

It is uniformly accepted the need for well-designed studies to firstly clarify the role of LHB in the kinematics of the shoulder and in the other hand to help the choice of the best method of treatment for each disease process.

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