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Current Concepts With Video Illustrations

Arthroscopic Anatomy, Variants, and Pathologic Findings in Shoulder Instability

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Abstract: Shoulder instability is a common diagnosis that often requires surgical treatment. A detailed knowledge of the shoulder anatomy and its stabilizing structures is of utmost importance for successful treatment of shoulder instabilities. Identifying anatomic variants (e.g., sublabral hole, meniscoid labrum, cordlike middle glenohumeral ligament, and Buford complex) and distinguishing them from pathologic findings may be especially difficult, as shown by the high interobserver variability. Over the last decade, basic research and arthroscopic surgery have improved our understanding of the shoulder anatomy and pathology. In the context of shoulder instability, injuries of the glenoid (bony Bankart), injuries of the glenoid labrum superiorly (SLAP) or anteroinferiorly (e.g., Bankart, anterior labroligamentous periosteal sleeve avulsion, and Perthes), capsular lesions (humeral avulsion of the glenohumeral ligament), accompanying cartilage lesions (Hill-Sachs, glenolabral articular disruption), and rotator interval and pulley lesions, as well as signs of dynamic instability impingement (posterior-superior impingement, anterior-superior impingement) can be exactly diagnosed (magnetic resonance imaging with intra-articular gadolinium, arthroscopy) and treated (arthroscopy). Therefore the purpose of this article is to review the current literature concerning shoulder anatomy/pathology related to shoulder stability/instability to improve clinical diagnosis and surgical treatment of our patients.

The combination of a large range of motion and insufficient bony stabilization makes the glenohumeral joint susceptible to instability and dislocation.¹ To ensure stability, the shoulder is stabilized by both static and dynamic mechanisms.^{2,3} The static

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mechanisms include the bony configurations of the glenoid and the humerus, the glenoid labrum, the joint capsule, and the glenohumeral ligaments. The dynamic mechanisms include the muscles of the rotator cuff and, to a lesser degree, the long head of the biceps and the deltoid muscle.

Not surprisingly, shoulder dislocation is especially common among young, active people, who show high redislocation rates after an initial traumatic dislocation.^{1,4} Therapeutically, arthroscopic shoulder stabilization is the treatment of choice in cases of recurrent instability.⁵⁻⁸ The outcome is usually excellent when performed by experienced surgeons, and additional procedures may be performed to address concomitant pathologies such as a SLAP lesion or a wide rotator interval.^{9,10} For optimal outcome, the surgeon must be well aware of the normal anatomy, the various normal anatomic variants (e.g., sublabral hole, Buford complex, and meniscoid labrum), and the various pathologic presentations. This distinction is especially chal-

Note: To access the video accompanying this report, visit the October issue of *Arthroscopy* at www.arthroscopyjournal.org.

TABLE 1. Classification of Different Glenoid Types According to Anetzberger and Putz¹³

Glenoid Type	Characteristics	Incidence
Ia	Teardrop shape with a small notch in its anterior-superior part	59%
Ib	Teardrop shape without a notch	29%
II	Oval shape, in which the cranial and caudal diameters are roughly the same	12%

lenging, as shown by a recent study that highlighted that the inter-rater reliability for the exact description of anatomic structures such as the inferior glenohumeral ligament (IGHL) or the bony glenoid size is below 40% during shoulder arthroscopy.¹¹ Therefore the aim of this review is to improve the understanding of the normal anatomy of the glenohumeral joint, including its anatomic variants and pathologic lesions, to enhance the outcome of arthroscopic shoulder stabilization.

BONY CONFIGURATION

Normal Anatomy and Variants

Compared with other joints (e.g., hip), the bony stabilization of the glenohumeral joint is insufficient.¹² There is a large mismatch between the size of the glenoid fossa (6 to 7 cm²) and the humeral head (20 to 24 cm²). According to Anetzberger and Putz,¹³ the glenoid cavity can have a teardrop shape (also called pear shape) (88%) or an oval shape (where the cranial and caudal diameters of the glenoid are similar) (12%) (Table 1). The functional relevance of these different shapes is not fully understood. Most likely, the anchorage of the capsulolabral complex in the anterior-superior area is influenced by the bony con-

figuration.¹⁴ In the center of the glenoid, the bare spot can be found. This area, which is a physiologic thinning of the central articular cartilage, can be found reproducibly and should not be confused with a cartilage defect (Fig 1A).¹⁵ In contrast, the humeral head cartilage is thickest in the center of the humeral head. Similar to the bare spot, an area with no cartilage physiologically (bare area) can be found posterolaterally on the humeral head, adjacent to the rotator cuff insertion, as a transitional zone to the posterior humeral cartilage (Fig 2A). The glenoid version, as well as humeral torsion, also influences glenohumeral stability.^{16,17} Normally, the humeral inclination is about 130° to 150°, and the humeral retroversion is about 30° to 45°. Meanwhile, the glenoid version is about 2° to 5° of retroversion.

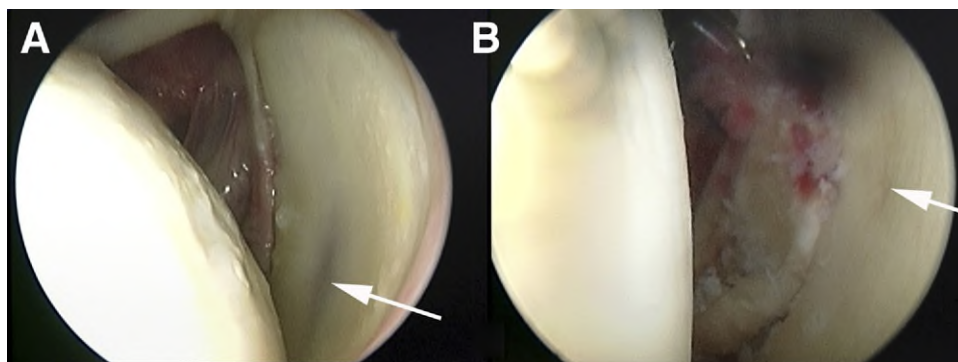
Pathologic Anatomy

Anterior-inferior bone loss of the glenoid, as found in acute or chronic bony Bankart lesions, is an important contributor to shoulder instability.^{12,18} The physiologic glenoid configurations must be distinguished from the inverted pear-shape glenoid, in which the shoulder is significantly destabilized in the anterior direction.¹⁹ Furthermore, the bare area must be differentiated from a Hill-Sachs defect, which is usually more prominent proximally (Fig 2B).²⁰

Clinical Relevance and Tips

Untreated glenoid bone loss is an important and often overlooked factor leading to recurrent shoulder instability after arthroscopic repair.¹⁸ The amount of glenoid bone loss can be estimated preoperatively by use of computed tomography scans or intraoperatively during arthroscopy by use of the bare spot as a reference point (Fig 1B).^{15,21} If a large Hill-Sachs defect is found, dynamic investigation (external rotation/abduction) should be performed to determine whether

FIGURE 1. Arthroscopic view through posterior portal of left shoulder. (A) Normal articulating surfaces with a central bare spot (arrow) in the center of the glenoid. The cartilage covering of the bare spot is extremely thin. (B) Glenoid with a bony Bankart lesion. The bare spot (arrow) can be used to estimate the size of the bony defect.



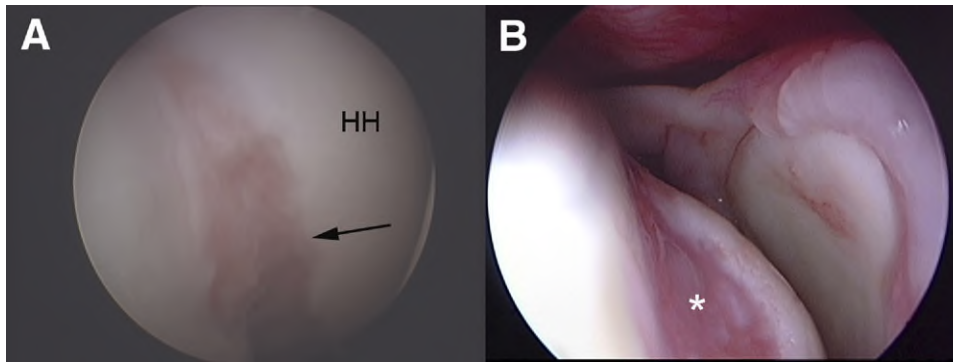


FIGURE 2. Arthroscopic view through posterior portal of left shoulder. (A) The physiologic “bare area” (arrow) is shown next to the posterior insertion of the rotator cuff. (B) In contrast, a huge Hill-Sachs lesion (asterisk) is displayed. One must be sure to test that the defect does not engage with the anterior glenoid during external rotation/abduction. (HH, humeral head.)

the Hill-Sachs lesion engages at the anterior glenoid. An engaging Hill-Sachs lesion can cause recurrent dislocations and should be separately treated.²² In rare cases excessive variations of the glenoid version can also be responsible for recurrent shoulder instability.¹⁶

GLENOID LABRUM

Normal Anatomy

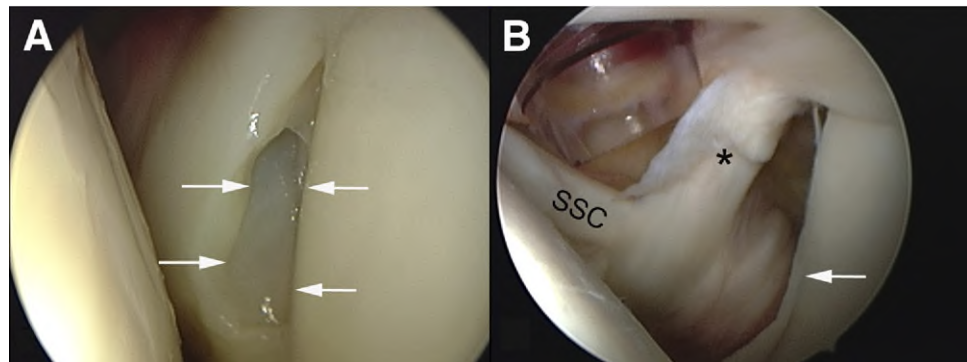
The glenoid labrum circularly covers the glenoid cavity and forms a functional unit with the capsule, glenohumeral ligaments, long head of the biceps, and long head of the triceps.²³ The labrum consists mostly of circular and partly of radially aligned collagenous fibers and a small fibrous cartilage insertion zone at the scapular neck.²³ From a functional perspective, the labrum serves several different functions. The bony contact size is increased by about one-third, and the congruity of the joint surfaces is increased. Thereby, the stress distribution is positively influenced and stability is increased.³ Moreover, the labrum contributes to the ligamentous stability by acting like a “tension brake” through the firm connection to the scapula and the interaction with the capsule and the glenohumeral ligaments.²³ The vascular

supply to the labrum is provided by branches from the suprascapular artery, the anterior humeral circumflex artery, and the posterior humeral circumflex artery.²⁴ The blood supply is minimal in the superior and anterosuperior parts, which may contribute to the limited regeneration potential in these regions.

Normal Anatomic Variants

The glenoid labrum often exists with normal variations, such as small recesses or a meniscoid (like a small meniscus) appearance, in the superior and anterior-superior regions. Anterosuperiorly, the sublbral hole may be found (Fig 3A). It is defined by the complete separation of the labrum from the glenoid in this region. The incidence of the sublbral hole varies between 12% and 18.5%.^{25,26} Rarely, the Buford complex, in which the anterior-superior labrum is absent and replaced by a cordlike middle glenohumeral ligament (MGHL), can be encountered and mistaken for a separation of the anterior labrum (Fig 3B). The incidence of the Buford complex varies between 1.5% and 6%.^{25,27,28} In shoulders with a Buford complex, the superior glenohumeral ligament (SGHL) may be absent, whereas the IGHL is usually well developed.

FIGURE 3. Arthroscopic view through posterior portal of left shoulder. (A) A sublbral hole is shown, in which the labrum is detached from the anterior-superior glenoid (arrows). (B) In the displayed Buford complex, the anterior-superior labrum is completely missing. Instead, a cordlike MGHL is present (asterisk). The beginning of the anterior labrum is marked with an arrow. (SSC, subscapularis.)



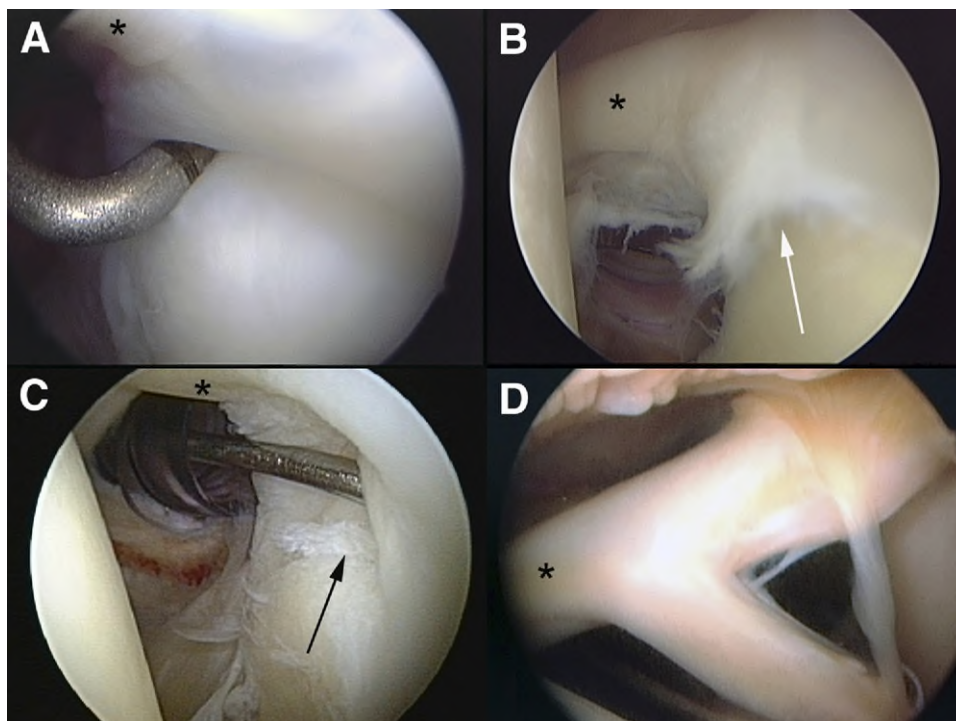


FIGURE 4. Arthroscopic view through posterior portal of left shoulder. The long head of the biceps tendon is marked by asterisks. (A) The sublabral recess is seen superiorly. No instability is noted when probed with a hook. (B) Degenerative fraying of the biceps anchor (arrow) as seen in type I SLAP lesions. (C) Typical type II SLAP lesion with instability of the biceps anchor, fully exposing the superior glenoid (arrow). (D) Example of a type IV SLAP lesion. The rupture extends into the long head of the biceps tendon and is combined with a bucket-handle tear of the labrum posteriorly.

In shoulders with significant SLAP lesions (described later), the incidence of a sublabral hole increases to 40% and the incidence of a Buford complex increases to 20%. Whether these anatomic variants place increased load on the biceps anchor is not known.

Pathologic Anatomy

SLAP lesion: Superiorly, small recesses and labral detachments must be differentiated from pathologic

lesions and separations of the biceps anchor (Fig 4, Table 2).^{25,27,29,30}

Anterior-Superior Impingement: Labral fraying in the anterior-superior part may also be caused by anterior-superior impingement, a dynamic impingement of the anterior-superior capsule and subscapularis with the coracoid process. Radas and Pieper³¹ found no relation between the coracoid impingement and the size of the subcoracoid space in their

TABLE 2. Classification of SLAP Lesions I-IV* With Modification of V-VII**

SLAP Type	Biceps-Labral Complex	Comments
I	Partial tear/fraying of labrum with intact biceps anchor	Incidental finding; predominantly in young people involved in overhead activities
II	Complete tear of biceps-labral complex with instability of biceps anchor	Most common type; associated with acute traction, repetitive overhead motion, and microinstability
III	Bucket-handle tear with intact biceps anchor	Associated with fall on outstretched arm
IV	Bucket-handle tear including biceps anchor	More severe than type III; associated with fall on outstretched arm
V	SLAP II lesion + anteroinferior Bankart lesion	Mostly associated with anterior shoulder dislocation
VI	SLAP II lesion + anterior or posterior unstable tear of labrum	Probably represents type IV or less likely type III with tear of bucket-handle component
VII	SLAP II lesion + long anterior tear of labrum to MGHL	Association with acute trauma with anterior dislocation

NOTE. Types I through IV describe isolated lesions of the biceps anchor, whereas types V through VII are combined lesions.

*Based on data from Snyder et al.²⁹

**Based on data from Maffet et al.⁵³

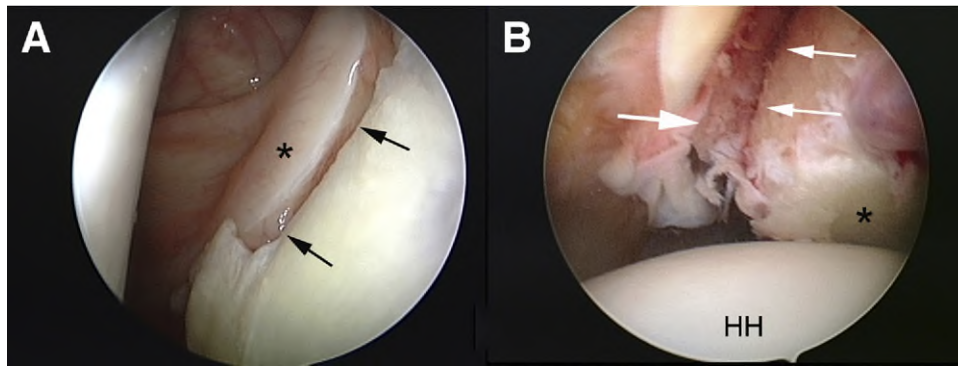


FIGURE 5. Arthroscopic view of left shoulder. (A) Classical Bankart lesion as seen through a posterior portal. The arrows mark the separation of the anterior-inferior labrum (asterisk) from the glenoid. (B) Scarring of the anterior-inferior labrum to the glenoid (ALPSA lesion), as seen with the arthroscope from the superior portal. The medial glenoid neck is marked by the 2 small arrows, the anterior-inferior capsulolabral complex by the large arrow, and the glenoid surface by an asterisk. It should be noted how far medially one can see, as compared with from the standard posterior portal.

anatomic study on 124 human cadaveric shoulders. They concluded that most cases of coracoid impingement are not caused by aberrant bony variations but are the result of anterior instability leading to a functional narrowing of the coracohumeral distance and subsequent fraying of the anterior-superior structures.

Bankart Lesion: Labral detachments inferior to the equator are most likely pathologic and can often be found in cases of traumatic shoulder dislocation or even glenohumeral subluxations.³² The most common labral lesion is the classic Bankart lesion, in which the anterior-inferior labrum is separated from the glenoid (Fig 5A).

Anterior Labroligamentous Periosteal Sleeve Avulsion: In the case of an anterior labroligamentous periosteal sleeve avulsion (ALPSA) lesion, the torn labrum and IGHL are scarred to the medial glenoid neck (Fig 5B). The incidence of ALPSA lesions increases in patients with multiple dislocations.³³

TABLE 3. Arthroscopic Classification of Posterior Labral Tears According to Kim et al.³⁵

Type of Posterior Labral Tear	Characteristics	Incidence
I	Incomplete detachment	36%
II	Marginal crack or incomplete and concealed avulsion of posterior-inferior labrum	39%
III	Chondrolabral erosion	19%
IV	Flap tear	6%

NOTE. In 81% of cases, the labral tear is isolated without a chondral lesion.

Perthes Lesion: Much more seldom, Perthes lesions—subperiosteal rupture of the anterior labrum and the IGHL without scar formation and dislocation to the medial glenoid neck—are found.

Glenolabral Articular Disruption Lesions: Sometimes cartilage lesions leading to pain without any signs of instability, because the damaged anterior labrum is not significantly displaced, can be observed.

Posterior-Inferior Labral Lesions: Posterior-inferior labral lesions are associated with posterior and often multidirectional instability. In an arthroscopic study

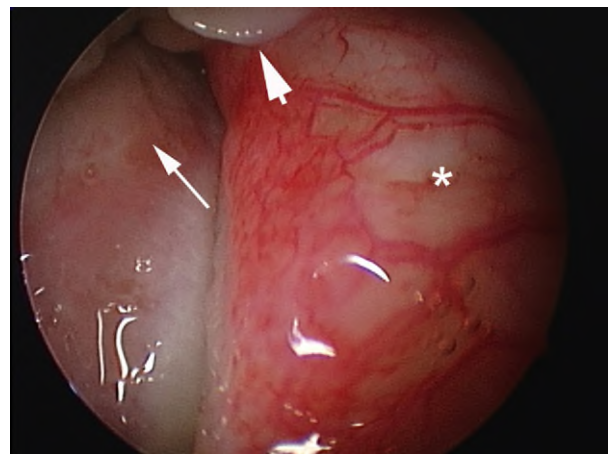


FIGURE 6. Near arthroscopic view through the posterior portal of a left shoulder showing the superior-posterior labrum. The typical signs of a posterior impingement lesion can be seen: hypervascularity of the labrum (asterisk), a small cartilage defect at the insertion of the supraspinatus tendon on the humeral head (small arrow), and partial articular-sided fraying of the supraspinatus tendon (large arrow). The dynamic exploration is shown in Video 1 (available at www.arthroscopyjournal.org).

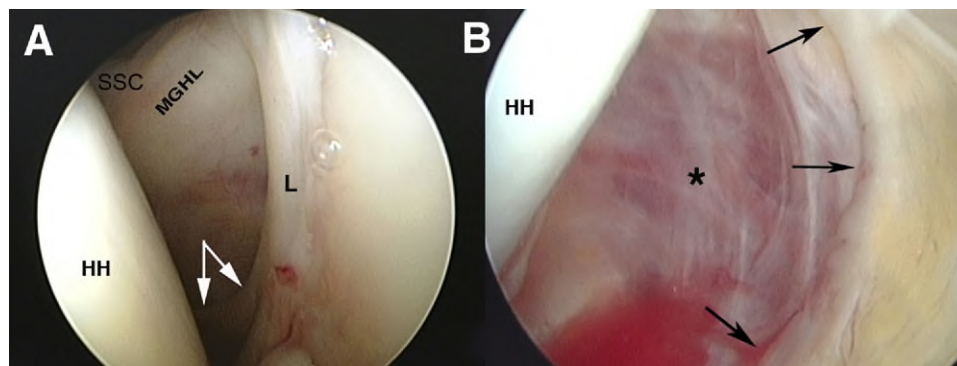


FIGURE 7. Arthroscopic view through posterior portal of left shoulder. (A) The MGHL can be observed crossing the subscapularis tendon (SSC) at an angle of approximately 45°. The anterior band of the IGHL is marked by the arrows. (L, labrum; HH, humeral head.) (B) A rare case of a humeral avulsion of the glenohumeral ligaments (HAGL lesion) is shown. The arrows mark the ruptured and retracted capsule exposing the muscle fibers laterally. (HH, humeral head.)

on 100 patients with posterior instability, Bradley et al.³⁴ found pathologies such as lax capsules without any labral changes (43%), incomplete labral tears (27%), or complete detachments of the posterior labrum (30%). Of the 57% of cases with labral tears, 40% also had combined laxity of the capsule. The posterior-inferior labral tears may also be classified arthroscopically into 4 different types according to Kim et al.³⁵ (Table 3).

Posterior-Superior Impingement: Fraying of the posterior-superior labrum, even with cartilage lesions, can sometimes be observed in association with dynamic instability impingement (posterior-superior impingement [Fig 6; dynamic examination in Video 1, available at www.arthroscopyjournal.org]). The exact etiology of this internal impingement syndrome is cause for much debate. Current hypotheses include anterior shoulder instability or microinstability, contracture of the posterior capsule, reduced humeral retroversion, and scapular dyskinesis.

Clinical Relevance and Tips

Anatomic refixation of pathologic labral separations is the mainstay of therapy in traumatic shoulder instability. A Buford complex must not be mistaken for an anterior labral lesion. A misdiagnosis with fixation of the thickened glenohumeral ligament significantly reduces external rotation of the shoulder.

The examination of the anatomic structures with a probe during arthroscopy greatly improves the differentiation between anatomic variants and pathologic separations. In type II SLAP lesions, the biceps anchor is unstable and can be widely lifted up or pulled into the joint (dynamic investigation in Video 2, available

at www.arthroscopyjournal.org). It should be noted that ALPSA lesions or lesions of the posteroinferior glenoid can only be fully assessed when viewed from an anterior portal (Fig 5B).

CAPSULE AND GLENOHUMERAL LIGAMENTS

Normal Anatomy

The capsule consists of a complex system of circularly and radially arranged collagen fibers and is strengthened by multiple reinforcements (coracohumeral ligament, SGHL, MGHL, IGHL, and circumferentially inserting rotator cuff tendons).³⁶ The space between the supraspinatus and subscapularis muscle is called the rotator interval (RI).³⁷

The coracohumeral ligament radiates laterally from the coracoid process and inserts at the greater and lesser tuberosities.³⁸ By the transverse humeral ligament, the roof of the bicipital sulcus is reinforced.

TABLE 4. Anatomic Variations of Glenohumeral Ligaments*

Type	Appearance of Glenohumeral Ligaments	Incidence
I	SGHL, MGHL, and IGHL can be separately identified	66%
II	IGHL and MGHL are fused	7%
III	MGHL has a strong cordlike shape	19%
IV	All ligaments missing completely	8%

NOTE. In two-thirds of shoulders, all 3 ligaments (SGHL, MGHL, and IGHL) can be separately identified.

*Based on data from Morgan et al.⁴¹

TABLE 5. Characteristics of Glenohumeral Ligaments in All Shoulders Compared With Shoulders With Anatomic Variants (Sublabral Hole and Buford Complex), According to Ilahi et al.²⁵

	SGHL		MGHL				IGHL	
	Present	Absent	Thick Cord	Thin Sheet	Absent	Bifid	Well Developed	Poorly Developed
All shoulders	40.7%	59.3%	21.3%	70.4%	7.4%	0.9%	79.6%	20.4%
Sublabral hole	50%	50%	65%	35%	—	—	75%	25%
Buford complex	28.6%	71.4%	100%	—	—	—	85.7%	14.3%

The SGHL bulges out at the inner side of the joint capsule, running from the supraglenoid tubercle to the lesser tuberosity. The coracohumeral ligament and the SGHL are the main constituents of the biceps reflection sling, the so-called pulley system, which will be described later.³⁹ The SGHL stabilizes the shoulder against posterior and inferior instability and prevents dislocation of the biceps tendon from its sulcus.

The MGHL is variably developed and not clearly discernible in every case. It radiates from the superior glenoid tubercle to the inferior parts of the lesser tuberosity and functions as an important anterior stabilizer during abduction/external rotation.³⁶

The IGHL consists of a strong anterior band and a posterior band, with the axillary recess residing between them (Fig 7A). Both parts of the ligament stabilize the humeral head in the vulnerable abduction

and external rotation movements. Because of its configuration, this complex is also called the hammock.³⁶

Normal Anatomic Variants

In the anterior-superior part, a subscapular recess (subscapular bursa, Weitbrecht foramen), which is an opening of the anterior capsule into the subtendinous bursa of the subscapularis muscle and is mostly located between the SGHL, the subscapularis tendon, and the MGHL, can be found in about 80% of cases.²⁴ According to DePalma et al.,⁴⁰ 6 different variants of this recess can be found. The variations of the glenohumeral ligaments according to Morgan et al.⁴¹ are shown in Table 4. Sometimes a cordlike variant of the MGHL can be found. The MGHL is often missing or indistinct in patients with anterior-inferior shoulder instability, whereas the presence of a cordlike MGHL is regarded as a protective factor against shoulder instability.²⁵ In the anterior-superior part, some fibers of the IGHL run upward and connect to the fibers of the SGHL, thereby forming a “connecting band.” This band can be differently developed and may replace the labrum in this part.²³ In shoulders with a sublabral hole or Buford complex, the characteristics of the glenohumeral ligaments are somewhat changed, as shown in Table 5.²⁵

Pathologic Anatomy

It is often difficult to quantify a widened RI. The tension of the RI is influenced by the coracohumeral ligament and SGHL and the capsule in between. Arthroscopically, the RI may be noted to be wide when the coracohumeral ligament and SGHL do not sufficiently tension during external rotation and the capsule in between bulges out.

A rare case of recurrent instability can also occur as a result of the humeral avulsion of the glenohumeral ligaments (HAGL lesion), which can be easily over-

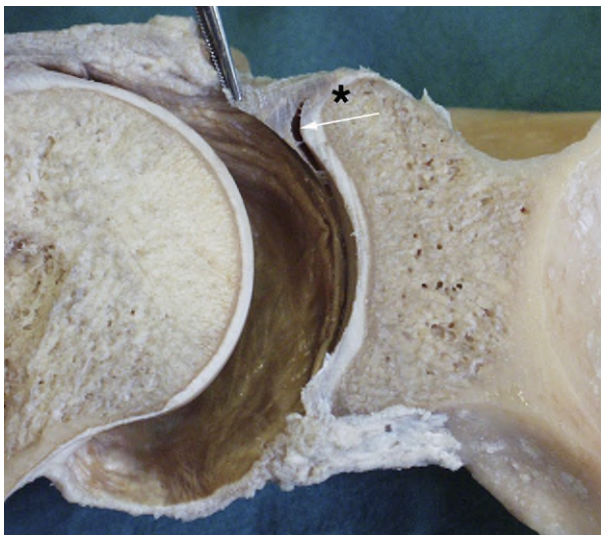
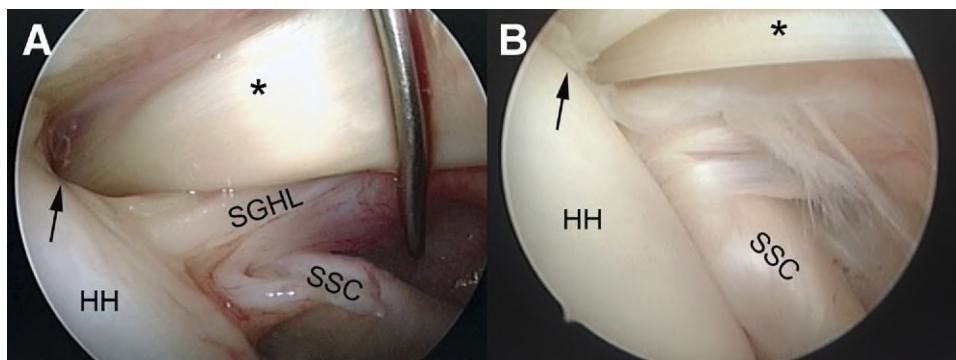


FIGURE 8. Macroscopic specimen sectioned through the supraglenoid tubercle (asterisk), showing the insertion of the long head of the biceps. The sublabral recess with meniscoid labrum (arrow) should be noted.

FIGURE 9. Arthroscopic view through posterior portal of left shoulder. (A) Pulley sling with intact SGHL guiding long head of biceps (asterisk) into intertubercular sulcus (arrow). (B) Lesion of medial pulley sling with fraying of SGHL, leading to medial subluxation of biceps tendon. The asterisk shows the biceps tendon, and the arrow shows the intertubercular sulcus. (SSC, subscapularis muscle; HH, humeral head.)



looked if not specifically evaluated during shoulder arthroscopy (Fig 7B).⁴²

Clinical Relevance and Tips

A wide RI can play a role in inferior, anterior, and posterior instability.⁴³ In experimental and clinical studies, it has been shown that a wide RI may lead to inferior shoulder instability.^{36,44}

For a complete visualization of the glenohumeral ligaments, multiple portals should be used during arthroscopy. In this context, the humeral avulsion should be inspected during internal and external rotation either from posterior or, even better, after the arthroscope has been placed in an anterior portal.

A further indication of laxity of the capsule and ligaments is the so-called positive drive-through sign, in which the arthroscope may slide without significant resistance through the glenohumeral space from superior into the inferior recesses.

LONG HEAD OF BICEPS

Normal Anatomy

The primary function of the long head of the biceps is the stabilization of the glenohumeral joint when it is

in the abducted and externally rotated position, thereby reducing the stress on the IGHL.⁴⁵ The fibers of the long head of the biceps tendon insert into both the labrum and the supraglenoid tubercle (Fig 8). The biomechanical importance of these insertions was examined by Healey et al.⁴⁶ The sole cutting of the labral fibers reduced the stability of the biceps anchor only insignificantly, whereas the sole cutting of the deep fibers at the supraglenoid tubercle greatly reduced the pullout strength. According to this finding, the biomechanically important fibers insert at the supraglenoid tubercle, whereas the remaining fibers insert variably in the labrum and have mostly a shaping function.⁴⁶

The long head of the biceps is guided by the pulley sling into the intertubercular sulcus (Fig 9A). The pulley sling is formed mainly by the SGHL, which forms the medial wall and the bottom (together with fibers from the subscapularis muscle), and the coracohumeral ligament, which forms the roof and lateral wall (together with fibers from the supraspinatus muscle).^{39,47}

Normal Anatomic Variants

According to a study of 100 cadaveric shoulders by Vangness et al.,⁴⁸ 4 different variants of the origin of

TABLE 6. Classification of Origin of Long Head of Biceps Tendon From Glenoid and Labrum*

Type	Biceps Origin	Incidence
I	The labral attachment is entirely posterior, with no contact with the anterior labrum	22%
II	Most of the labral contribution is posterior, with a small contribution to the anterior labrum	33%
III	There are equal contributions to both the anterior and posterior parts of the labrum	37%
IV	Most of the labral contribution is anterior, with a small contribution to the posterior labrum	8%

NOTE. Fifty percent of the biceps fibers insert consistently at the supraglenoid tubercle. The remaining fibers insert variably into the labrum, as shown in the table.

*Based on data from Vangness et al.⁴⁸

the long head of the biceps can be found. Forty to sixty percent of the biceps fibers insert constantly at the supraglenoid tubercle. The remaining fibers insert variably into the labrum (Table 6).

Pathologic Anatomy, Clinical Relevance, and Tips

Pathologic lesions of the biceps tendon are described previously in the section on SLAP lesions. Lesions of the pulley sling (Fig 9B) can be a cause of pain as well. Simple visualization of the labrum may not exclude a labral detachment. A complete evaluation of the attachment of the biceps anchor on the supraglenoid tubercle is only possible with a probe. In type II SLAP lesions, additional abduction and external rotation of the arm pull on the biceps anchor and often allow for better visualization of a labral tear.

ROTATOR CUFF

Normal Anatomy

The 4 muscles of the rotator cuff (subscapularis, supraspinatus, infraspinatus, and teres minor) contribute to dynamic shoulder stabilization.^{3,49} The humeral head is centered, and thereby stabilized, by the muscular force of the rotator cuff. A detailed review of the anatomy of the rotator cuff was recently published by DeFranco and Cole.⁵⁰ According to other recent investigations, the deltoid muscle may also have some stabilizing effect on the glenohumeral joint.^{49,51}

Clinical Relevance and Tips

In patients aged over 40 years, rotator cuff tears are a well-known complication of shoulder dislocation and must be included in the evaluation and treatment of shoulder instabilities.⁵²

CONCLUSIONS

Shoulder stability is maintained by the complex interplay between static and dynamic mechanisms, as described previously. A detailed knowledge of the anatomic structures, including their normal anatomic variations and functional relevance, is the prerequisite for adequate diagnosis and therapy. Only shoulder arthroscopy allows for the detailed visualization and the dynamic examination with probes of all relevant structures and pathologic findings.

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REFERENCES

- Owens BD, Duffey ML, Nelson BJ, DeBerardino TM, Taylor DC, Mountcastle SB. The incidence and characteristics of shoulder instability at the United States Military Academy. *Am J Sports Med* 2007;35:1168-1173.
- Abboud JA, Soslowky LJ. Interplay of the static and dynamic restraints in glenohumeral instability. *Clin Orthop Relat Res* 2002;48-57.
- Lippitt S, Matsen F. Mechanisms of glenohumeral joint stability. *Clin Orthop Relat Res* 1993;20-28.
- Brophy RH, Marx RG. The treatment of traumatic anterior instability of the shoulder: Nonoperative and surgical treatment. *Arthroscopy* 2009;25:298-304.
- Imhoff AB, Anshah P, Tischler T, et al. Arthroscopic repair of anterior-inferior glenohumeral instability using a portal at the 5:30-o'clock position: Analysis of the effects of age, fixation method, and concomitant shoulder pathology on surgical outcomes. *Am J Sports Med* 2010;38:1795-1803.
- Castagna A, Markopoulos N, Conti M, Rose GD, Papadakou E, Garofalo R. Arthroscopic Bankart suture-anchor repair: Radiological and clinical outcome at minimum 10 years of follow-up. *Am J Sports Med* 2010;38:2012-2016.
- Bak K, Wiesler ER, Poehling GG, ISAKOS Upper Extremity Committee. Consensus statement on shoulder instability. *Arthroscopy* 2010;26:249-255.
- Seroyer ST, Nho SJ, Provencher MT, Romeo AA. Four-quadrant approach to capsulolabral repair: An arthroscopic road map to the glenoid. *Arthroscopy* 2010;26:555-562.
- Yiannakopoulos CK, Mataragas E, Antonogiannakis E. A comparison of the spectrum of intra-articular lesions in acute and chronic anterior shoulder instability. *Arthroscopy* 2007;23:985-990.
- Boileau P, Richou J, Lisai A, Chuinard C, Bicknell RT. The role of arthroscopy in revision of failed open anterior stabilization of the shoulder. *Arthroscopy* 2009;25:1075-1084.
- Sasyniuk TM, Mohtadi NG, Hollinshead RM, Russell ML, Fick GH. The inter-rater reliability of shoulder arthroscopy. *Arthroscopy* 2007;23:971-977.
- Bushnell BD, Creighton RA, Herring MM. Bony instability of the shoulder. *Arthroscopy* 2008;24:1061-1073.
- Anetzberger H, Putz R. The scapula: Principles of construction and stress. *Acta Anat (Basel)* 1996;156:70-80.
- Shortt CP, Morrison WB, Shah SH, Zoga AC, Carrino JA. Association of glenoid morphology and anterosuperior labral variation. *J Comput Assist Tomogr* 2009;33:584-586.
- Burkhart SS, Debeer JF, Tehrany AM, Parten PM. Quantifying glenoid bone loss arthroscopically in shoulder instability. *Arthroscopy* 2002;18:488-491.
- Churchill RS, Brems JJ, Kotschi H. Glenoid size, inclination, and version: An anatomic study. *J Shoulder Elbow Surg* 2001;10:327-332.
- Brewer BJ, Wubben RC, Carrera GF. Excessive retroversion of the glenoid cavity. A cause of non-traumatic posterior instability of the shoulder. *J Bone Joint Surg Am* 1986;68:724-731.
- Provencher MT, Bhatia S, Ghodadra NS, et al. Recurrent shoulder instability: Current concepts for evaluation and management of glenoid bone loss. *J Bone Joint Surg Am* 2010;92:133-151 (Suppl 2).
- Lo IK, Parten PM, Burkhart SS. The inverted-pear glenoid: An indicator of significant glenoid bone loss. *Arthroscopy* 2004;20:169-174.

20. Saito H, Itoi E, Minagawa H, Yamamoto N, Tuoheti Y, Seki N. Location of the Hill-Sachs lesion in shoulders with recurrent anterior dislocation. *Arch Orthop Trauma Surg* 2009;129:1327-1334.
21. Dettlerline AJ, Provencher MT, Ghodadra N, Bach BR Jr, Romeo AA, Verma NN. A new arthroscopic technique to determine anterior-inferior glenoid bone loss: Validation of the secant chord theory in a cadaveric model. *Arthroscopy* 2009;25:1249-1256.
22. Koo SS, Burkhart SS, Ochoa E. Arthroscopic double-pulley remplissage technique for engaging Hill-Sachs lesions in anterior shoulder instability repairs. *Arthroscopy* 2009;25:1343-1348.
23. Huber WP, Putz RV. Periarticular fiber system of the shoulder joint. *Arthroscopy* 1997;13:680-691.
24. Cooper DE, Arnoczky SP, O'Brien SJ, Warren RF, DiCarlo E, Allen AA. Anatomy, histology, and vascularity of the glenoid labrum. An anatomical study. *J Bone Joint Surg Am* 1992;74:46-52.
25. Ilahi OA, Labbe MR, Coscelluela P. Variants of the anterosuperior glenoid labrum and associated pathology. *Arthroscopy* 2002;18:882-886.
26. Pfahler M, Haraida S, Schulz C, et al. Age-related changes of the glenoid labrum in normal shoulders. *J Shoulder Elbow Surg* 2003;12:40-52.
27. Ide J, Maeda S, Takagi K. Normal variations of the glenohumeral ligament complex: An anatomic study for arthroscopic Bankart repair. *Arthroscopy* 2004;20:164-168.
28. Williams MM, Snyder SJ, Buford D Jr. The Buford complex—the “cord-like” middle glenohumeral ligament and absent anterosuperior labrum complex: A normal anatomic capsulolabral variant. *Arthroscopy* 1994;10:241-247.
29. Snyder SJ, Karzel RP, Del Pizzo W, Ferkel RD, Friedman MJ. SLAP lesions of the shoulder. *Arthroscopy* 1990;6:274-279.
30. Tischer T, Salzmann GM, El-Azab H, Vogt S, Imhoff AB. Incidence of associated injuries with acute acromioclavicular joint dislocations types III through V. *Am J Sports Med* 2009;37:136-139.
31. Radas CB, Pieper HG. The coracoid impingement of the subscapularis tendon: A cadaver study. *J Shoulder Elbow Surg* 2004;13:154-159.
32. Owens BD, Nelson BJ, Duffey ML, et al. Pathoanatomy of first-time, traumatic, anterior glenohumeral subluxation events. *J Bone Joint Surg Am* 2010;92:1605-1611.
33. Spatschil A, Landsiedl F, Anderl W, et al. Posttraumatic anterior-inferior instability of the shoulder: Arthroscopic findings and clinical correlations. *Arch Orthop Trauma Surg* 2006;126:217-222.
34. Bradley JP, Baker CL III, Kline AJ, Armfield DR, Chhabra A. Arthroscopic capsulolabral reconstruction for posterior instability of the shoulder: A prospective study of 100 shoulders. *Am J Sports Med* 2006;34:1061-1071.
35. Kim SH, Kim HK, Sun JI, Park JS, Oh I. Arthroscopic capsulolabroplasty for posteroinferior multidirectional instability of the shoulder. *Am J Sports Med* 2004;32:594-607.
36. Burkart AC, Debski RE. Anatomy and function of the glenohumeral ligaments in anterior shoulder instability. *Clin Orthop Relat Res* 2002;400:32-39.
37. Hunt SA, Kwon YW, Zuckerman JD. The rotator interval: Anatomy, pathology, and strategies for treatment. *J Am Acad Orthop Surg* 2007;15:218-227.
38. Yang HF, Tang KL, Chen W, et al. An anatomic and histologic study of the coracohumeral ligament. *J Shoulder Elbow Surg* 2009;18:305-310.
39. Dierickx C, Ceccarelli E, Conti M, Vanlommel J, Castagna A. Variations of the intra-articular portion of the long head of the biceps tendon: A classification of embryologically explained variations. *J Shoulder Elbow Surg* 2009;18:556-565.
40. DePalma AF, Callery G, Bennett GA. Variational anatomy and degenerative lesions of the shoulder joint. *Instr Course Lect* 1949;6:255-281.
41. Morgan CD, Rames RD, Snyder SJ. Anatomical variants of the glenohumeral ligaments. In: Snyder SJ, ed. *Shoulder arthroscopy*. New York: McGraw-Hill, 1993:33-41.
42. Bui-Mansfield LT, Banks KP, Taylor DC. Humeral avulsion of the glenohumeral ligaments: The HAGL lesion. *Am J Sports Med* 2007;35:1960-1966.
43. Provencher MT, Dewing CB, Bell SJ, et al. An analysis of the rotator interval in patients with anterior, posterior, and multidirectional shoulder instability. *Arthroscopy* 2008;24:921-929.
44. Warner JJ, Deng XH, Warren RF, Torzilli PA. Static capsuloligamentous restraints to superior-inferior translation of the glenohumeral joint. *Am J Sports Med* 1992;20:675-685.
45. Rodosky MW, Harner CD, Fu FH. The role of the long head of the biceps muscle and superior glenoid labrum in anterior stability of the shoulder. *Am J Sports Med* 1994;22:121-130.
46. Healey JH, Barton S, Noble P, Kohl HW III, Ilahi OA. Biomechanical evaluation of the origin of the long head of the biceps tendon. *Arthroscopy* 2001;17:378-382.
47. Werner A, Mueller T, Boehm D, Gohlke F. The stabilizing sling for the long head of the biceps tendon in the rotator cuff interval. A histoanatomic study. *Am J Sports Med* 2000;28:28-31.
48. Vangsness CT Jr, Jorgenson SS, Watson T, Johnson DL. The origin of the long head of the biceps from the scapula and glenoid labrum. An anatomical study of 100 shoulders. *J Bone Joint Surg Br* 1994;76:951-954.
49. Ackland DC, Pandey MG. Lines of action and stabilizing potential of the shoulder musculature. *J Anat* 2009;215:184-197.
50. DeFranco MJ, Cole BJ. Current perspectives on rotator cuff anatomy. *Arthroscopy* 2009;25:305-320.
51. Kido T, Itoi E, Lee SB, Neale PG, An KN. Dynamic stabilizing function of the deltoid muscle in shoulders with anterior instability. *Am J Sports Med* 2003;31:399-403.
52. Porcellini G, Paladini P, Campi F, Paganelli M. Shoulder instability and related rotator cuff tears: Arthroscopic findings and treatment in patients aged 40 to 60 years. *Arthroscopy* 2006;22:270-276.
53. Maffet MW, Gartsman GM, Moseley B. Superior labrum-biceps tendon complex lesions of the shoulder. *Am J Sports Med* 1995;23:93-98.