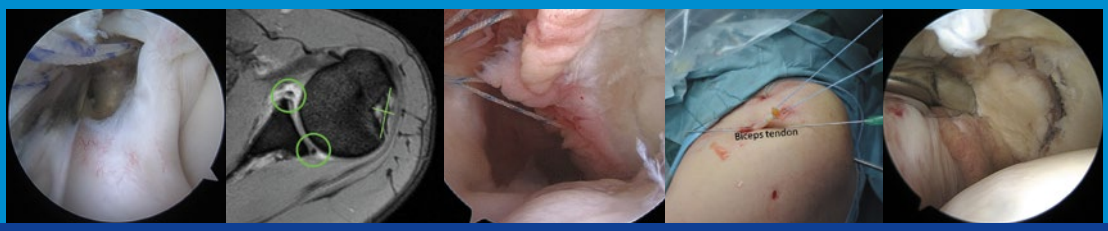
Roman Brzóska · Giuseppe Milano
Pietro S. Randelli · Ladislav Kovačič
Editors



360° Around Shoulder Instability

European Society of Sports Traumatology, Knee Surgery & Arthroscopy
ESSKA

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To Mariangela. Don't look back in anger. There is always a good chance to take. Someday, somehow…

GM

To Krištof, Klemen and Jernej - amazing boys. Being with you is pleasure and happiness. The future is all in front of you. Go ahead with brave and decisive steps. You can realize your dreams.

Ladislav Kovačič

Preface

As a continuation of the tradition, initiated during the first ESA Biennale Meeting in Rome, we share with you another monograph, which is a collection of speeches that were presented during the ESA second meeting. The title of this book is uniform with the title of the last meeting "360 degrees around shoulder instability" which took place on 5–7 October 2017 in Kraków/Bielsko-Biała, Poland.

The book you are holding is a compendium which includes summaries of scientific reports on the treatment of shoulder instability, but also original studies, made available by many experienced and widely recognized shoulder surgeons.

In this book, as the title says, we started a discussion on the diagnosis and treatment of anterior instability in all its aspects. Then we tried to discuss the problems in the treatment of posterior, multidirectional instability and those less common forms of instability whose recognition and effective treatment still pose many problems in daily practice even for the most experienced surgeons. Scientific reports indicate that we still lack clear and transparent guidelines in the treatment of shoulder instability and we are still looking for new solutions to expand the portfolio of modern treatment methods, giving new tools and solutions to shoulder surgeons.

The dynamic development of shoulder surgery enables continuous progress in this field, bringing new methods, especially in arthroscopic techniques. We know that the effectiveness of instability treatment is best judged by time. Many methods, especially at the beginning of the arthroscopic era, did not withstand the test of time.

Enriched by these experiences, we are still looking for better surgical solutions; furthermore our knowledge about indications is also maturing. Learning from experience, we also know more and more about the irreplaceable role of rehabilitation and physiotherapy in the treatment of shoulder instability. Contemporary treatment, especially of posterior and multidirectional instability, practically would not exist without the correct cooperation of orthopedic surgeons, physiotherapists, and sometimes doctors of other specialties.

This book is composed according to the program of the ESA meeting in Krakow/Bielsko-Biała, presenting the problem of shoulder instability in all its aspects. You will find in it explanation of the underlying causes of these pathologies, a multidisciplinary approach facilitating understanding of etiology, diagnostics, and finally treatment methods.

The involvement of scientists representing basic sciences gives a broad perspective to the issues raised and sets out some new directions for cooperation between various fields of medicine and medical science.

We have long stopped treating the problem of instability as a mechanical issue—consisting only in the treatment of organic damage. We have an increasing knowledge of the pathophysiology of these phenomena. We are able to determine the probability of recurrence of instability with much greater precision, combining facts from the interview, constitutional conditions of the patient and his activity. The defining feature of this monograph is numerous algorithms that are useful in making decision when planning the treatment.

Almost all lecturers who participated in the ESA meeting in Krakow/Bielsko-Biała agreed to participate in the creation of this book and share their experiences. We also invited our American, Canadian, and Indian colleagues to share with us their perspective on the current approach to anterior instability.

Thanks to the support of ESSKA management and Springer's professional help, this monograph could be created in such high quality. We hope that readers will receive answers to many bothering questions and help in making daily decisions in effective treatment of patients suffering from various forms of shoulder instability.

Bielsko-Biała, Poland — Roman Brzóska
Brescia, Italy — Giuseppe Milano
Milan, Italy — Pietro S. Randelli
Ljubljana, Slovenia — Ladislav Kovačič

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Part I

Anterior Shoulder Instability

1 Historical Outline of Anterior Shoulder Instability Treatment

Radovan Mihelic and Tomislav Prpic

The history of shoulder instability goes far back into the ancient era. It was on Egyptian papyrus dated some 2000 years. BC that a shoulder reduction was drawn (Fig. 1.1). This is the first known document on the subject. Next description comes from Hippocrates 400 BC showing a shoulder traction to reduce a dislocation (Fig. 1.2). This same method was later reintroduced by Kocher in 1870 [1]. It was even documented in *Hippocratic Corpus*, and some doctors use it still today [2].

The middle age has a lack of medical texts and methods, the Inquisition has occupied the attention of the unbelievers, and it was not popular to mess around with anatomy. Therefore, the work of Jean-François Malgaigne in 1855 is important; he described a bony lesion of the humeral head after anterior dislocation, which we now call the Hill-Sachs lesion [3]. Harold A. Hill and Maurice D. Sachs were two radiologists who have described a humeral defect as a result of repeated dislocations in 1940 [4].

The nineteenth century brings us some interesting papers explaining surgical methods to address the anterior instability. In 1819 Weinhold has published about a surgical reduction to the dislocated shoulder [5, 6]. The Czech surgeon named Eduard Albert made the first fusion of the shoulder in 1878 after serious condition due to recurrent instability, and he named it the arthrodesis [7]. Today it might seem a little exaggerated, but in 1882, Cramer in Germany used a humeral head resection for the chronically unstable shoulder [8].

Auguste Broca and Henri Albert Charles Antoine Hartmann in a paper published in 1890 in French have explained the anatomy of anterior capsular complex in unstable shoulder. They emphasized the role of the glenoid labrum for the joint stability [9].

At the turn to the twentieth century, two important papers appeared thus starting the modern era of shoulder treatment. The first was published in Germany by B. Perthes in 1906 about the surgery of the unstable shoulder [10]. In his paper he has explained the type of anterior capsular detachment that we today call "the Perthes lesion." The second, a paper that we consider a historic turn in shoulder understanding, was published in 1923 by Arthur Sidney Blundell Bankart [11]. He has explained that the detachment of the anterior capsule causes the anterior instability and it is necessary to reattach it in order to stabilize the joint. This short publication on two pages with no images has caused an impact so important that we consider this paper as a basic science in shoulder instability.

Almost at the same time, Vittorio Putti and Harry Platt (1923–1925) have published their surgical method for capsulolabral plication

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Fig. 1.1 Egyptian papyrus showing a shoulder reduction. (Reproduced from Davies, N. de Garis. Two Ramesside Tombs at Thebes. Robb de Peyster Tytus Memorial Series, Vol 5 1927. New York the Metropolitan museum of Art)

Fig. 1.2 "Inquisition type" of reduction on the Hippocratic device. (An Illustrated history by Ira M Rutkow pub 1993. A woodcut probably after a drawing by Francesco Salviati)

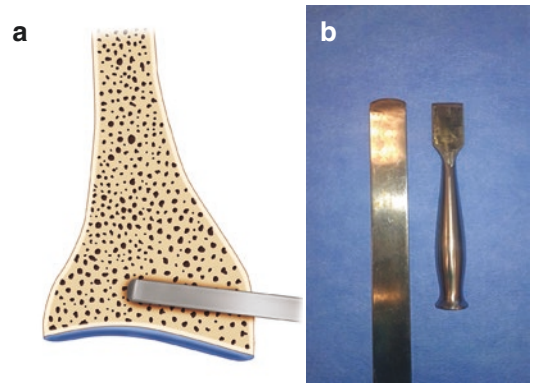

Fig. 1.3 (**a**) Schematic image of the bone block by Eden. (Drawing by the author). (**b**) Special chisels for Eden-Hybinette procedure used in our clinic in the 1980s

including the subscapularis muscle. There were two groups of surgical techniques: the first group have performed various variations of soft tissue tensioning, while the other group introduced a bone block to be used as an anterior plug that will prevent the humeral head to dislocate. So, let us go back to 1917. Then the first paper about the bone block procedure appeared by Eden (Fig. 1.3a, b). He used a cortical tibia graft and introduced it into the anterior glenoid rim [12]. In 1932 the similar procedure was published by Hybinette, only he used an iliac bone block [13]. At that time with no Internet, it was possible that two surgeons invented the same method and publish it in their country without knowing for each other. Around the year 1980 when I discovered the orthopedic world in the late 1970s of the twentieth century, this method was widely used in my country, so in my clinic we still preserve (in the museum) special chisels for this purpose.

Magnuson-Stack method in 1943 described transferring the subscapularis under the coracoid and over the biceps tendon to stabilize the joint [14]. Next similar soft tissue procedure was the one by Russian surgeon Boicev (1951) who has also transferred the subscapularis but over the conjoined tendon and fixed it more laterally [15].

The next important method was again the bone block stabilization. It was published in 1954 by Latarjet [16]. Four years later Helfet has published a similar method invented by Bristow who died before publishing it [17]. Therefore, it is now known as "Bristow-Latarjet" procedure which consisted of the coracoid transfer to the anterior glenoid rim. This method was so successful that nowadays it is wary popular, especially as a difficult arthroscopic procedure, just to show what

we can achieve with mini invasive techniques. This technique has two effects: the first is a bone block limitation, and the second is a sling effect of the attached conjoint tendon which brings more tension to the anterior aspect of the joint.

It is a fact that hip and knee surgery developed much faster than shoulder. We can speculate about the reasons, but it is a fact. So, the 1st Symposium on Surgery of the Shoulder Region was held in Montreal in 1963. The first International Conference on Surgery of the Shoulder was organized in London in 1980.

1.1 Arthroscopy: The Modern Era

Lanny Johnson in London has performed the first arthroscopic stabilization of the shoulder in 1980, the same year of the London Conference [18]. He used staples and had recurrence in 15–25%, but it was only the beginning. In fact, arthroscopic techniques had the same aim as Bankart, that is, to refix the labrum to the glenoid. For this purpose, some implants were necessary. Seven years later, two Americans Morgan and Caspari have introduced transglenoid sutures with no implants (Fig. 1.4) [19, 20]. The 1990s was the era of huge development of arthroscopic techniques and solutions. All kinds of implants were invented including wires, staples, screws, and anchors. Still the recurrent rate was between 4 and 35% long after that. Arthroscopy enabled a precise visualization of the entire shoulder joint, and new precise classification of several types of capsulolabral tear was introduced.

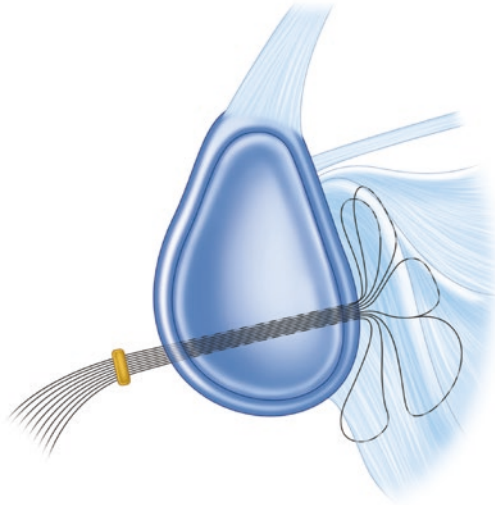

Fig. 1.4 Transglenoid sutures by Caspari. (Caspari RB (1988) Arthroscopic reconstruction for anterior shoulder instability. Tech Orthop 3:1)

The evolution of arthroscopic techniques enables complex and demanding extraarticular surgeries where almost everything is possible. The technology and industry encourage the surgeons to it. New generations of arthroscopic equipment and young and courageous surgeons send us the message: "only the sky is our limit"!

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2 The Anatomy in Shoulder Instability

Ángel Calvo Díaz, Pablo Carnero Martín de Soto, and Néstor Zurita Uroz

2.1 Introduction

The glenohumeral joint is the most commonly dislocated joint of the body [1], with an incidence of 24 per 100,000 cases per year [2]. The bony anatomy of the articular surfaces of the humeral head and the glenoid allows a great arc of mobility but leads to a relatively unstable joint. The glenoid fossa is a shallow articular surface that covers a small portion of the humeral head. Thus, the surrounding soft tissues, such as the labrum, capsular attachment, and glenohumeral ligaments, have a key role on maintaining articular congruency. The mechanisms responsible for compensating the bony instability of the joint are varied and complex. In general, they can be divided into static and dynamic stabilizers (Fig. 2.1). Static stabilizers are the most important, as their isolated injury can develop recurrent instability. This group comprises the labrum, the articular capsule, and the glenohumeral ligaments. These last are often visualized as reinforcements of the capsule, so the term capsuloligamentous complex is frequently used to describe its anatomy and combined function. The main goal of the static stabilizers is to maintain congruency during the last degrees of movement [3] and to provide passive stability to the joint. The dynamic stabilizers are the musculotendinous structures whose contractions maintain the humeral head centered during joint movement [4, 5] and include the rotator cuff, the scapular muscles, and the neuromuscular control that allows coordinated contraction of all these structures.

Understanding the anatomy of the bony and soft-tissue components of the glenohumeral joint is crucial to identify the pathological changes that occur in shoulder instability to optimize treatment procedures. Moreover, it is mandatory to recognize the normal variants, which are not infrequent, to avoid overtreating our patients, which can lead to suboptimal outcomes.

2.2 Anatomy

2.2.1 Bone Anatomy

The glenohumeral joint is a ball-and-socket joint formed by the rounded head of the humerus and the cup-like depression of the scapula called the glenoid fossa. The glenoid articular surface covers about 25–33% of the surface of the humeral head, leaving a relatively unstable joint (Fig. 2.2).

The glenoid cavity is pear- or oval shaped (in 88% and 12% of cases, respectively [6]) and retroverted 5° to 7°, whereas the humeral head is retroverted 30°. The angle between the humeral head and the diaphysis is about 130°–150° [7].

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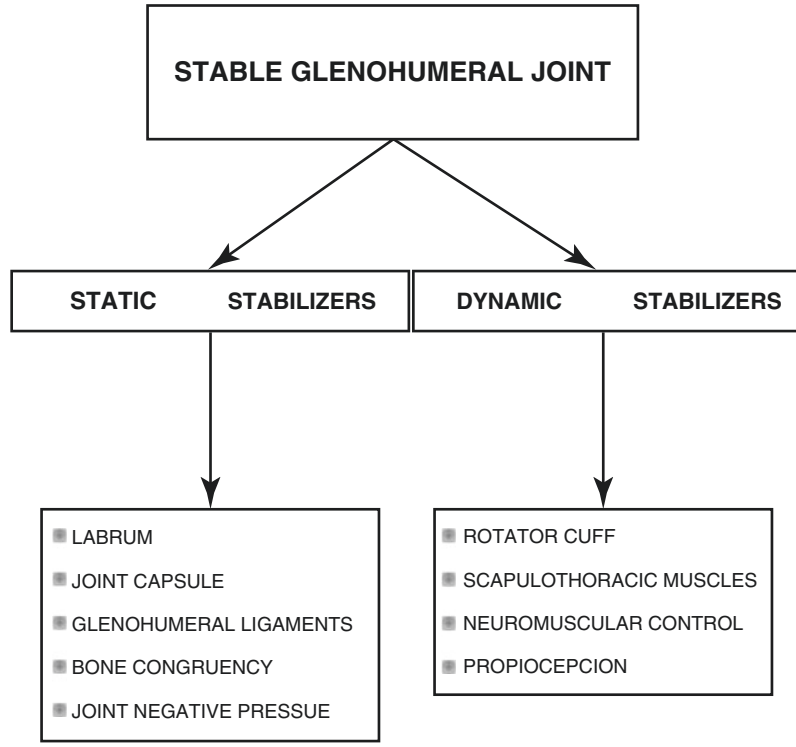

Fig. 2.1 Stabilizers of the shoulder

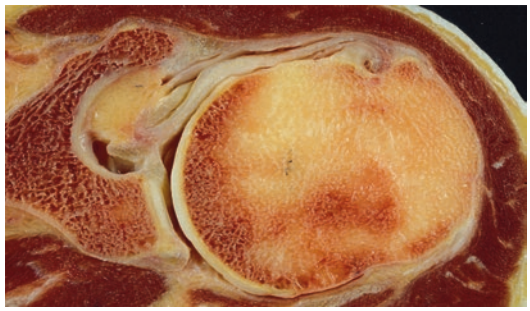

Fig. 2.2 Axial cut of the shoulder in cadaveric specimen. A large mismatch between articular surfaces of the humeral head and the glenoid fossa is visible

The humeral head cartilage is thicker centrally and thinner peripherally, in contrast to the glenoid articular cartilage, which is thinner centrally and thicker peripherally. The central area of the glenoid, also called the "bare area" or "bare spot," has a recognizable depression of the cartilage and should not be mistaken for a cartilage defect. This area has double function in providing osseous stability to the joint. First, it deepens the concave shape of the glenoid to increase the contact with the humeral head. Second, this greater contact between articular surfaces creates a negative pressure environment in the joint in which the glenoid fossa "suctions" the humeral head, impeding its migration during movement [8]. The labrum, the capsule, and the synovial fluid are also important in this mechanism [9].

An analogous bare area can be found on the posterolateral zone of the humeral head, between the cartilage and the insertion of the infraspinatus. A Hill–Sachs lesion should not be confounded with this physiological finding (Fig. 2.3).

2.2.2 Soft-Tissue Anatomy

2.2.2.1 Labrum

The labrum is a fibrous and fibrocartilagenous ring attached around the margin of the glenoid cavity. Peripherically, it is composed of collagen fibers disposed circularly and radially and has close relationships with the attachment of the long head of the biceps tendon, the joint capsule, and the glenohumeral ligaments. Medially, the transitional zone provides firm attachment to the glenoid.

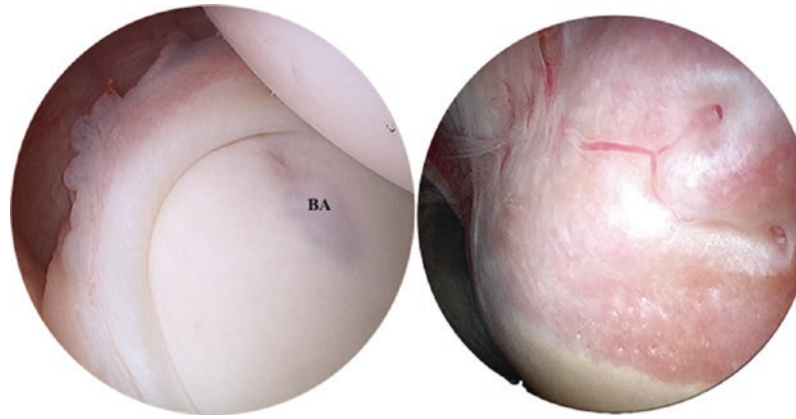

Fig. 2.3 Glenoid and humeral head cartilage: bare areas (BA)

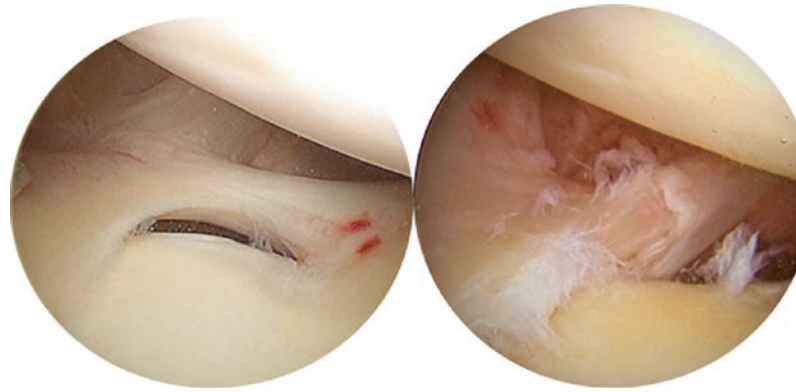

Fig. 2.4 Left: Sublabral foramen. Right: Buford complex, in which a cord-like thick middle glenohumeral ligament and absent anterosuperior labrum are visible

The main function of the labrum is increasing the contact area between the glenoid fossa and the humeral head by about 30–50% [10], which favors a greater articular congruency. In addition, it restrains the anterior or posterior displacement of the humeral head and "seals" the space between the articular surfaces, helping to maintain the negative pressure in the joint.

Different anatomical variants have been described in labrum insertion and should not be confounded with pathological changes (Fig. 2.4). The *sublabral recess* may be found superiorly in the glenoid. It is a variation of the insertion of the bíceps–labral complex at the 11 to 1 o'clock positions. It is frequently seen as an incomplete detachment of the labrum that partially shows the superior border of the glenoid neck. It is formed by a reflexion of the synovial layer that covers the articular margin of the glenoid and the labrum, and does not generate instability of the labrum or the long head of biceps tendon insertion, so it should not be addressed as a superior labrum anterior-posterior (SLAP) tear. The *sublabral foramen* is a complete detachment of the anterosuperior labrum that does not extend inferiorly to the 9 o'clock position in the left shoulder or the 3 o'clock position in the right shoulder. This finding is not involved in the development of shoulder instability, as the remaining inferior labrum, which is the most important in providing stability, remains intact. The sublabral foramen is visible in less than 20% of patients [11]. The *Buford complex* was described as a "cord-like" middle glenohumeral ligament that originated directly from the superior labrum at the base of the biceps tendon associated with no anterior-superior labral tissue present between this attachment and the mid-glenoid notch [12]. Its incidence ranges from 1.5 to 6% [11–13], and there is common agreement in considering it a normal variant, as its surgical fixation to the glenoid rim could cause important restriction to external rotation and

elevation [12]. Indeed, the presence of a cord-like middle glenohumeral ligament has been identified as a protective factor against instability [11, 14]. However, few case reports have been published about patients with recurrent shoulder instability associated with the Buford complex [15, 16]. In addition, the abnormalities of the anterosuperior labrum, including the Buford complex and sublabral hole, may influence shoulder biomechanics as patients usually present an increased internal rotation and variations at the superior glenohumeral ligament and the rotator interval [17]. However, the clinical implications of these findings are uncertain. Although a relationship between variants of the anterosuperior labrum and SLAP lesions has been documented [11, 17, 18], it has not been established with shoulder instability.

2.2.2.2 Capsuloligamentous Complex

The joint capsule inserts into the glenoid margin of the scapula and the anatomic neck of the humerus. It is made of collagen fibers disposed circularly and radially and support the tensile forces when the joint abducts and rotates. It is reinforced by the glenohumeral ligaments that, together with the capsule, tighten when the shoulder reaches the last degrees of movement.

There are two recesses located between the reinforcements of the glenohumeral ligaments and the rotator cuff muscles. The subscapular recess, or *Weitbrecht foramen*, is an opening of the anterior capsule located between the superior glenohumeral ligament and the superior border of the subscapularis tendon and communicates the joint with the subtendinous bursa of the subscapularis. The *axillary recess* or *axillary pouch* is located between the anterior and the posterior bundles of the inferior glenohumeral ligament.

The anterior capsular insertion can be divided into three types depending on the location on the glenoid margin. In type I, capsular attachment reaches the glenoid and labrum. In type II, the capsule attaches on the glenoid within 1 cm of the labrum. In type III, the capsule attaches more than 1 cm medial to the labrum [19]. As the capsule attachment becomes farther away from the labrum, it becomes thinner, the recesses are larger, and the capsuloligamentous complex is less resistant [19, 20].

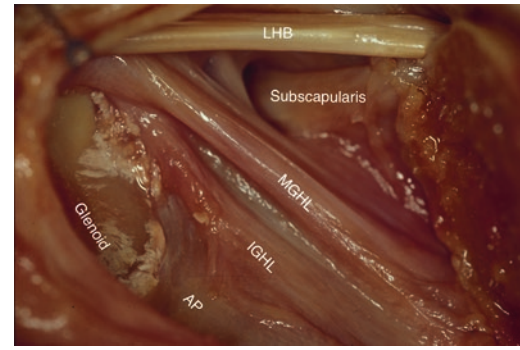

Fig. 2.5 Anterior structures of the shoulder seen from posterior on a cadaveric specimen. *LHB* long head of the biceps tendon, *MGHL* middle glenohumeral ligament, *IGHL* inferior glenohumeral ligament, *AP* axillary pouch

The glenohumeral ligaments are fibrous reinforcements of the joint capsule that restrain the humeral head translation when the range of motion reaches its maximum. Different ligaments have been described with diverse functions (Fig. 2.5).

- The superior glenohumeral ligament (SGHL) originates from the supraglenoid tubercle, anterior to the insertion of the long head of the biceps tendon, and inserts on the cephalic side of the lesser tuberosity, medial to the bicipital groove. Its location and thickness are very variable, as it can be not visible in 59% of cases [11]. It acts stabilizing the shoulder in adduction, limiting the inferior and posterior translation. The SGHL also forms the "*bicipital pulley*" together with the coracohumeral ligament, which prevents dislocation of the biceps tendon intraarticularly.
- The middle glenohumeral ligament (MGHL) originates from the anterosuperior glenoid rim, close to the origin of the SGHL, and inserts onto the anatomic neck of the humerus, adjacent to the lesser tuberosity. It is often seen as a thin layer anterior to the subscapularis tendon, but can be visualized as a thick cord-like structure, as in the Buford complex. It prevents anterior translation of the humeral head when the shoulder is in mid-abduction of 45° and external rotation.

- The inferior glenohumeral ligament (IGHL) has three distinct parts: the anterior bundle, the axillary pouch, and the posterior bundle. The anterior bundle of the IGHL originates from the anteroinferior labrum and glenoid neck, the posterior bundle of the IGHL from the posterior labrum and the glenoid neck, and the axillary pouch from the inferior labrum. The common insertion is at the inferior and medial part of the anatomic neck. The IGHL has the least variability of the glenohumeral ligaments and is the main stabilizer of the shoulder. When it is in adduction, it remains lax and folded, but when the shoulder is at 90° of abduction and external rotation, the axillary pouch unfolds and the anterior bundle tightens to limit anterior translation of the head. At abduction and internal rotation, the posterior bundle tightens and limits posterior translation and excessive internal rotation. This selective function of its parts has been described as a "*hammock effect*" that allows great range of motion while maintaining stability of the joint.

2.2.2.3 Rotator Interval

The rotator interval is a triangular space marked by the anterior border of the supraspinatus tendon, the superior border of the subscapularis tendón, and the base of the coracoid process. It contains several structures such as the SGHL, the coracohumeral ligament, the long head of the biceps tendon, and the anterosuperior capsule. It has been reported that it has a role in glenohumeral stability. Its functions are limiting inferior translation of the humeral head with the arm adducted; limiting external rotation; and controlling anterior and posterior translation during adduction and flexion–extension. Therefore, a wide rotator interval leads to an increased anterior, posterior, and inferior humeral head translation [21]. However, during shoulder arthroscopy it is difficult to assess whether a rotator interval is widened as there are no measurement methods described, so the indication of performing a rotator interval closure as an associated procedure to treat shoulder instability is debated [22].

2.3 Dynamic Stabilizers

The musculotendinous structures of the rotator cuff reinforce the whole capsular area except in the axillary recess and the rotator interval. Their contraction keeps the humeral head centered during joint movement in the mid-range of motion. Thus, the action of the stabilizers of the shoulder can be explained as a continuum, in which at the beginning and the mid-phase of the movement the static stabilizers remain lax and the contraction of the rotator cuff tendons provide congruity. When the extreme range of motion is reached, stability depends on the static elements.

Proprioception of the shoulder is crucial in its dynamic stabilization. The glenohumeral capsule is richly innervated by mechanoreceptors [4] that send information to the cerebral cortex to establish a pathway that finishes with a coupled contraction of the muscles of the rotator cuff and the scapulothoracic space to provide stability of the joint during movement [5]. A torn or stretched capsule can cause disturbance of the mechanoreceptors and delay of the proprioceptive signal, slowing the feedback response of the musculotendinous units, so that injury of the static stabilizers can also cause disruption of the dynamic stabilizers.

2.4 Pathology

Shoulder instability is a complex pathological entity with different clinical presentations. Several classifications have been described according to its etiology (traumatic versus atraumatic instability), direction of instability (anterior, posterior, multidirectional), timing (acute, locked, recurrent), associated injuries (with versus without bone loss), or a combination of these factors (TUBS versus AMBRII). This variety of classifications shows that the clinical spectrum of shoulder instability is wide. Therefore, the anatomical lesions present depend on the clinical setting of the patient. Thus, injuries found during surgery will be different in patients with recurrent anterior traumatic instability than in those with atraumatic multidirectional instability with

associated hyperlaxity, and, consequently, the surgical approach should be different in each case.

It is accepted that the origin of the dysfunction in most cases is the combined injury of the static stabilizers. The labrum avulsion secondary to the dislocation or subluxation of the humeral head is often associated with pathological capsular redundancy, whether congenital or acquired. In addition, the mechanoreceptors of the capsule can become damaged in capsular injury, which promotes an impaired response of the dynamic stabilizers as well. However, in some cases the isolated capsular redundancy can cause shoulder instability without labrum detachment.

In other cases, the origin of the instability can be found in an abnormal pattern of contraction of the musculotendinous units around the shoulder. Neuromuscular pathology, such as muscular dystrophy, cause a weakness of the rotator cuff muscles that impedes keeping the humeral head centered during shoulder motion. In these cases, a certain capsular laxity is needed to develop the instability, so the static stabilizers are not disconnected from these infrequent types of instability.

2.5 Labrum Injury

Our understanding of labrum injuries has increased in recent years thanks to the development of arthroscopy, and different lesions have been described in both acute dislocations and chronic instabilities.

After an anterior traumatic shoulder dislocation, the most common sequel is a complete avulsion of the anteroinferior labrum of the glenoid rim and the periosteum, known as the *Bankart lesion*. This finding is almost constant in cases of recurrent instability [23], as it is a major cause of the instability [24]. When the injury comprises a marginal fracture of the anteroinferior portion of the glenoid neck instead of labrum detachment, it is known as a Bony-Bankart.

The *anterior labroligamentous periosteal sleeve avulsion* (ALPSA lesion) is often visualized in patients with multiple dislocations [23, 25]. It consists of a complete detachment of the labrum and the glenohumeral ligaments from the glenoid rim but maintaining a bundle of the glenoid neck periosteum, so the detached structures retract medially and are scarred to the medial glenoid neck, being unable to limit the anterior translation of the humeral head (Fig. 2.6).

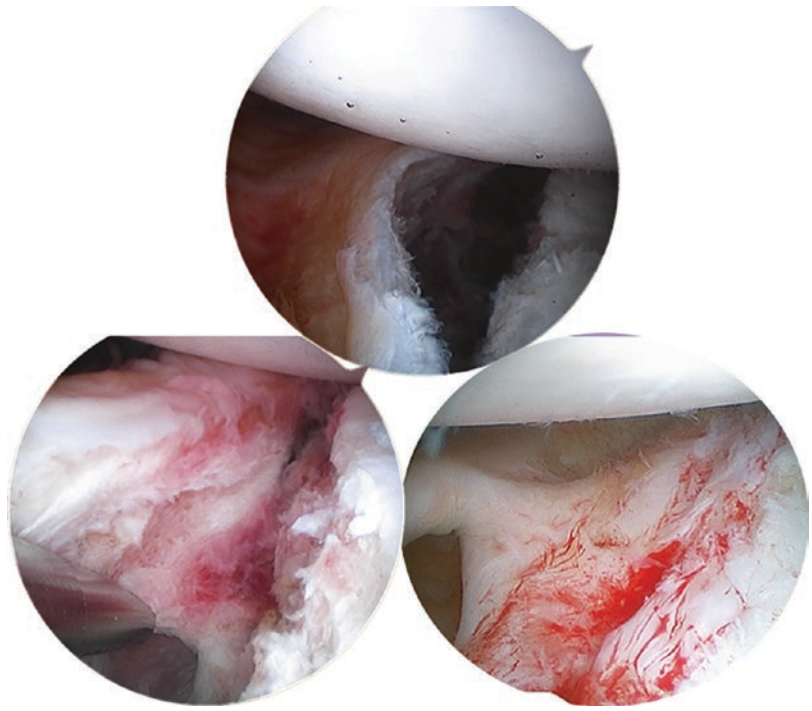

Fig. 2.6 Up: Anterior Bankart lesion; down left: anterior Bony-Bankart; down right: ALPSA lesion

A *Perthes lesion* is an incomplete avulsion without displacement of the anteroinferior labrum with a medially striped but intact periosteum [26]. It is an uncommon lesion that rarely causes gross instability, but should be suspected in patients with subluxation of the joint and recurrent pain.

The *glenoid labral articular disruption* (GLAD) *lesion* was first described by Neviaser in 1993. This lesion occurs when there is a defect in the articular cartilage of the anteroinferior glenoid in addition to the labral tear, which is not fully detached [27]. Similar to the Perthes lesion, the predominant symptom is these cases is not instability, but pain.

All these injuries just described are not exclusive to the anteroinferior labrum. In cases of posterior shoulder instability, analogous lesions can be found on the posteroinferior labrum, added to incomplete detachments of the superficial portion of the posterior labrum, which are known as *Kim's lesions*. These incomplete tears usually appear as a consequence of repetitive movements of flexion, adduction, and internal rotation, so athletes such as throwers or weightlifters are prone to these disruptions. They do not cause gross instability symptoms, but instead origin pain and shoulder dysfunction.

2.6 Capsular Injury

A plastic irreversible elongation of the anteroinferior capsule is frequently seen in patients with anterior recurrent instability [23]. Global capsular redundancy is a usual feature in hyperlaxity and multidirectional instability, whereas in unidirectional instability stretching of the anteroinferior portion of the capsule is paramount [28]. It is difficult to determine when an increased capsular volume is congenital or acquired, but it seems logical that perhaps the most frequent origin combines an inherent predisposition and a traumatic component [29]. However, capsular insertions below the labrum (i.e., type III) predispose to glenohumeral hypermobility or even instability without traumatic antecedent.

The humeral avulsion of the glenohumeral ligaments (*HAGL lesion*) is a variant of capsular injury after an acute dislocation. Reported in 2–9% of patients with shoulder instability [30, 31], this injury usually happens when the arm is placed in maximal abduction and external rotation. Anterior HAGL represents 93% of the cases, whereas posterior accounts for only 7% [30]. These lesions can be easily missed during shoulder arthroscopy if they are not suspected, especially in cases without a concomitant Bankart lesion. Consequently, placing the scope on the anterosuperior and anterior portals while abducting and externally rotating the arm must be routinely performed to visualize the humeral insertion of the capsuloligamentous complex.

2.7 Bone Injury

2.7.1 Glenoid

Several glenoid bony configurations can predispose to recurrent instability. Glenoid bone loss is the most common and has received more attention than other matters, as it is considered an important contributor to recurrent shoulder instability. Previous reports show that recurrence rates after arthroscopic soft-tissue procedures for anterior instability are 4–6% [29, 32], but when there is significant bone loss, either at the glenoid rim or at the posterolateral aspect of the humeral head, the rate is as high as 89% [32] even after lower-energy traumas.

Following an initial shoulder dislocation, an osseous defect on the anteroinferior margin of the glenoid is present in up to 22% of patients and in up to 88% of patients with recurrent instability [33, 34]. This defect predisposes to instability during the middle range of motion as the concavity of the rim is lost, so the humeral head finds no stop to anterior translation. Moreover, the suction effect of the glenoid is missed as it loses its cup shape.

The loss of the normal shape of the glenoid can be assessed radiographically or arthroscopically. Computed tomography scans permit obtaining tridimensional reconstructions of the glenohumeral joint that allow quantifying the bone defect. On the other hand, arthroscopic

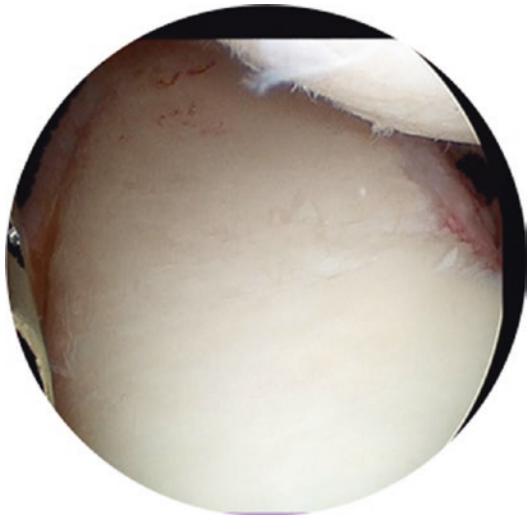

Fig. 2.7 Anteroinferior significant glenoid bone loss causing an "inverted pear" morphology of the glenoid

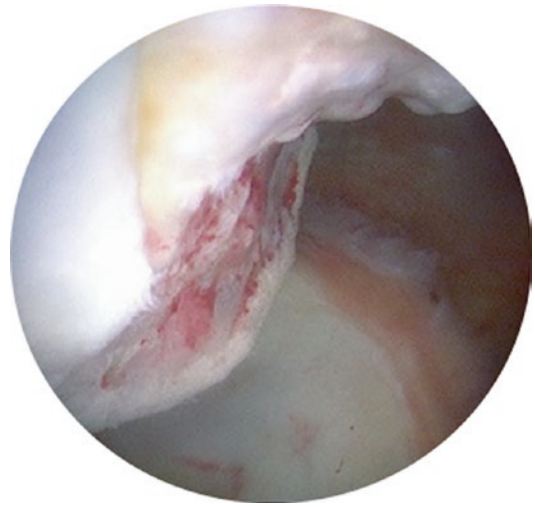

Fig. 2.8 Hill–Sachs lesion

examination provides direct visualization of the glenoid, as it would appear as an "inverted pear" shape in cases of significant bone loss (Fig. 2.7). Furthermore, anteroinferior bone defects can be measured with a calibrated probe inserted through the posterior portal. Using the bare area of the glenoid as the landmark, the posteroinferior and anteroinferior radii of the glenoid can be measured and compared.

There is common agreement on considering 25% of anteroinferior glenoid bone loss as the critical point at which arthroscopic soft-tissue procedures are not sufficient for correcting instability, so a glenoid grafting technique should be performed. This limit was determined after biomechanics studies reported that a defect measuring 30% of the diameter of the inferior glenoid causes a decrease in the contact area across the entire glenoid of 40%, whereas the mean contact pressure for the entire glenoid increased by nearly 100% and mean contact pressures in the anteroinferior quadrant increased by 300–400% [35]. If an isolated soft-tissue repair were to be performed in a patient with this glenoid bone loss, it would have to resist this overload at the repair interface, dramatically increasing the likelihood of failure.

Abnormalities of glenoid anatomy and version have been studied in the setting of multidirectional and posterior instability. It has been shown that glenoid retroversion is higher in patients with posterior instability compared to control subjects or patients with anterior instability [36]. Interestingly, when shoulder retroversion reaches 16°, the incidence of contralateral injuries is increased [37]. However, it is unknown whether osseous changes precede the development of instability or whether instability itself causes the bony changes. Furthermore, although the exact amount of glenoid retroversion necessary to affect shoulder joint stability is unclear, the connection between retroversion and posterior instability exists, so future research about this issue will aid us to obtain clear conclusions.

2.7.2 Humeral Head

A posterolateral bone defect is frequently seen after initial shoulder dislocation. This finding, called the *Hill–Sachs lesion*, is present in up to 51–65% of cases after the first episode of dislocation, and the rate is higher in chronic instability [23, 29] (Fig. 2.8). As previously stated, there is a bare area between the insertion of the rotator cuff and the humeral head cartilage that should not be confused with an injury.

The presence of a Hill–Sachs lesion predisposes to recurrent instability, even after an arthroscopic

soft-tissue procedure stabilization [32, 38]. Therefore, correct identification and quantification of the deformity is mandatory during shoulder arthroscopy. Complete visualization of the injury can be obtained by placing the scope in the anterosuperior portal and rotating the shoulder.

The orientation of the lesion is important in the development of recurrent instability. If the medial border of the Hill–Sachs defect passes the medial border of the glenoid during external rotation, it will "engage" and facilitate dislocation, so it will be considered an "engaging" injury. If the medial border of the Hill–Sachs lesion does not overpass the glenoid, rather if it is not large enough or the orientation of the injury does not fit with the medial border of the glenoid, the complete arc of motion of the shoulder can be achieved and there will be a small likelihood of dislocation, so it will be considered as a "non-engaging" lesion. In case of an engaging lesion, additional surgical procedures such as infraspinatus tendon tenodesis may be required.

2.8 Conclusions

Shoulder anatomy is particularly complex and requires a thorough knowledge by the orthopedic surgeon. Anatomical variants should not be confounded with pathological findings. Moreover, different anatomical lesions can be found depending on the type of instability and the functional requirements of the patient, so the surgical procedures must be carefully chosen to achieve optimal outcomes when treating shoulder instability.

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3 Predictor Factors in Anterior Shoulder Instability

Boris Poberaj

3.1 Age

The age is one of the key risk factors for primary and recurrent instability. The epidemiologic study [1] using cohort of patients aged 10–16 years has shown that among 10- to 13-year-old patients, there was considerably lower rate of primary and recurrent dislocations. The reason for lower recurrence rate in younger adolescents seems to be more elastic capsule resilient to structural damage and more lateral attachment of the anterior capsule to the glenoid. The statistics among 14- to 16-year-old individuals for primary and recurrent dislocations is comparable to high-risk adults 17–20 years old. Recurrent dislocation in adolescents after a primary anterior dislocation usually occurs within 2 years with incidence of 76.7% [2]. The patients in this study were treated after first dislocation with sling immobilization in internal rotation for 1 week; then early movement was allowed as pain allows with physiotherapy for 8 weeks. Another systematic review and meta-analysis [3] that included 1324 patients have shown 51% of recurrence after first dislocation in the age group of 15–20 years, 49% in the age group of 15–30 years, and 36% in the group of 21–40 years.

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3.2 Gender and Race

In general males have greater risk of shoulder dislocation than females because they participate more commonly in higher-risk contact sports. Also many traditional collision sports have modified rules in the women's version. Kardouni et al. [4] reported 15,426 incident shoulder dislocations in US soldiers with greater risk in male population. At the same time, male soldiers had a 20% decreased odds of chronic or recurrent injury than female soldiers. This is in line with other reports of no significant difference in recurrence rates based on sex. Incidence on recurrent dislocation is similar in both genders during the adolescence [2]. In the study of Kardouni, results indicate that white people may have a greater risk for sustaining shoulder dislocations than other races.

3.3 Associated Fractures and Axillary Nerve Lesions

Bony fracture of anterior glenoid and Hill-Sachs deformity importantly increase the risk of recurrence.

The presence of a greater tubercle fracture of the humerus decreased the risk of recurrence rate for 4–7 times. This was postulated due to decreased range of motion in external rotation and abduction, which is usual sequel after such traumas.

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An axillary nerve palsy does not affect the structural integrity of the joint but results in decreased movement of the limb for a significant period of time.

3.4 Other Factors

Hyperlaxity increases recurrence for up to three times following a first-time dislocation.

Immobilization in external or internal position has no influence on recurrence rate, as well as duration of immobilization.

Overhead athletes, collision sports, and overhead manual workers have increased risk of recurrence.

3.5 Diagnostic Value of Clinical Tests

There are numerous clinical tests for anterior shoulder instability. The most commonly used are apprehension, relocation, and release tests.

The apprehension test is done with the patient supine with the arm in 90° abduction, the elbow in 90° of flexion, and progressive external rotation. The test is positive in case of an apprehensive feeling and negative if only pain is present.

The relocation test is literarily continuation of apprehension test with depression of the humeral head by posterior-directed force to the humerus. It is considered positive if it provides relief of the apprehensive feeling. At the same time, external rotation can be proceeded to its maximal range.

The anterior release test is continuation of relocation test with sudden release of posterior-directed force to the humerus. The test is positive in case of recurrent apprehensive feeling.

Kampen et al. [5] confirmed good diagnostic accuracy of individual tests with overall accuracy more than 80% with highest score for release test with 86.4%.

The assessment of anterior apprehension test was further studied by Milgrom et al. [6] in cohort of patients with first-time shoulder dislocations and minimum follow-up of 75 months. The mean age of the patients was 20 years, and the tests were performed 6 weeks after first traumatic dislocation, followed by rehabilitation. Those with a positive test result have had a 79% rate of recurrent dislocation, and those with a negative test result had a 53% rate. Also those with a positive test sustained redislocation earlier than those with a negative test result.

Finally, shoulder apprehension is more complex than a pure mechanical problem as it reflects the scar at the brain level that prevents the performance of specific movements [7]. Brain activity changes can predict the successful postsurgical outcome. Decreased activity in premotor and orbitofrontal cortex is a key factor for a successful surgical outcome.

3.6 Predictors of Dislocation After Shoulder Stabilization

Younger patient's age and increased numbers of documented preoperative dislocations increase the likelihood of stabilization failures [8]. It seems also that shoulder dislocations that require physician relocation are more likely to have significant pathological lesions like bigger bone defects, which potentially increase risk of failure of operative stabilization. The number of dislocations and age at first dislocation are the most significant predictors of glenoid bone loss [9]. Patients with three or more preoperative dislocations required physician relocation had postoperative recurrent dislocation rate of 24.4% compared with 2.4% for patients who had none in the cohort of 73 patients. These data support the promoters of surgical intervention for the first-time dislocation [10].

3.7 Surgery for the First-Time Dislocation

Main reasons to support surgery for the first-time dislocation are:

- At age of 16–27 years, the redislocation rate is up to 80%.
- Young patients with three or more dislocations before surgery have up to 25% recurrence rate after surgery.

– Glenohumeral osteoarthritis in chronic instability is 10–20 times greater.

The reasons to not support the first-time dislocation are:

– Approx. 20% of surgeries among young athletes would be unnecessary.
– Additional 14% of the surgeries would be unsuccessful.

3.8 Summary

Young age 16–27 years, male gender, and collision sports are predictors for primary glenohumeral (GH) dislocation.

The supine apprehension test 6 weeks [11] after first GH dislocation can help in predicting risk of recurrent instability.

Age 20 years or less with more than three preoperative dislocations predicts significant risk of revision surgery.

Any shoulder stabilization study with only 2-year follow-up should be interpreted with caution.

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4 Basic Science on Shoulder Instability

Tim Kraal, William D. Regan,
and Christiaan J. A. van Bergen

4.1 Introduction

The shoulder has an impressive wide range of motion in three dimensions, but sometimes, this comes at a price. The glenohumeral joint is the most commonly dislocated joint in the human body. Basic science research on shoulder instability is of paramount importance to understand the biomechanics of the shoulder, including static and dynamic constraints that control stability. Several studies have investigated joint contact pressures and contact areas in various arm positions, as well as the way they are affected by bone loss caused by instability [1–3]. Furthermore, other biomechanical studies have investigated the effects of surgical stabilizing procedures on joint biomechanics.

Glenohumeral stability is a complex issue, reliant on a multitude of static and dynamic factors, which cannot be simulated in a biomechanical study. There is intrinsic stability from the glenoid concavity and the congruency of the glenoid and labrum with the humeral head. Although the articular surface of the humeral head is about three times larger than the articular surface of the glenoid, the radius of curvature of the humeral head and the glenoid is within 2 mm of each other in most cases [4]. Furthermore the capsuloligamentous structures are a (patient-specific) static restraint, mainly important in the end range of motion [5, 6]. In the apprehension position, i.e., combined abduction and external rotation, the labrum and the inferior glenohumeral ligament (IGHL) resist antero-inferior translation [7]. The labrum itself is a fibrocartilaginous structure attached to the glenoid rim, and it increases the depth of the glenoid. Its collagen fibres are oriented in a circumferential manner, and are densely packed at the core layer. The antero-inferior part of the labrum has the highest elastic modulus and yield stress; it is the thickest and strongest part of the labrum, providing maximal resistance to translational forces of the humeral head [8]. In addition to these intrinsic and static restraints, there is dynamic muscle control. The prime movers of the shoulder include the rotator cuff and the deltoid. These, and other muscles to a lesser degree, create a joint reaction force, compressing the humeral head in the concave glenoid fossa. Muscle activation is directed by proprioception, mediated by mechanoreceptors in tendons. Although in a cadaveric specimen the individual muscles can be dissected, the tendons can be clamped and loaded individually with computer-controlled actuators; these models are still a simplification of reality [9, 10].

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