The Glenoid Labrum

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ORIGINAL ARTICLE

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ABSTRACT

The glenoid labrum is a critical structure within the gleno-humeral joint and commonly requires treatment by the shoulder surgeon. This review presents a concise summary of the embryology, anatomy, microscopy, biomechanical properties and clinical lesions involving the glenoid labrum. This knowledge will aid the clinician in understanding its function and pathology.

INTRODUCTION

The improvement in clinical imaging and the continual development of shoulder arthroscopy has allowed clinicians to diagnose and treat more subtle pathologies of the glenohumeral joint. Specifically, injuries to the labrum have been more recognized during this period as a source of symptoms and the optimal treatment of these lesions is evolving at a rapid pace. This review presents the current knowledge of the glenoid labrum.

EMBRYOLOGY

Synovial joints develop by the formation of a primitive anlage composed of the cartilaginous precursors to the individual bones, with a transverse band of flattened cells (interzone) separating these two sides of the joint. For the shoulder, the interzone within the anlage appears at 11 mm to 12 mm (crown–rump length (CRL) (6 weeks) with a vascular periphery, whereas the centre remains avascular and begins to cavitate at approximately 22 mm CRL (8 weeks) [1]. The dehiscence of the two sides is said to be complete by 34 mm CRL (9 weeks) [2] . The fibrous capsule is evident at 16 mm CRL (7 weeks) [2] and the anlage of the labrum by 17 mm CRL (7 weeks) [1]. By 21 mm CRL (8 weeks), the labrum can be seen to be deficient in the region deep to the coracoid process; in the rest of the circumference, a few collagenous strands start to appear. At 27 mm CRL (8.5 weeks), the labrum is more pronounced posteriorly than anteriorly. At this stage, it has been postulated that the anterior labrum deep to the subscapularis tendon has been formed from the synovial mesenchyme, as apposed to the labral anlage, because it does not join the cartilage of the glenoid [1].

More recent work [3] has specifically looked at the progression of the labrum and other intra-articular structures from 9 weeks gestation onwards. At 9 weeks (30 mm CRL), the glenoid labrum and biceps tendon can be visualized, with the biceps tendon inserting into the superior aspect of the labrum. At this stage, the glenoid fossa and humeral head are poorly developed. Microscopic assessment shows the labrum as a primitive fibrous condensation on the margin of the glenoid fossa, with intermingling of both the biceps and triceps tendons. A fibrocartilagenous transition zone can be seen between the fibrous labrum and the hyaline articular cartilage.

By 12 weeks (60 mm CRL), the fossa has become pear-shaped with increasing concavity. The humeral head is now a hemisphere and the surgical neck can be identified. The labrum has thickened, except in the anterosuperior part, where it appears meniscus-like. The superior, middle and inferior glenohumeral ligaments are beginning to appear as thickenings in the joint capsule.

At 16 weeks (120 mm CRL), the labrum has thickened again, the biceps appears as an extension of the superior labrum and the inferior ligament has a wide attachment to the anterior and inferior part of the labrum. Microscopically, at 16 weeks, the superior labrum has become more fibrous and vascular, whereas the posterior labrum appears more fibrocellular.

By 23 weeks (198 mm CRL), all the intra-articular structures had taken on the form that is seen in the adult shoulder, except the biceps tendon, which still appeared cord shaped. The posterior labrum is now more fibrocartilagenous and the whole of the labrum is more vascular.

At full term (40 weeks and 370 mm CRL), the labrum forms a well defined ring, deepening the concavity of the glenoid fossa. However, the thickness is still less anteriorly. The posterior labrum has become hypercellular with groups of chondrocytes,

chondrobalsts and fibroblasts. The biceps tendon now has a flattened appearance. Compared to the adult shoulder, the joint cavity is relatively smaller with less synovial covering and folds.

It appears that, even as early as 21 mm CRL (8 weeks), the anterosuperior part of the labrum is different to the rest of the labrum and may even be derived from different tissue. A further cadaveric study has also observed that an area of detachment of the labrum in the anterosuperior region was present in specimens beyond 22 weeks of gestation [4] and arthroscopic assessment of 20 foetuses found the anterosuperior labrum to be detached in 10% of the shoulders [5].

This area is of particular anatomical debate in the adult, and these studies suggest that this difference is presence from a very early age.

ANATOMY

The adult labrum is a circumferential fibrocartilagenous structure, variably attached to the glenoid rim, providing a site of attachment for the glenohumeral ligaments. The radial thickness, from its inner to outer circumference, varies from 2 mm inferiorly to 11 mm superiorly, and is between 5 mm and 9 mm deep, suggesting this fibrocartilagenous structure contributes to the glenoid depth by 30% to 50% [6,7]. It has been described as having a triangular cross-section superiorly and as a more rounded elevation inferiorly [8].

The anterosuperior region is the most inconsistent area of the labrum and, at arthroscopy, there may be a complete absence of any labral tissue in this region (1.5%), a sub-labral foramen (11.9%) [9] or a mobile labrum (26%) [10]. However, anatomical dissection studies have suggested the incidence of a sub-labral recess may be as high as 74% to 85% [11,12], with a sub-labral foramen in 11% [12]. A sub-labral foramen or hole is defined as a complete detachment of the labrum from the glenoid, as opposed to a sub-labral recess or sulcus, where the labrum is lifted off the glenoid at the articular surface but there is still a deeper attachment. It is probable that the variation in this region is a result of both the embryological development and confusion, or at least non-uniformity in nomenclature and recognition of these variables.

If this area has been derived from tissue other than the labral anlage, it is likely to have different mechanical properties. This may make it more susceptible to injury during further growth and could produce the wide range of variants seen in the adult shoulder. There is certainly an increasing prevalence of structural defects and tears with age [13–15] and it has been hypothesized that detachment in this region may represent a secondary synovialized labral tear sustained in the second decade of life [14,16]. The labrum may therefore have a loose attachment, attachment to the middle or inferior glenohumeral ligament or no attachment at all [8].

The Buford complex is an example of one of these variants whereby the anterosuperior labral tissue is absent and the middle glenohumeral ligament takes on a cord-like appearance [17,18]. Inappropriate 'repair' of this 'lesion' can result in both pain and limitation of external rotation.

As well as the glenohumeral ligaments, the biceps tendon inserts into the labrum. The point of insertion is variable but will attach at some point between the anterosuperior and posterosuperior quadrant [19–22]. The presence of the glenohumeral ligaments appears to be inconsistent [23,24] but, when present, the superior and middle glenohumeral ligament attach between the 12 and 1 o'clock position, whereas the anterior-inferior glenohumeral ligament has a much more variable insertion from between 1 and 5 o'clock [21,24] (The clock positions refer to a glenoid of a right shoulder, with the 12 o'clock position at the superior margin). A posterior superior glenohumeral ligament has also recently been described, although this does not arise directly form the labrum [25]. The long head of triceps, according to some reports [26,27], takes a partial origin from the inferior labrum as well as the infraglenoid tubercle.

The labrum takes attachment to the underlying glenoid. This is classically described as tear-drop or oval shaped [28] and has a small surface area compared to that of the humeral head. It has a mean anterior-posterior width of 27 mm, and a superior-inferior height of 34 mm to 35 mm [6,29]. The articular surface diameter of the humeral head varies from 36 mm to 52 mm [30]. As well as a difference in surface area, the radii of the hemi-ovoid humeral head and the concave glenoid differ significantly [31]. The radius of curvature of the humeral head is 23 mm to 24 mm in the anterior-posterior plane and 21 mm to 23 mm in the superior-inferior plane [29,32], whereas the radius of the glenoid is 32 mm in the anterior-posterior plane and 41 mm in the superior-inferior plane [29]. The bony glenoid depth is 5 mm in the superior-inferior plane, but only 3 mm in the anterior-posterior plane [29]. In summary, the bony dimensions demonstrate a large well-rounded humeral head balancing on a much smaller, shallow, relatively flat glenoid with unequal radii in the two planes. This allows for a large range of movement but does not produce a stable joint without substantial additional support by the soft tissues. These additional soft tissues are described as either passive or active stabilizers. The active stabilizers are the actuators that are also involved in moving the joint: the superficial muscles such as the deltoid, and the deep muscles of the rotator cuff. The static stabilizers include the capsule, gleno-humeral ligaments and the glenoid labrum.

MICROSCOPY

Microscopy of the labrum has again caused confusion with respect to its exact structure, with some studies describing the labrum as a fibrocartilagenous structure containing dense cartilaginous fibrous tissue and chondrocytes [14,33], whereas others describe the labrum as a fibrous structure with a small fibrocartilagenous transition zone between the hyaline cartilage and the fibrous labral tissue [8,26,34]. This discrepancy may be a result of both nomenclature and the exact position of the labrum from which the specimens were taken.

It has been demonstrated that the labrum is only vascular in its outer third and appears to obtain its blood supply from the periphery rather than the underlying bone. The superior and anterosuperior regions are less vascular than the inferior and posterior regions [8]. The suprascapular artery, circumflex scapular branch of the subscapular artery and the posterior circumflex humeral artery all contribute to the blood supply of the labrum [8].

It is known to contain free nerve endings in its periphery [35] and there is a suggestion that the labrum may be needed to allow full proprioceptive feedback [36]; however, as yet, there is no conclusive evidence. Golgi's, Ruffini's and Pacini's corpuscles, as well as free nerve endings, have been found in the glenohumeral ligaments, whereas the biceps tendon and labrum contain only free nerve endings [37].

Both scanning electron microscopy and transmission electron microscopy have helped to demonstrate the collagen architecture of the labrum [33,38]. This has revealed three layers: (i) the superficial layer, with randomly orientated and loosely packed collagen fibres, considered to aid in lubrication; (ii) an intermediate layer; and (iii) the core layer, which forms the bulk of the tissue. Within this core layer, the collagen fibres are tightly packed and well orientated in a circumferential manner. However, there is vertical, oblique and interweaving fibres anchoring the labrum to the underlying bone. There is connection between the labrum and the capsule, gastrohepatic ligaments (GHLs) and the biceps tendon with intermingling of fibres [33].

THE ROLE OF THE LABRUM

The exact functional role of the labrum is still undefined. It has been theorized to function as a chock-block, increasing the glenoid depth and resisting translation [6,39]. Concavity compression is an extension of this postulate, whereby the humeral head is compressed into the cavity of the glenoid by the rotator cuff musculature, further stabilizing the shoulder. This mechanism has been calculated to increase stability by 10% to 20% [6,40] with an intact labrum and may be a result of its role in centralizing the head within the glenoid [40]. The labrum also helps to maintain a negative intra-articular pressure within the joint, which itself confers stability [41]; the magnitude of this effect has not been quantified. Loss of the anterior-inferior labrum in cadaveric specimens has been shown to decrease the contact area of the articular surface of the glenohumeral joint and increase the mean contact pressure [42].

Huber and Putz [26] proposed the periarticular fibre system of the shoulder, which includes the GHLs, the labrum, the long head of triceps and the long head of biceps forming a complete ring of ligamentous and tendinous tissue that runs through the labrum in continuity. Their study of 42 cadaveric shoulders demonstrated the superior GHL inserting into the anterior aspect of the biceps anchor of the superior labrum. The biceps tendon then runs into the posterior labrum as far as the posterioinferior quadrant. In the inferior area, fibres from the long tendon of triceps run back into the labrum and this tendon often (38%) has fibres running into the anterior part of the inferior labrum. This is the insertional point of the inferior GHL, which they then describe joining the superior GHL by a 'band of tissue' spanning the anterosuperior quadrant and completing the ring. In agreement with this, other reports have also demonstrated a connecting band from the inferior GHL of the anterior inferior labrum to the biceps tendon [21]. This periarticular fibre system is proposed as a tension brace to buttress against the humeral head and spread the pressure over a wide area. The presence of the middle GHL is not commented upon at all.

Interestingly the Buford complex [18] may actually be the band described by Huber and Putz from the inferior GHL to the superior GHL. Even a 'normal' looking medial GHL may be the same band and highlights the difficulty in interpretation of the anatomy.

BIOMECHANICAL STUDIES

Simple shear tests around the circumference of the glenoid, which involved an increasing force applied to the labrum in a plane parallel to the surface of the glenoid [43,44] concluded that the 4 o'clock position was found to be the weakest and the 7 o'clock position the strongest. The interface of failure being between the labrum and the glenoid rather than the labrum itself.

Studies have been devised to try and recreate which loading scenarios may lead to a superior labral tear from anterior to posterior (SLAP) lesion. There is disagreement as to whether pure traction on the biceps tendon can create Type II SLAP lesions, with some reports claiming it can [45] and others claiming it cannot [46].

Other cadaveric studies have concentrated on the scenario during throwing with 25% of specimens creating a SLAP Type II lesion, of which 80% are created in late cocking position [47]. Another simulation of the late cocking phase of throwing with further external rotation of the humerus 20% beyond its normal maximal external rotation also created SLAP II lesions [48].

Biomechanical testing of the labrum itself has been performed with compressive testing on embalmed specimens [49]. The elastic modulus was found to be greater in the superior part of the labrum compared to the inferior sections and it was also noted that the dominant side of each shoulder pair consistently had a thicker labrum.

The compressive and tensile properties of the labrum have also been studied in fresh frozen specimens [50,51]. These studies demonstrated that the tensile elastic modulus was 22.8 MPa, which is much closer to articular cartilage (2 MPa to 20 MPa) than to the meniscus (170 MPa). Also, the anterosuperior portion had a lower tensile elastic modulus than the inferior portion. Conversely, the compressive modulus was higher in the anterosuperior portion.

CLINICAL LESIONS

Although there are many hypotheses on the function of the labrum, there is agreement that it aids in stability of the shoulder. The shoulder is the most mobile joint in the body, although is also the most commonly dislocated joint in the body, with an incidence reported at 1% to 1.7% of the adult population [52,53]. The prevalence of recurrent shoulder dislocation under the age of 21 years is 19.7 in 10,000 for males and 5.0 in 10,000 for females [54]. There has been multiple pathological lesions of the capsular-labral complex described associated with shoulder dislocation or recurrent subluxation.

The mostly commonly associated with an anterior dislocation is the Bankart lesion. This was originally described as a detached segment of the anterior-inferior labrum with its attached inferior glenohumeral ligament complex [55]. It has been found in 39% of first time dislocators as seen on magnetic resonance arthrogram [56], 75% as seen on plain magnrtic resonance

imaging [57] and as high as 83% to 97% in the young patient seen at arthroscopy [58,59].

The failure point in a Bankart lesion is at the interface between the labrum and the glenoid rim or, in a bony Bankart lesion, failure is within the glenoid bone itself.

An anterior labrocapsular periosteal sleeve avulsion (ALPSA) lesion differs from a Bankart lesion in that the periosteum of the anterior scapular does not rupture [60]. A variant of the ALPSA affecting the superior part of the anterior labrum has also been described [61]. Neither or these lesions compromise the labrum itself.

The posterior labrocapsular periosteal sleeve avulsion (POLPSA) lesion has been described predominantly in American football players with posterior shoulder instability [62–64], although the original description was in a patient with a locked posterior dislocation [65]. Again, the point of failure is between the labrum and the glenoid.

Kim's lesion appears to be a partial POLPSA with incomplete tearing between the posterioinferior labrum and the glenoid articular cartilage associated with multi or unidirectional instability [66].

Although all of the above lesions do affect stability of the shoulder, none are a direct injury to the labrum itself. The SLAP lesion is a tear of the labrum itself. The original terminology and classification was by Snyder et al. [67]. This described four lesions:

Type I — fraying and degenerative of the superior labrum, but peripheral labral edge still attached to underlying glenoid

Type II — detachment of the superior labrum and biceps tendon from underlying glenoid. Also has fraying and degeneration as Type I

Type III — bucket handle tear of superior labrum. Remaining labral tissue anchored to glenoid rim

Type IV — extension of displaced bucket handle tear into biceps tendon

Lesions have also been described similar to a Type II SLAP, but with associated detachment of articular cartilage and exposure of underlying cartilage [68].

Maffet et al. [69] extended the classification to add a further three types:

Type V — a Bankart lesion that continues superiorly to include separation of the biceps tendon

Type VI — biceps separation with an unstable flap tear of the labrum

Type VII — superior labral biceps tendon separation that extends anteriorly beneath the middle GHL

In 1998, Morgan et al. [70] sub-divided Type II lesion into three groups depending on the anatomical location of the lesion, anterior, posterior and combined.

More recently, Nord and Ryu have added a further three types of lesions to the classification [71]:

Type VIII — a SLAP extension along the posterior glenoid labrum as far as the 6 o'clock position

Type IX — a pan-labral SLAP injury extending the entire circumference of the glenoid labrum

Type X — a superior labral tear associated with a posterior-inferior labral tear

The classifications for SLAP lesions are arbitrary and can really be divided into three broad groups:

1. Fraying/degeneration of the labrum (Type I)
2. Detachment between the labrum and the glenoid (Type II, V, VII, VIII, IX and X)
3. Mid-substance tears (Type III and IV)

Type VI appears to be a combination of a detachment and mid-substance tear.

Although Snyder et al. [67] first coined the term SLAP lesion; the original description of injuries affecting the anterosuperior region of the labrum was by Andrews et al. [72]. They considered that injury to this part of the shoulder was associated with throwing and carried out further analysis by electrical stimulation of biceps tendon *in vivo*. This demonstrated the raising of the superior portion of the labrum off the glenoid and compression of the humeral head into the glenoid. They also used three-dimensional high speed cinematography to show that external rotation during throwing reached up to 160°.

The motion of throwing was originally described by four stages: wind up, cocking, acceleration with ball release and follow through [73]. This has now been extended to six phases [74]:

1. Wind-up — shoulder in slight internal rotation with minimal shoulder abduction
2. Early cocking — the arm gets abducted to 90°
3. Late cocking — relates to planting of the striding leg. Abduction and external rotation of the humerus, reaching up to 180° maximal external rotation
4. Acceleration — the humerus is internal rotated to the object release point at 90°
5. Deceleration — begins once the object is released with violent eccentric contraction of all muscles. Large compressive forces on glenohumeral joint (1000 N) with posterior (400 N) and inferior (300 N) shear forces.
6. Follow through — the body moves forward with the arm until motion stops

Andrews et al. [72] suggested that the high rate of humeral internal rotation, during the start of the deceleration phase, places large forces on the section of biceps tendon between the bicipital groove and its glenoid insertion. The biceps tendon also being important in the acceleration and follow through phases of throwing by the compressive force at the glenohumeral joint giving stability and stress protecting the humerus. Certainly electromyography studies demonstrate that biceps tendon's peak activity is during eccentric contraction in the deceleration stage immediately after ball release or follow through and appears to be relatively inactive during the acceleration phase of throwing [72,75].

Snyder et al. [67] also acknowledged that throwing injuries were a mechanism of creation of SLAP lesions but, in their series, only 8% were caused by this mechanism. Forty-eight percent were caused by a compression injury (a fall on the outstretched hand), 22% resulted from pure traction injuries and 22% were insidious in nature. They considered that the predominant cause of SLAP lesions was a combination of a compression force on superior joint

surface and proximal subluxation force on humeral head. The injury to the labrum was caused by the pinching mechanism between the humeral head and the glenoid. They further postulated that if the proximally directed force continued, a traction tear of the rotator cuff may develop. Rotator cuff tears are associated with SLAP lesions in 31% to 40% of cases [70,76], whereas, external rotation of the arm on impact may lead to anterior propagation of the tear and anterior instability. Further abduction of the arm could cause Type III and Type IV lesions, helped by the strong reflex contraction of biceps [67].

Jobe [77] described five structures at risk with 'posterior superior glenoid impingement'. These are the greater tuberosity, the rotator cuff, the superior labrum, the inferior glenohumeral ligament and associated labrum in tension (Bankart), and the superior glenoid bone. These injuries were proposed to be caused by impingement between the humeral head and the glenoid, although this time when the position of the arm is extreme abduction with external rotation or pure extreme abduction. The superior labrum if injured resulted in a SLAP lesion. Any instability of the anterioinferior capsule causes anterior translation of the humeral head and further impingement.

Burkhart and Morgan [78] proposed the 'peel-back' mechanism of the initiation of Type II Slap lesions. This suggests that a torsional force is applied to the posterior labrum through the biceps insertion in the externally rotated abducted position seen in late cocking. In this position, the biceps tendon has a more vertical and posterior angle, resulting in twisting the base of the biceps tendon. This is often associated with contracture of the posterior and inferior capsule. This can either be a one-off acute injury, such as a baseball base-runner who slides head first into base, or a repetitive injury in the throwing athlete. The peel-back test during arthroscopy recreates this externally rotated and abducted position to determine whether the injured and will peel back over the glenoid [78]. In the cadaver, this sign was demonstrated only when the biceps anchor and the anterior labrum were detached to the 1 o'clock position. None demonstrated the sign with just the biceps anchor detached on its own. Repair in cadaveric models with a single suture at the 12.30 o'clock position eliminated peel-back test in all specimens [79].

Certainly, the position of the arm will influence the force applied to the shoulder by the biceps tendon and appears to acts as a posterior stabilizer in external rotation and an anterior stabilizer in internal rotation [80].

Therefore, more than one mechanism for the creation of SLAP lesions has been proposed. The incidence of mechanisms involved reported by different surgeons is likely to reflect their caseloads, so that a sports surgeon may report almost all of his cases being related to throwing for example. It may be that the three broad groups of SLAP lesions described previously share a mechanism of injury within each group but differ between groups and therefore should not be thought of under the same heading. The nomenclature of a tear should be limited to those affecting the mid-substance, as opposed to detachment.

Also, the anatomical variations that have previously been discussed may play a role in the susceptibility of a patient to SLAP tears. Certainly, a Buford complex has been noted to increase the rate of SLAP injuries [81,82]. From 235 arthroscopic assessments, six patients were found to have Buford complexes, of which five (83%) had an associated SLAP lesion (predominantly Type II).

SLAP tears in general have been associated with shoulder dislocations seen on MR arthrogram in 14% of patients (majority Type II) [80] and at arthroscopy in 7% to 20% of patients [58,59,83,84]. When seen at arthroscopy, the majority (up to 81%) of SLAP lesions are Type II [59,67,69,76,85,86]. However, other reports have described Type I as the most common lesion (74%) [87], especially in atraumatic dislocations [83]. Only Type I and II SLAP lesions were found in first time dislocators, whereas all types including the more severe Type III and IV were found in recurrent dislocators [59].

When present, the frequency of the type of SLAP lesion found varied in the literature. Amalgamating results from multiple studies would give a combined incidence of 36% Type I, 53% Type II, 6% Type III and 5% Type IV [76,86,87]. However, there appears to be a difference in reporting of Type I lesions between studies and this poor inter-observer and intra-observer variability of the Snyder SLAP classification [67] has been noted in the literature [88].

SUMMARY

In summary, from both embryological and anatomical studies, the literature shows that the anterosuperior region of the labrum is different to the remainder of its circumference. The histology suggests that it is essentially a structure designed to resist circumferential tensile loading. The superior region of the labrum appears to the most prone to intra-substance tears. Multiple theories for the mechanism of tearing have been proposed, although essentially the two schools of thought are that the labrum is either pinched and compressed with internal impingement or the labrum is pulled directly or twisted off by the biceps anchor. Therefore, the mechanism of failure may be by compression, direct tension or torsional tension. These advances in the knowledge of this important structure are essential to the understanding of the pathology and treatment of common lesions seen by the shoulder surgeon.

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