Giuseppe Milano
Andrea Grasso
Editors

Shoulder Arthroscopy

Principals and Practice

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To Federica, Francesca, and India, to remind them that "He who becomes the slave of habit, who follows the same routes every day, who never changes pace, who does not risk and change the color of his clothes, who does not speak and does not experience, dies slowly" (M. Medeiros).
To Adriana, for the meaning she gave to the journey of my life.

G.M.

To Federica, my wife, the most inspiring person in my life and my work, for every day of our lives together. And also to our five children.

A.G.

Foreword 1

When I first began to do arthroscopic side-to-side rotator cuff repairs in 1987 using very crude instruments, I was struck by how much better I could see and evaluate the repair pattern using the arthroscope than when I did open surgery. Despite the enthusiasm of a small group of talented arthroscopic surgeons including Lanny Johnson, Howard Sweeney, Jim Esch, Steve Snyder, and Dick Caspari, the shoulder establishment of that time rejected the idea that arthroscopy might have any potentially meaningful role in shoulder surgery. Nonetheless, the "true believers" in arthroscopy persisted against the mainstream.

Now, 26 years later, this marvelous book entitled *Shoulder Arthroscopy*: *Principles and Practices* by Dr. Giuseppe Milano and Dr. Andrea Grasso clearly demonstrates that the world's thought leaders in shoulder surgery have totally embraced shoulder arthroscopy and are now its strongest advocates and practitioners. The list of authors reads like a "Who's Who" of shoulder surgery. This book is also a very comprehensive resource, beginning with general principles, progressing to routine arthroscopic procedures, and continuing into extremely complex reconstructive and revision surgeries that can and should be done arthroscopically.

The fact that they were able to assemble such a distinguished cast of authors for the chapters of their book is a tribute to the high regard in which Drs. Milano and Grasso are held by their peers. When I first met Dr. Milano about 15 years ago, I was struck by his tenacity and by his scientific approach to the shoulder, and these characteristics have prominently influenced his new book. This book is a magnificent work that captures the essence of state-of-the-art shoulder surgery. It is an indispensable resource for today's shoulder surgeon. I congratulate Drs. Milano and Grasso on a job well done.

San Antonio, TX, USA — Stephen S. Burkhart, MD

Foreword 2

Shoulder arthroscopy is nowadays a very fast-developing surgical technique. A need for evaluation of different treatment options, clinical investigation methods, education support and clinical research has brought together orthopaedic surgeons from all over Europe, who have founded European Shoulder Associates (ESA) under the patronage of ESSKA.

Some of our Board members have contributed to different chapters of this book and it is a special honour to announce that Dr. Giuseppe Milano, the editor of this book, is the Scientific Secretary of ESA Board.

On behalf of ESA, I wish all the readers to benefit from this book as a guide to improve their practice.

Boris Poberaj, MD

Preface

During the last decades, shoulder arthroscopy gained much popularity among surgeons involved with shoulder surgery and sports medicine, and new several surgical techniques have been rapidly developed.

For a long time, hot topics like massive rotator cuff tears, subscapularis tendon tears, anterior instability, glenoid bone loss, and revision rotator cuff and instability surgeries were addressed only to invasive, often non-anatomical, open surgical procedures. Few years ago, it was unimaginable to perform an arthroscopically assisted latissimus dorsi transfer or an arthroscopic Latarjet procedure. Nowadays, all the above mentioned surgeries can be performed arthroscopically.

Arthroscopy has undeniable advantages: it provides a better visualization of anatomical structures and allows anatomical reconstruction and better bleeding control, it reduced surgical times, and it is not perceived as invasive by the patient, thanks to the absence of surgical incision and early recovery after surgery. Few years ago, first studies compared open and arthroscopic procedures; recently, biomechanical and clinical studies comparing different arthroscopic techniques exploded and technological improvements run fast.

The need to stay updated and to have, at the same time, a comprehensive textbook on the entire spectrum of shoulder diseases and arthroscopic techniques was the driving force behind "*Shoulder Arthroscopy*: *Principles and Practice*." It covers from basic science (anatomy and biomechanics of the shoulder) through arthroscopic basic procedures up to the advanced reconstructive surgeries.

Differently from many other books on surgical techniques, which generally seem to be "how-to-do" handbooks, this book tries to give a comprehensive overview on shoulder pathologies with a special focus on surgical approach.

"*Shoulder Arthroscopy*: *Principles and Practice*" is highly organized, and all the chapters follow the same format: from detailed descriptions regarding epidemiology, pathophysiology, clinical findings, imaging, indication for treatment up to a step-by-step description of up-to-date surgical techniques, including tips and tricks on how to avoid the most common mistakes and complications. Finally, there is also an entire section dedicated to the evaluation of outcome measurements.

It is important to note that the tips and techniques presented in this book represent the personal opinion of the authors and are based on their individual experiences. It is necessary, therefore, to consider variations to the techniques described below; these variations might be specifically designed for certain procedures, or they might reflect the preferences of the individual surgeon.

We want to further highlight that reading this book is far from sufficient to acquire comprehensive knowledge in shoulder arthroscopy. Rather, an extensive period of study, practice, and experience will be needed.

This book is supposed to be a guide for orthopedic residents and fellows who would like to focus on shoulder diseases, but it could also be a reference landmark for expert surgeons already involved in shoulder surgery.

We hope that "*Shoulder Arthroscopy*: *Principles and Practice*" will help the readers in improving their knowledge on shoulder disorders and, subsequently, their arthroscopic techniques and skills. The ultimate goal of our job is taking best care of our patients. A deep knowledge is the only way to achieve this aim.

Rome, Italy — Giuseppe Milano, MD
Rome, Italy — Andrea Grasso, MD

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Part I

Basics

Anatomy of the Shoulder

1

Enrico Bellato, Davide Blonna,
and Filippo Castoldi

When we study arthroscopic anatomy of the shoulder, we must not focus solely on structures visible from inside the joint. Whatever encloses the glenohumeral joint is important, in particular with reference to surgical approaches and portals. Moreover, we have to know the structures to be avoided so that we do not cause damages and complications.

Anatomical Landmarks

Before starting arthroscopy, we have to identify the acromioclavicular joint, the acromion, the scapular spine, and the coracoid process.

Acromioclavicular Joint

This is a diarthrodial joint with an articular disc, usually perforated at its center [1]. It is the only articulation between the clavicle and the scapula. However, 1 % of people have a coracoclavicular joint [2], and about 30 % have articular cartilage on the coracoid and clavicular surfaces without a real joint [3].

Articular surfaces of the acromioclavicular joint are on the medial edge of the acromion and the distal aspect of the clavicle. Superior aspect of the distal clavicle is covered by insertion of the deltoid and trapezius muscles; inferiorly, it is characterized by the coracoclavicular processes where the coracoclavicular ligaments originate: the conoid tubercle medially and the trapezoid line laterally.

The acromioclavicular joint is enveloped by the articular capsule that is thinner inferiorly and by ligaments. The *acromioclavicular ligaments* (capsular ligaments) are responsible for controlling posterior translation of the clavicle, and there are two of them: the superior (which blends with trapezius and deltoid fibers) and inferior (thinner than the superior and sometimes absent) [4]. The coracoclavicular ligaments (Fig. 1.1), on the other hand, control vertical stability [4]. We can distinguish two coracoclavicular ligaments:

- The *conoid ligament* originates from the conoid tubercle, characterized by an inverted cone shape, with a base wider than the surface where the ligament inserts. Variations of the insertion on the coracoid process have been described [5]:
  1. It inserts on the most posterior part of the dorsal aspect of the coracoid process, behind the insertion of the trapezoid ligament; it runs posteriorly up to the highest point of the vertical part of the coracoid, where we can find the "Testut tubercle" (52 %) [6].

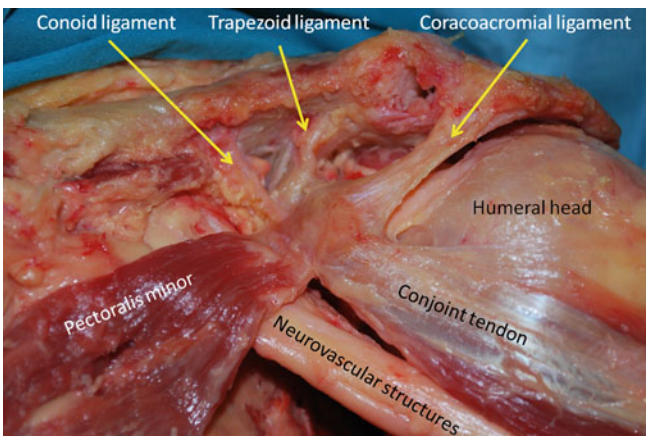

Fig. 1.1 The coracoid origin of the conjoint tendon and of the pectoralis minor tendon; medial to the coracoid, there are important neurovascular structures

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2. It runs posteriorly to join the transverse scapular ligament (33 %).
3. A well-defined accessory conoid ligament, which originates at the base of the coracoid, runs superolaterally and inserts on the clavicle, just lateral to the trapezoid ligament.

- The *trapezoid ligament* originates from the trapezoid line of the clavicle which is three times thicker than the ligament insertion surface on the most posterior aspect of the horizontal part of the coracoid; it takes 15.3 mm from this ligament and the lateral edge of the clavicle [5].

Blood supply to the acromioclavicular joint derives mainly from the acromial artery; innervation is supplied by the pectoral, axillary, and suprascapular nerves [7].

Acromion

It translates as the "highest point of the shoulder," even though the highest point is actually the lateral edge of the clavicle. It is the rectangular extension of the scapular spine.

According to Bigliani et al., the slope of the acromion can present in three different ways [8, 9]: type I "flat," type II "curved," and type III "hooked"; the last one is supposed to be associated with a rotator cuff lesion in 70 % of cases [8, 9]. Recently, an anatomical study found type I in 10.2 % of cases and type II in 89.8 % of cases; the absence of type III may indicate that it is a misinterpretation of the so-called acromial spur [10]. After Bigliani, the acromion was also classified as "cobra shaped" (associated with degenerative changes in 26 % of cases), "square-tipped," and "intermediate" type [11]. The mean distance between acromion and humerus is 9–10 mm (6.6–13.8 mm for males and 7.1–11.9 for females) [12].

Ossification nuclei of the acromion may be not fused by the age of 25, so we can see the so-called os acromiale. This variation was first described by the anatomist Cruveilhier [13] and by the radiologist Lilienfeld [14]. Its frequency is approximately 7–15 % [15, 16], and there are several possible shapes in anterior-posterior order: pre-acromion, meso-acromion, meta-acromion, and basi-acromion. Meso-acromion and meta-acromion are thought to be the most frequent [17].

Moreover, Lilienfeld drew a distinction between "typical" and "atypical" (also known as "secundarium") os acromiale, a term that should be used for displaced acromial ossification centers [14]. This condition is rare (if it really exists) and must not be confused with calcifications due to degenerative or post-traumatic events.

The acromion and the coracoacromial ligaments define the supraspinatus outlet, also called "fornix humeri." The coracoacromial ligament (Fig. 1.1) runs from the coracoid to the acromion edge. It has several different parts: the principal one consists of fibers of the conjoint tendon, the clavipectoral fascia, and the rotator interval. The most lateral aspect originates at the lateral edge of the rotator interval and inserts on the conjoint tendon forming a structure called "falx." Biomechanical studies have shown a tension-band wiring function of the coracoacromial ligament: after a complete lesion of this ligament, the acromion experiences a ten times higher bending force [18].

Spine of the Scapula

It is an oblique process that runs from the medial margin to the upper lateral part of the scapula and becomes gradually thicker; it ends at the acromion process. It is the boundary between the infraspinatus fossa and the supraspinatus fossa and functions as part of insertion of the trapezius and the posterior deltoid. Size and shape of the scapular spine are quite steady, varying less than 1.5 cm from the mean in any dimension [19].

Coracoid Process

The name comes from its similarity to a "crow bill." It comes off the base of the glenoid anteriorly and it hooks laterally; its smooth apex is the insertion surface of the conjoint tendon anteriorly (footprint: 15.5 ± 1.8 mm in width) and of the pectoralis minor tendon medially (footprint: 11.8 ± 2.8 mm in width) [20] (Fig. 1.1). Its dimensions and relationships with ligaments and tendons are fundamental to coracoid osteotomy and transfer, which nowadays can be carried out arthroscopically [21–25]. Coracoid length is about 45.6 mm; width and height in the mid-portion are about 16.1 mm and 13.5 mm, respectively. The mean distance between the tip of the coracoid and the coracoclavicular ligaments insertion is about 28.5 mm. The mean distance from the posterior extent of coracoacromial ligament footprint to the anterior extent of the coracoclavicular ligament footprint is about 2.8 mm, while the mean distance from the posterior extent of pectoralis minor tendon footprint to the anterior extent of the coracoclavicular ligament footprint is about 3.7 mm [20].

Vascularization is also very important: some authors believe that the coracoid process receives blood supply from its apical muscular insertions, and the bony fragment ischemia is held responsible for postoperative complications after coracoid transfer procedures, such as nonunion and bone resorption [26–28]. In a recent study, no vessels were identified at the osteotendinous junction and a complete ischemia of the coracoid follows the osteotomy. The vertical portion of the coracoid is supplied by a branch of the suprascapular artery [29, 30]; the horizontal portion is supplied by a branch of the acromial branch of the thoracoacromial

artery, which runs beneath the coracoacromial ligament and enters next to the insertion of this ligament [29]. A direct branch from the second portion of the axillary artery, behind the pectoralis minor muscle, can seldom be found [29].

The coracoid process is a fundamental landmark for shoulder arthroscopy since the arthroscopic instruments must not go beyond it. In fact just medial to the coracoid, under the pectoralis minor tendon, there are important neurovascular structures such as the axillary artery and the cords of the brachial plexus (Fig. 1.1).

Muscles

Deltoid

This is the most important and largest among the shoulder muscles. Its external boundary is the subcutaneous fat, while the bursa and fascial spaces bound the deep side. The thick and deep fascia hosts vessels and nerves supplying the deltoid. Three portions are identified [31]:

- The anterior deltoid: it originates from the lateral third of the clavicle and from the anterior edge of the acromion.
- The middle third: it originates from the lateral acromion.
- The posterior deltoid: it originates at the posterior edge of the acromion and the spine of the scapula.

These portions differ in structure [32]: the anterior and posterior thirds have parallel fibers, while the middle third is multipennate and stronger. Three collagenous raphae divide these sections. One is anterior and originates from the anterolateral corner of the acromion, between the anterior and middle third of the deltoid. The other two are posterior: one is between the middle and posterior portion of the muscle, about 16 mm medial to the posterolateral corner of the acromion; the second ("mid-deltoid") originates from the posterolateral corner of the acromion and lies within the most posterior aspect of the middle third of the deltoid [33]. These three "classical" portions have been reassessed by different authors [34–37]. Recently, Sakoma et al. [38] identified seven segments, each one characterized by an intramuscular tendon: three posterior, three anterior, and a middle one. Each one is proximally separated from the others by precise landmarks. The middle segment and the second posterior one are bounded by the two acromial corners; within this section of the acromion, we can find two little bony tubercula which divide the lateral border of the acromion into three facets, called anterior, middle, and posterior facets: their widths are 19.5, 14.2, and 17.9 mm, respectively. Thanks to a PET study, the seven segments also seem to represent the functional units of the deltoid [38].

Innervation is supplied by the axillary nerve. Its anterior branch supplies in 100 % of cases the middle and the anterior third of the deltoid and in 18 % of cases also the posterior third; the posterior branch supplies the posterior third in 90 % of cases and the middle third in 38 % of cases. As a consequence, the middle and the posterior portions are doubly supplied in 38 and 18 % of cases, respectively [39]. Vascular supply is mainly derived from the posterior humeral circumflex artery and from the deltoid branch of the thoracoacromial artery [40].

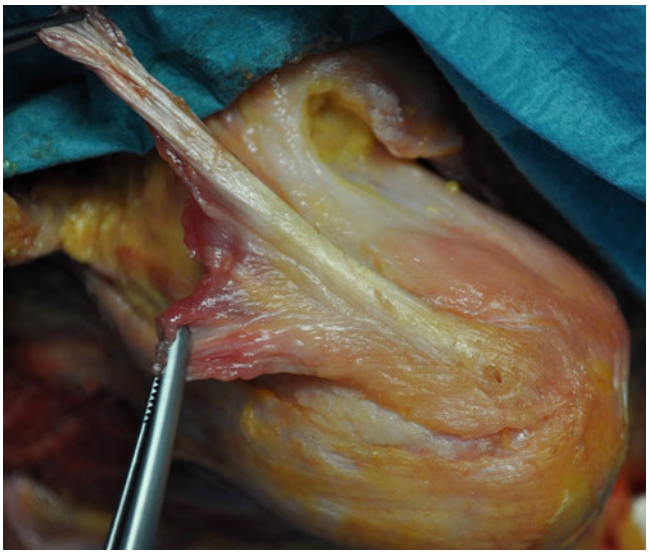

Fig. 1.2 The supraspinatus muscle has an internal tendon within its anterior portion

Rotator Cuff

Supraspinatus

This lies on the superior portion of the scapula and has a fleshy origin from the supraspinatus fossa. Two portions are described [41]:

- Anterior (40 % of the tendon width): it is fusiform and entirely originates at the supraspinatus fossa; within the center of the muscle belly, we find an internal tendon onto which the larger anterior muscle mass inserts (Fig. 1.2) [42, 43].
- Posterior (60 % of the tendon width): it is smaller than the anterior and originates from the spine of the scapula and the glenoid neck; it does not show an internal tendon.

Both of these portions are again divided into superficial, middle, and deep sections, based on fiber orientation and insertion [43].

The tendon runs beneath the coracoacromial arch and inserts into the superior facet of the greater tuberosity of the humerus (Fig. 1.3). It may have an asymptomatic calcium deposit in as many as 2.5 % of shoulders [44]. Inferiorly, the tendon is quite difficult to distinguish from the articular capsule; it is provided with a synovial sheath, which merges into the capsule of the shoulder joint (Fig. 1.4) [45]. The

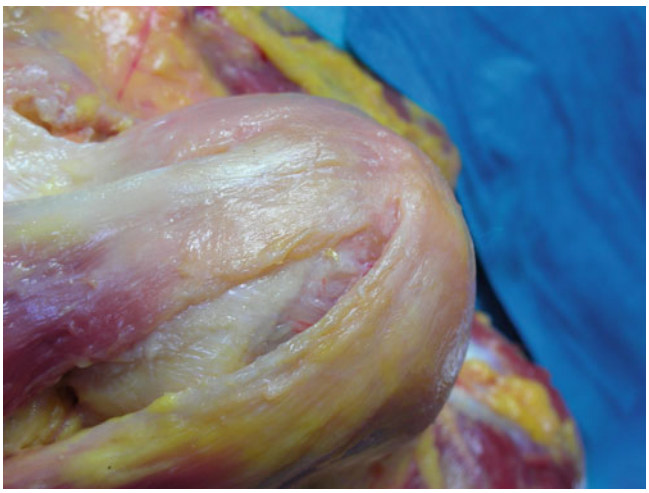

Fig. 1.3 The supraspinatus and infraspinatus tendons

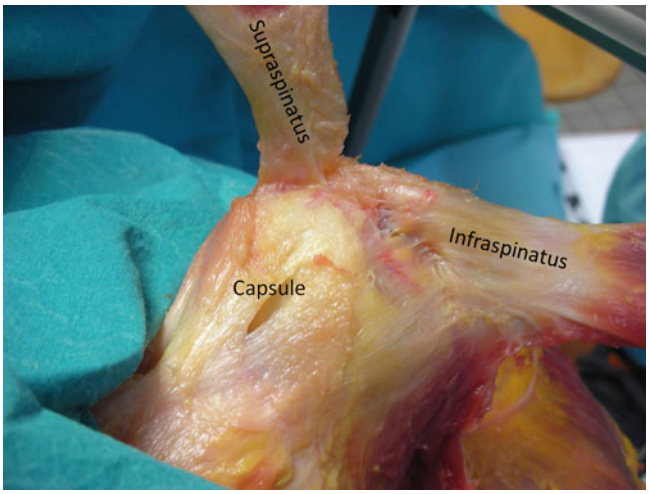

Fig. 1.4 Both the supraspinatus and the infraspinatus tendons are covered by a synovial sheath which merges into the capsule of the shoulder joint

insertional footprint has a trapezoidal or triangular shape with a major proximal base; its average length is 6.9–23 mm and its average width is 12.6–16 mm [46, 47]. The insertional portion of the tendon is characterized by two peculiar structures [48]:

- Crescent: the most distal aspect of the tendon; it is a crescent-shaped sheet of rotator cuff comprising the distal portions of the supraspinatus and infraspinatus insertions; it is very thin, avascular, and so prone to lesions.
- Cable: it is a thick pre-insertional portion (partially involving also the other tendons of the rotator cuff) acting as a suspension bridge capable of stress transfer to protect the crescent; its outer border extends anteriorly to the long head of the biceps and posteriorly to the inferior border of the infraspinatus; some authors stated that it could be an extension of the coracohumeral ligament [49].

Mean tendon width and thickness are 25 mm and 10–12 mm, respectively [50, 51]. It is important to remember the distance between articular cartilage of the humeral head and supraspinatus footprint is about 0.9–1.9 mm [46, 50]: this bare area, which we can calculate during arthroscopy, seems to correlate with partial tendon lesions [46]. Microscopic studies have illustrated a more or less defined layered appearance [46, 52].

Infraspinatus

This is the main external rotator muscle and accounts for 55–60 % of external rotation force [53]. It is a pinnate muscle with a median raphe (often confused with the boundary between infraspinatus and teres minor muscles). It is characterized by three pennate origins in 80 % of cases, bipennate or monopennate origins in 20 % of cases [45]. It has a fleshy origin from the medial aspect of the spine of the scapula and from the infraspinatus fossa. It is sheathed with a dense fascia along with the teres minor; this fascia reflects anteriorly to blend with the fascia of the long head of the triceps. Above the glenohumeral joint, it is 29 mm in width (in neutral rotation) and is characterized by its raphe [33]. As a consequence, this could be considered for the posterior portal as much more suitable and safer than the infraspinatus/teres minor interval (Fig. 1.5) [54]. In fact this interval is often difficult to find, crosses the most inferior margin of the joint, and can host a venous plexus [33]. It has a tendinous insertion into the middle impression of the greater tuberosity, more distal than the supraspinatus footprint (Figs. 1.3 and 1.4). It has a trapezoidal shape with an average maximum length of 10–29 mm and width of 19–32.7 mm [46, 47]. As the supraspinatus muscle, it is sheathed with a synovial sheath which merges into the capsule of the shoulder joint (Fig. 1.4) [45]. During arthroscopy it is difficult to draw a distinction between supraspinatus and infraspinatus muscles. Codman described an area lacking in cartilage, called "rim rent," on the uppermost margin of the anatomic humeral neck: this landmark could be useful to identify the infraspinatus/supraspinatus interval [55]. Minagawa et al. stated that the interval is about 4.3 mm posterior [47].

Both tendons display a layered structure (from the most superficial to the deepest) [46]:

- Superficial fibers of the coracohumeral ligament
- Tendinous fibers parallel to the muscle fibers
- Rare fibers with orientation not well defined
- Flat connective tissue
- Articular capsule

Teres Minor

This is the smallest of the rotator cuff muscles (Fig. 1.5). It originates on the middle portion of the lateral border of the scapula and rarely overlies the infraspinatus as far as the medial border of the scapula [56]. Some fibers originate

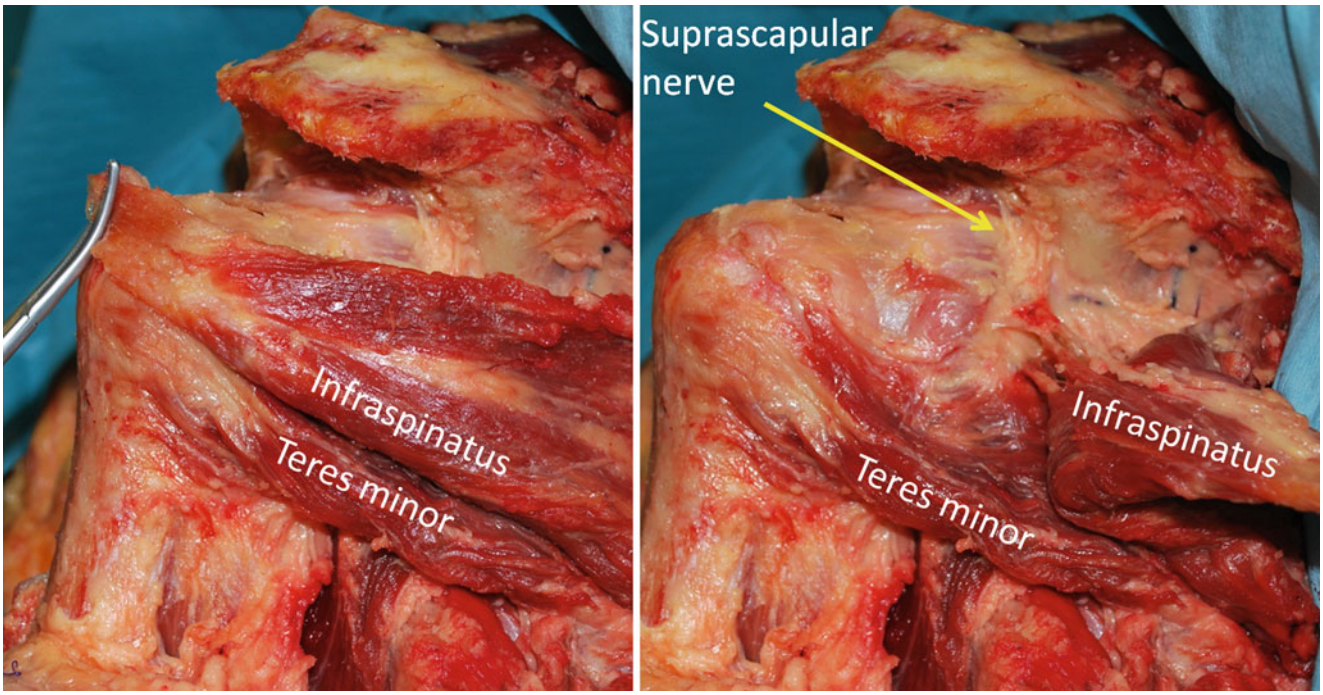

Fig. 1.5 The infraspinatus/teres minor interval crosses the most inferior margin of the joint. The suprascapular nerve is about 1.5–2 cm from the posterior border of the glenoid

from the connective tissue in the infraspinatus/teres minor interval. The tendon inserts into the inferior impression of the greater tuberosity, which displays a triangular shape (average maximum length, 29 mm; average width, 21 mm). It quickly becomes thinner and is characterized by few tendinous fibers superiorly and a fleshy portion inferiorly. The mean distance between the footprint and the articular cartilage is 10 mm [46].

Subscapularis

This is a triangular-shaped multipennate muscle. It has a fleshy origin from the subscapularis fossa. The superior portion (60 %) inserts into the lesser tuberosity with a tendinous insertion, while the inferior portion (40 %) is mainly fleshy and inserts under the lesser tuberosity enclosing humeral head and neck [57].

The subscapularis tendon is 155 mm in length and about 31 mm in width [58]. However, the portion we can see during arthroscopy represents only 26–36 % of the entire tendon (Fig. 1.6) [58, 59].

Many anatomical variations have been described [60–63]. Recently, Staniek and Brenner [64] described the so-called infraglenoid muscle. They identified this structure in 64 % of cadavers: it originates from the upper/lateral third of the lateral border of the scapula and inserts at the crest of the lesser tuberosity (86 %), at the lesser tubercle itself (12 %) or at both anatomical structures (2 %).

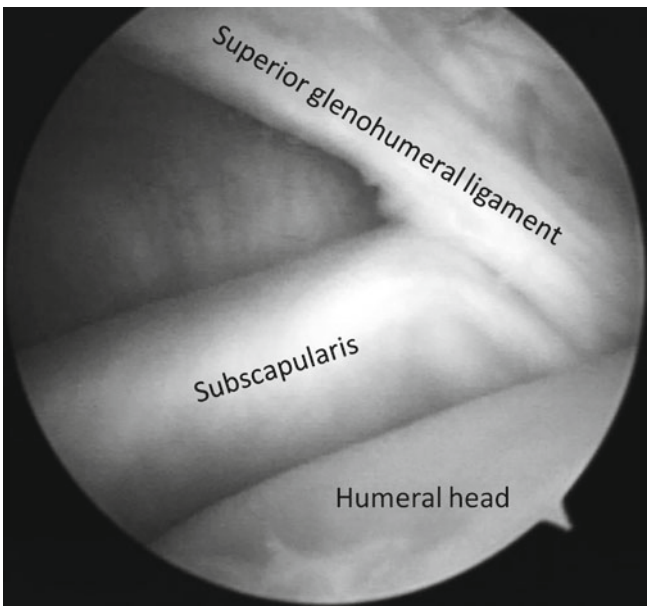

Fig. 1.6 During arthroscopy we can see only 26–36 % of the entire subscapularis tendon. The subscapular recess is between the subscapularis tendon and the superior glenohumeral ligament

Biceps Brachii

Even though it is considered mainly an elbow muscle, the biceps brachii is often involved in shoulder pathologic processes. It has a short head originating from the coracoid tip (laterally to the coracobrachialis muscle) and a

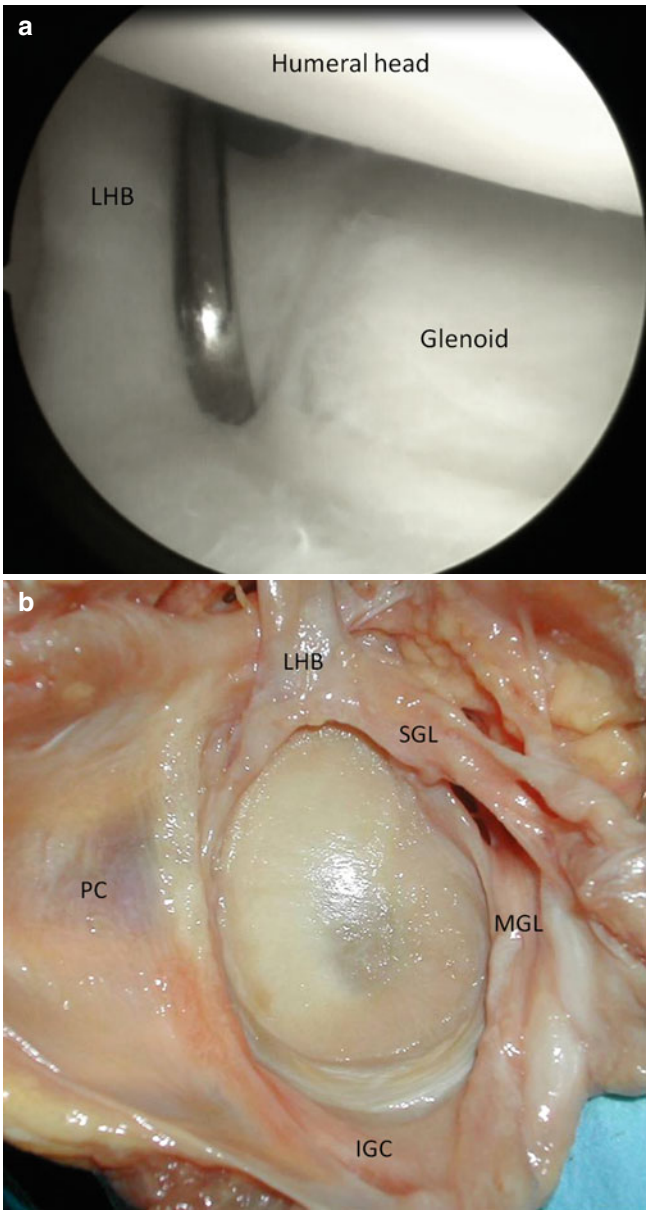

Fig. 1.7 (**a**) The long head of the biceps (*LHB*) originates from both the supraglenoid tubercle and the superior labrum. (**b**) *LHB* long head of the biceps brachii, *SGL* superior glenohumeral ligament, *MGL* middle glenohumeral ligament, *IGC* inferior glenohumeral complex, *PC* posterior capsule

long head (LHB) which originates from both the supraglenoid tubercle and the superior labrum (Fig. 1.7) [65]. Anterior labral attachment is observed in 33 % of cases, posterior labral attachment is seen in 100 % of cases, and isolated posterior labral attachment is seen in 67 % of shoulder joints [66]. Four types of LHB origin have been described [65]:

- Type I: the entire LHB attaches to the posterior labrum.
- Type II: most of the LHB fibers attach to the posterior labrum, but a small portion attaches to the anterior labrum.

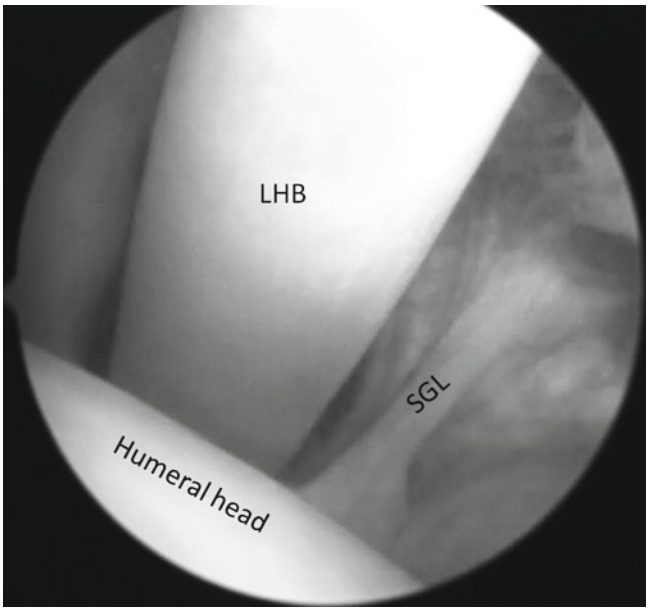

Fig. 1.8 The superior glenohumeral ligament (*SGL*) is one of the four structures forming the "pulley" which stabilizes the long head of the biceps (*LHB*)

- Type III: the LHB attaches equally to the anterior and posterior part of the labrum.
- Type IV: most of the LHB fibers attach to the anterior labrum, but a small portion attaches to the posterior labrum.

Sometimes (25 % of cases, particularly among young patients) we can see small fibrovascular bands of synovium that run from the LHB to the surrounding synovium and capsule; these anatomical variants have been called "vincula biceps" [67].

The intra-articular portion of the LHB (Fig. 1.8) is stabilized by a structure called "pulley" which is medial to the tendon and just above the bicipital groove [68–71]. It consists of four principal structures:

- Coracohumeral ligament: it is characterized by an anterior and posterior portion.
- Superior glenohumeral ligament (Fig. 1.8): it originates from the supraglenoid tubercle; its medial aspect forms a fold parallel to the LHB and laterally it becomes a U-shaped sling; it blends into the coracohumeral ligament, just before the insertion on the lesser tuberosity, forming a sling similar to the ring bends of a finger flexor tendon.
- Fibers of the supraspinatus and subscapularis tendons: these tendon fibers arise from the "fasciculus obliquus," a thin connective structure which runs from the supraspinatus to the subscapularis tendon and helps to build the roof of the rotator cuff interval. The uppermost aspect of the subscapularis tendon is considered by some authors the most important structure to stabilize the LHB [72].

Vascular supply of the tendon has been an interesting object of investigation as particular areas of the LHB are supposed to be prone to rupture because of mechanical or vascular factors. The LHB seems to be characterized by double/triple vascularization:

- One vessel at the osteotendinous junction (the acromial branch of the thoracoacromial artery)
- One vessel at the musculotendinous junction (the most important; a branch of the brachial artery)
- One intermediate mesotendon-derived vessel (rare; a branch of the anterior humeral circumflex artery)

As a consequence, we can see two regions of poor blood supply: the proximal being as much as 1.2–3 cm from the supraglenoid tubercle [73].

Bursae

Many bursae are described in the shoulder. They are totally avascular hollow spaces. The most important are:

1. *Subacromial bursa*: lubricates motion between the rotator cuff and the acromion; it does not usually connect with the glenohumeral joint [74] and has a capacity of 5–10 ml [75].
2. *Subdeltoid bursa*: usually fused with the subacromial bursa.
3. *Subscapularis bursa*: lubricates motion between the subscapularis tendon and the coracoid. It is located between the upper portion of the subscapularis tendon and the glenoid neck and should be more correctly considered a recess of the joint as it actually connects with the glenohumeral joint (Fig. 1.6). Free bodies or inflammatory synovial processes can be found in this recess.
4. *Coracobrachialis bursa*: not always detachable; in 20 % of cases, it is an extension of the subacromial bursa [75].

We can also find other bursae:

- Between the infraspinatus and the capsule
- Between the supraspinatus and the capsule
- Between the coracoid and the capsule
- Between the teres minor and the capsule
- Between the trapezius and the spine of the scapula
- Between the latissimus dorsi and the teres major

Articular Surfaces

The glenohumeral joint is formed by the humeral head and the glenoid fossa. The glenohumeral surfaces have been classified into three types [76]:

- Type A: the humeral surface has a radius of curvature smaller than that of the glenoid → small circular contact area.
- Type B: the humeral and glenoid surfaces have similar radii of curvature → larger circular contact area.
- Type C: the humeral surface has a radius of curvature larger than that of the glenoid → peripheral ring-shaped contact.

Glenoid

The glenoid cavity is slightly concave and measures about 6–8 cm^2 [77]. It is pear-shaped (Fig. 1.7) because of the so-called glenoid notch, well expressed in 55 % of the population [78]. It is covered with hyaline cartilage and displays a thinner central circular portion known as a "bare area." The cartilage is thickest at the periphery (3.81 mm) and thinnest at the center (1.14 mm) [79]. Beneath this thin area of cartilage is an area of subchondral bone thickening termed "Asskay tubercle" [80].

Glenoid vertical axis measures 39 mm, while the horizontal one is about 23–29 mm. Its vertical radius of curvature is usually 2.3 mm larger than the humeral [81]. The glenoid could be either anteverted or retroverted: it is retroverted about 7.5° in 75 % of the population and anteverted about 2–10° in 25 % of cases. It is angled at an average of 15° medially with regard to the scapular plane [76, 82, 83].

The glenoid is completely rounded by the labrum, a circular rim which slightly increases glenoid concavity (Fig. 1.7). It is commonly triangular, but it may also be round, crescent in shape, or blunted [84]. It consists of dense fibrous tissue with a few elastic fibers: we can find fibrocartilage only in a small transitional zone between the labrum and the glenoid bone [85]. Detrisac and Johnson described two anatomical variants [86]:

- Meniscal-shaped: it inserts on the glenoid through a transitional fibrocartilaginous part and is centrally lifted off.
- The labrum inserts centrally and peripherally on all sides.

Cooper et al. [87] stated that the labrum is meniscal-shaped only apparently in its upper portion; in fact this portion inserts directly into the biceps tendon distal to the insertion of the tendon to the supraglenoid tubercle, and the collagen fibers of the labrum and biceps tendon intermingle in this area. Two other important anatomical variants of labrum insertion have been described:

- *Buford complex*: it is detected in 1.5 % of cases; the absence of the anterosuperior labrum is associated with a cord-like middle glenohumeral ligament originating anteriorly to the LHB and running over the subscapularis tendon. It should not be confused with a labrum tear or a SLAP lesion [88].
- *Sublabral hole*: it is detected in 12 % of cases; in the anterosuperior area, there is a hole beneath the labrum insertion [70].

Humeral Head

The humeral head has a wide, almost hemispheric articular surface. It is medially and proximally bent with an

inclination angle of about 137° [89]. It is covered with articular cartilage 1.5–2 mm thick, which is usually thicker in the upper portion [90]. The humeral head is characterized by two "bare areas": one is located in the posterior aspect of the head, between the posterior insertion of the rotator cuff and articular surface; it is 2–3 cm long and usually cannot be seen in young patients; it is probably associated with age-related degenerative processes and must not be confused with Hill-Sachs lesion [91]. The second one is anterior, between the subscapularis footprint and the articular surface, and has a trapezoidal shape [92].

The average radius of curvature of the humeral head is 24 mm in the coronal plane and 22 mm in the axial plane [81]. Articular surface diameter is about 43.3 mm [93]. Humeral head retroversion may be calculated between a line perpendicular to the articular margin plane and the transepicondylar (about 18°) or the tangent elbow axes (about 21–23°) [89, 93].

The anatomical neck of the humeral head is the boundary between the articular surface and the tuberosities. These display different impressions hosting the footprints of the rotator cuff tendons and ligaments [46]. The tuberosities distally continue as the two lips of the bicipital groove. The groove, in its central portion, is about 6.2 mm wide and 5 mm deep [94]. The roof of the groove is formed by:

- Some fibers of the supraspinatus and subscapularis tendon; they form a sheath 7 mm long which envelops and stabilizes the LHB in its middle portion; this sheath is also strengthened by some fibers of the coracohumeral ligament.
- Superior glenohumeral ligament.
- Transverse humeral ligament.
- Falciform ligament: it is the main distal stabilizer, but is not always present; it is a tendinous expansion from the insertion of the sternocostal portion of the pectoralis major tendon.

Shoulder Capsule

The capsule is large: it has twice the surface area of the humeral head. Its normal capacity is 10–15 ml, but this can decrease to 5 ml or less in cases of adhesive capsulitis and increase to 30 ml or more in cases of laxity [95]. It normally extends from the glenoid neck to the anatomical humeral neck. However, its anterior insertion is quite variable and we can see three main types [96]:

- I: the capsule inserts in or next to the labrum
- II: the insertion is at the level of the scapular neck
- III: the insertion is more medial

It is mainly made of types I, III, and V collagen [97] and is strengthened around 360° by the rotator cuff. The tendons blend 2.5 mm into the capsule, especially the subscapularis, and form the so-called musculotendinous/capsulotendinous cuff [95]. Capsule thickness is not uniform, the inferior glenohumeral ligament being the thickest portion and the posterior capsule the thinnest portion (Fig. 1.7). The thickness is mainly determined by the middle collagen layer and ranges from 1.32 to 4.47 mm. The thickest portion is next to the axillary nerve [98].

Ligaments

The ligaments are thickenings of the shoulder capsule.

Coracohumeral Ligament

The coracohumeral ligament (CHL) is believed to represent phylogenetically the old insertion of the pectoralis major, since in 15 % of the population, part of this muscle crosses the coracoid process to insert on the humeral head [99]. It originates from the proximal third of the horizontal part of the coracoid, under the insertion area of the coracoclavicular ligament, and has a variable insertion:

- Superficial fibers insert into the greater tuberosity; only 15–50 % of the fibers insert into the lesser tuberosity; they blend into muscular fibers of the supraspinatus and subscapularis tendons.
- Deep fibers blend into expansions of the subscapularis tendon; only a few fibers run over the LHB and insert into the lesser tuberosity [100, 101].

Anterior border of the CHL is well defined medially, while it blends into the capsule laterally. The posterior border is normally difficult to distinguish. Although it is considered a ligament, its histological features are more similar to capsular tissue [72, 100, 102].

Several authors showed that the CHL is the key ligament which keeps the LHB aligned within the bicipital groove [72, 103–105]. Its contribution to the stability of the joint is very small, best demonstrated with the arm at the side, and consists of a triple function: support of the arm, restraint of external rotation below 60° of abduction, and stabilization of the LHB [103].

Glenohumeral Ligaments

Glenohumeral ligaments are collagenous reinforcements of the capsule (Fig. 1.7).

Superior Glenohumeral Ligament

Superior glenohumeral ligament (SGL) is a fairly constant structure, missing in 5–10 % of the population [91, 106]. However, there is a wide variability in its dimensions and

consistency. The SGL (also called Flood ligament) arises from the superior tubercle of the glenoid, displaying three possible origins [71, 107]: along with the LHB, just anterior to the LHB, and along with the middle glenohumeral ligament. It inserts into the fovea capitis of the humerus, just above the lesser tuberosity. The gap between the superior and the middle glenohumeral ligament is called the Weitbrecht foramen.

SGL contributes very little to static stability of the joint; it develops the most strain at 0° of abduction. It is considered one of the most important stabilizers of the LHB in the bicipital groove [72, 104].

Middle Glenohumeral Ligament

Middle glenohumeral ligament (MGL) is missing in 8–30 % of the population and shows the greatest variations among the glenohumeral ligaments: it can be very thin or as thick as the LHB [106, 108, 109]. MGL originates just beneath or just medial to the SGL and inserts just medial to the lesser tuberosity, under the subscapularis tendon which blends into. Morgan et al. [110] showed the following anatomic variants for MGL:

- Normal (66 %).
- Cord-like (19 %): it is separated from the inferior glenohumeral ligament by a recess or by the so-called Rouviere foramen.
- Fan-like (5 %): it is often associated with a thickening of the anterior band of the inferior glenohumeral ligament.
- Missing (10 %).

MGL stabilizes the joint in a 45° abducted position; additionally, it is tightened over the anterior aspect of the humeral head when a position of extension and 45° of external rotation is held as the arm is abducted from 0° to 90°[71, 103].

Inferior Glenohumeral Ligament

Inferior glenohumeral ligament (IGL) is missing in 7–25 % of population and is the main static stabilizer of the abducted arm [107, 111].

IGL is a hammock-like structure which originates on the inferior aspect of the glenoid and consists of two different structures: the anterior and posterior bands. The anterior band originates on various portions from 2 to 4 o'clock [106, 108]; the posterior band (generally thinner than the anterior one) originates from various portions from 7 to 9 o'clock [108]. Between them there is the axillary pouch or recess. All together these structures identify the so-called inferior glenohumeral complex (IGC) (Fig. 1.9), which inserts on the humeral head in two possible ways:

- "Collar-like" attachment, just under the articular margin
- "V-shaped" attachment, with the anterior and posterior band attaching close to the articular surface and the axillary recess inserting further from the articular edge [95]

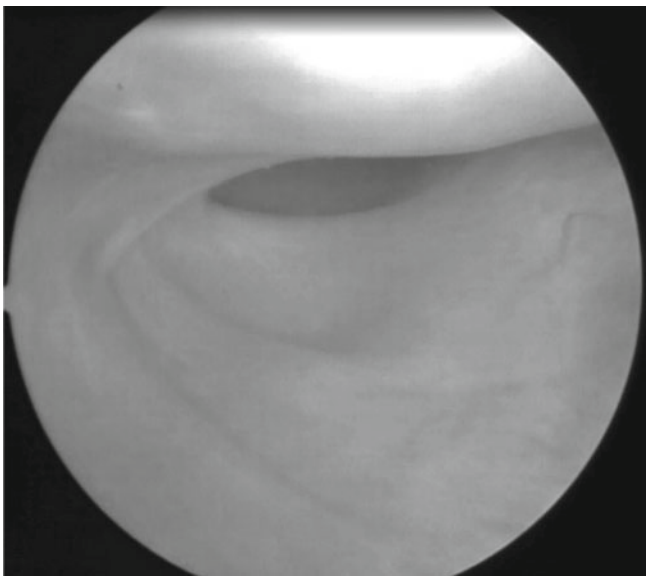

Fig. 1.9 The inferior glenohumeral complex

Rotator Interval

The space between the upper border of the subscapularis tendon and the anterior margin of the supraspinatus tendon is called the rotator interval (RI). It has a triangular shape with its apex above the bicipital groove. The rotator interval consists of four layers [112]:

- The superficial layer of the CHL
- Supraspinatus and subscapularis fibers
- The deep layer of the CHL ligament
- The SGL

The RI is believed to perform different roles. Jost et al. distinguished two regions: a medial one, from the coracoid to the cartilage boundary, controlling the inferior translation with the arm at the side and a lateral one, which covers the humeral head up to the greater tuberosity, controlling the external rotation with the arm at the side [112]. Harryman et al. showed how it stabilizes the shoulder posteroinferiorly and anteroinferiorly in a 60° abducted position [101]. Slatis and Alto underlined its role in the medial stabilization of the LHB [105].

Neurovascular Structures

Axillary Nerve

The axillary nerve runs from the anterior to the posterior aspect of the shoulder through the quadrangular space. Here the nerve is medial to the posterior circumflex humeral artery [33] and can suffer from impingement lesions [113]. It splits into the anterior and posterior branches either within the quadrangular space (65–66 %) or within the posterior

deltoid (33–35 %) [39, 114]. The anterior branch gives off one branch to the joint capsule, one to the anterior portion of the deltoid, one to the middle portion of the deltoid, and one to the posterior third of the deltoid [39]. The posterior branch pierces the deep fascia and develops into different branches:

- One branch to the teres minor (the nearest branch to the glenoid rim) [115]
- The superior lateral brachial cutaneous nerve
- One branch to the posterior portion of the deltoid (78–90 %) [39, 116]
- One branch to the middle portion of the deltoid (38 %) [39]
- One branch to the posterior joint capsule [117]

Some anatomical studies have evaluated the axillary nerve position relative to specific anatomical landmarks:

- Wright et al. stated that it is 32.8 ± 6 mm from the most inferior arthroscopically detectable aspect of the subscapularis tendon [59].
- Lin et al. showed it to be about 45.5 mm from the tip of the greater tuberosity [118].
- Kamineni et al. measured the distance between the nerve and the lateral border of the acromion, both laterally (about 57 mm) and anteriorly (about 51 mm) [119].
- The inferior glenohumeral ligament is on average 2.5 mm from the axillary nerve [115]; moreover, the nerve is closer to the humeral than the glenoid insertion of the capsule and is probably closest in the neutral position and during internal rotation [120].
- The nearest aspect of the glenoid border to the nerve is from 4:30 to 7:00 o'clock [115, 120, 121].

Suprascapular Nerve

Suprascapular nerve is a mixed motor and sensory nerve which typically stems from the fifth and sixth cervical roots [122], even though a contribution from the fourth cervical root has been described [123]. The nerve enters the supraspinatus fossa passing through the suprascapular notch, under the superior transverse scapular ligament (STLS) (Fig. 1.10). This ligament has a variable relationship with the origin of the omohyoid muscle, which is adjacent medial to the ligament in 44 % of cases or partly located on the ligament in 18 % of cases [124]. The suprascapular notch is about 6 mm high and about 8 mm wide [124]. At least five subtypes have been described [125] and type I (U-shaped) is the most common (43.7 %) [126]. The STLS is about 9–11.5 mm long and about 3.5 mm thick [124, 127]. The ligament is missing in 1.5–9.5 % of cases and is substituted by a bony bridge in 3–12.5 % of cases [5, 124, 128, 129]. In 5.7 % of cases, it consists of two distinct parts [130]. Normally the nerve runs under the ligament while the suprascapular artery and vein run over the ligament. However, some anatomical variations

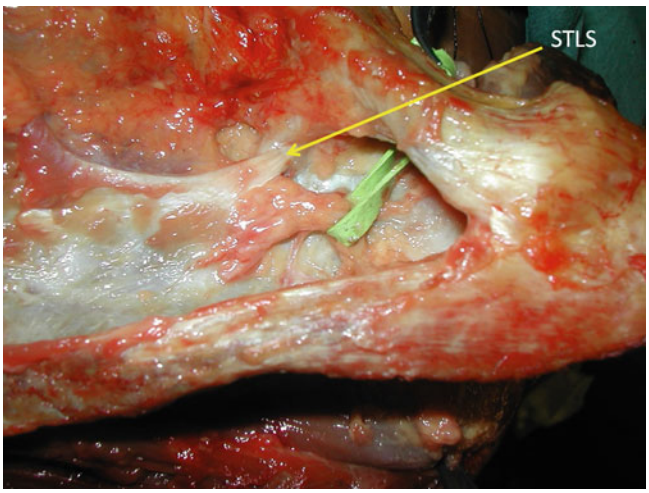

Fig. 1.10 The suprascapular nerve passes under the superior transverse scapular ligament (*STLS*)

are described: in type I (59.4 %) all suprascapular vessels run over the STSL; in type II (29.7 %) the vessels run over and under the STSL simultaneously; in type III (10.9 %) all vessels run under the STSL [124].

After passing through the suprascapular notch, the nerve gives off a motor branch to the infraspinatus muscles and sensory branches to the glenohumeral joint and to the coracoacromial and coracoclavicular ligaments. Then, it runs over the spinoglenoid ligament and enters the infraspinatus fossa. At the spinoglenoid notch, it is about 1.5 cm from the posterior border of the glenoid [33]. The nerve splits into various branches (from four to six) which pierce the infraspinatus muscle generally 2 cm from the articular surface [33] (Fig. 1.5). It does not usually supply the skin; however, some rare branches are described supplying the posterior aspect of the shoulder [131–134].

Musculocutaneous Nerve

It is one of the two terminal branches of the lateral cord of the brachial plexus. It pierces the coracobrachialis muscle as much as 1.5–9 cm from the tip of the coracoid [135–137]. It runs inferiorly supplying the biceps brachii and the brachialis muscle, whose nerves are about 14 cm from the musculocutaneous nerve [138]. We find it again at the elbow as the lateral cutaneous nerve of the forearm.

There are many possible anatomical variations: there can be communications between the musculocutaneous and the median nerve (10–53 %) [139–142]; the nerve can be missing (1.4–5 %) [142–145] or may not pierce the coracobrachialis muscle (7.5–11 %) [142, 143]. Many different ways to classify these anatomical variations have been proposed [143, 146, 147]. Recently, Guerri-Guttenberg and

Ingolotti published a new classification according to four items [142]:

- Presence/absence of the nerve
- Perforation or not of the coracobrachialis muscle
- Presence of communications/fusion or not
- Relationships between the communication and the point of entry of the nerve into the coracobrachialis muscle

Anterior Humeral Circumflex Artery

Anterior humeral circumflex artery originates from the third portion of the axillary artery and runs lateral and parallel to the inferior border of the subscapularis muscle, beneath the LHB, and partially supplies the infraspinatus and subscapularis muscles. It gives off an important branch to the humeral head: the anterolateral ascending artery, which runs along the lateral border of the bicipital groove and pierces the greater tuberosity. The terminal intraosseous portion is called "arcuate artery" because of its shape and gives off many branches supplying the humeral head [148, 149].

Brooks et al. [150] described three intraosseous anastomoses between the arcuate artery and:

- Three to four posteromedial vessels coming from the posterior humeral circumflex artery (These vessels can supply the humeral head despite a lesion of the anterior humeral circumflex artery)
- Metaphyseal vessels
- Vessels of the greater and lesser tuberosities

The authors showed that the arcuate artery alone can supply the whole humeral head. However, after a four-part fracture, blood is mainly supplied by posteromedial vessels [150]. This theory was later confirmed by Hertel [151].

Posterior Humeral Circumflex Artery

Posterior humeral circumflex artery is greater than anterior humeral circumflex artery. It runs lateral to the axillary nerve within the quadrangular space [33] and then splits into two branches:

- Anterior branch: it runs about 5 cm from the acromion and communicates with the acromial branch of the thoracoacromial artery and with the deltoid branch of the brachial artery; moreover, it gives off branches for the glenohumeral joint and for the skin above the middle third of the deltoid.
- Posterior branch: it provides a higher blood supply to the bone than the anterior branch (Fig. 1.11).

Some studies showed that the posterior humeral circumflex artery only supplied the posterior aspect of the greater tuberosity and a tiny posteroinferior portion of the humeral head [148, 149]. More recently, Duparc et al. showed that this is the main vessel supplying the subchondral bone of the humeral head and the greater tuberosity [152].

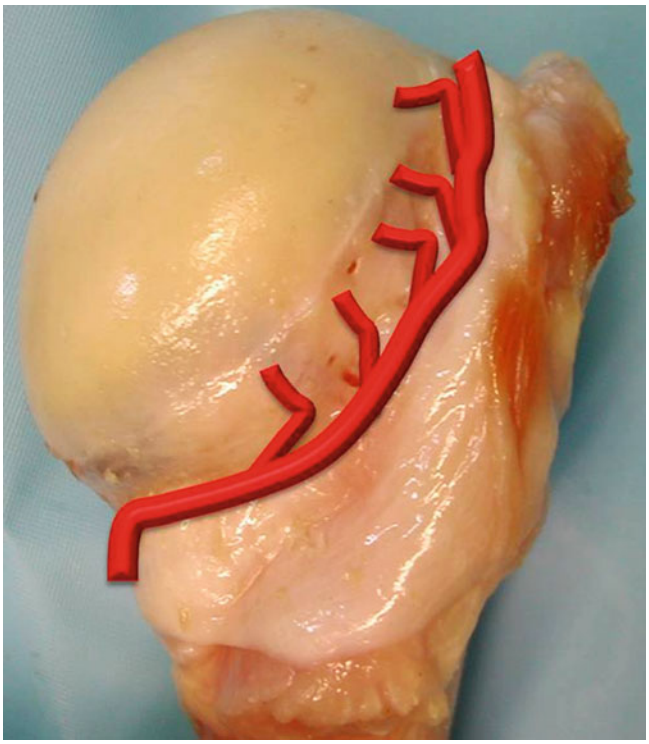

Fig. 1.11 The posterior humeral circumflex artery

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Biomechanics of the Shoulder

2

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Introduction

The glenohumeral (GH) joint has the greatest range of motion of any joint in the human body. As such, it has inherent instability, as its great range of motion is afforded by the lack of bony restraint [1–5]. Its functional structure permits significant rotation while maintaining the humeral center of rotation to within 1–2 mm with respect to the glenoid. Limitation of translation during active shoulder motion occurs through complex interactions between passive structures (ligaments, capsule, labrum, articular surfaces) and active structures (rotator cuff muscles, biceps brachii muscle, deltoid), which produce a concavity-compression effect of the humeral head on the glenoid [6–10]. The humeral head has an articulating surface area that is approximately three times that of the glenoid despite a similar radius of curvature, which means the humerus is only loosely constrained by the glenoid bony anatomy. Simple geometry shows that dislocation of the humeral head from the glenoid fossa should require a translation approximately half of the sum of the glenoid and humeral head axes in the direction of dislocation (Fig. 2.1). In the normal shoulder,

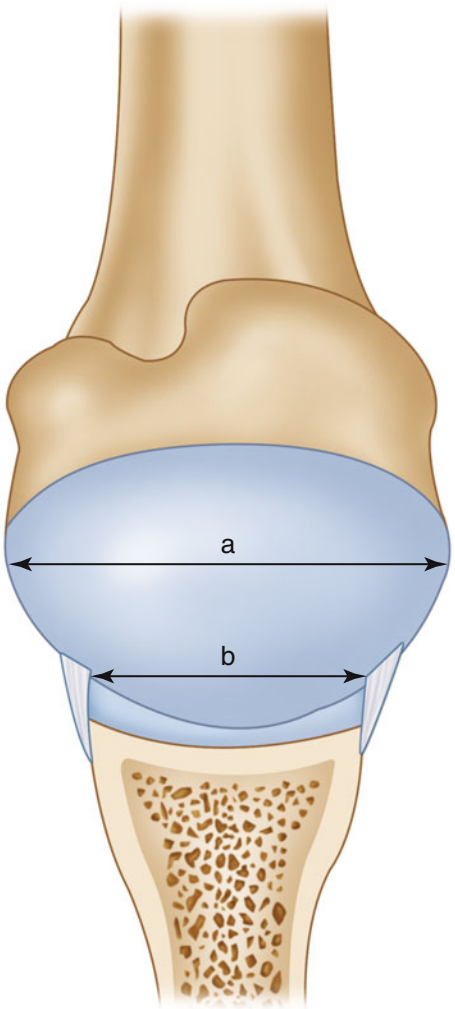

Fig. 2.1 Note the larger humeral head (*a*) as compared to the smaller glenoid articular surface (*b*). The functional structure allows for a large range of motion while the humeral head is maintained in the glenoid fossa by a combination of the dynamic and static stabilizers. Translation of more than one half the sum of the distances of humeral head axis (*a*) and glenoid fossa (*b*) will result in dislocation

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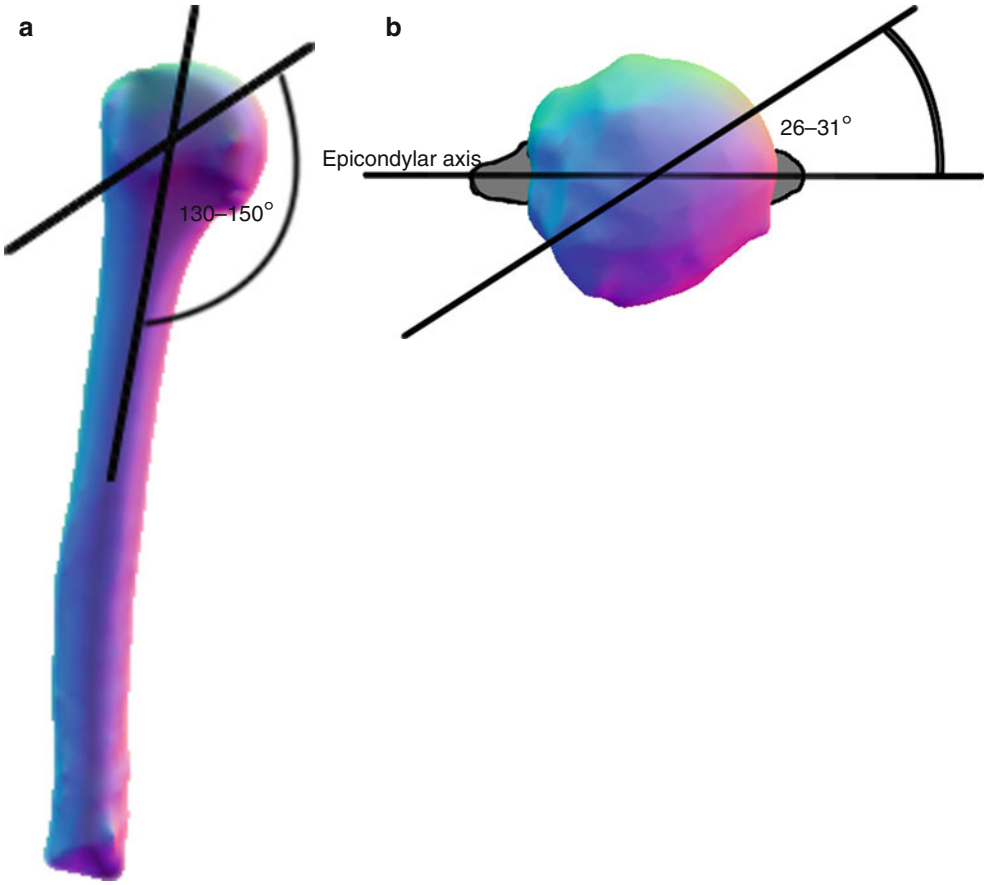


Fig. 2.2 The inclination and version of the humeral head is 130–150° and 26–31°, respectively. Inclination is measured as the angle between humeral shaft and the articular surface of the humeral head in the coronal plane (**a**). Version is measured as the angle between the epicondylar axis distally and the articular surface of the humeral head in the axial plane (**b**)

passive and active stabilizing mechanisms prevent such translations. As the shoulder changes position, different structures are responsible for stabilizing the GH joint. Damage to various anatomic structures can produce shoulder instability through different mechanisms. For example, the mechanics of atraumatic multidirectional instability are usually very different from those of posttraumatic unidirectional anterior instability [5, 7, 11–15].

Anatomy

Humerus

The humerus is the longest bone of the upper extremity and its articulating surface is a hemisphere. The head is inclined relative to the shaft at the anatomical neck at an angle of 130–150° and is retroverted 26–31° from the coronal plane defined by the epicondyles distally (Fig. 2.2). The insertion of the rotator cuff is a continuous crescent interrupted by the bicipital groove, through which the long head of the biceps brachii passes laterally and distally from its origin on the superior aspect of the glenoid [16–19].

Scapula

The scapula forms the posterior aspect of the shoulder girdle and lies atop the posterolateral thoracic cage from ribs 2 through 7. It is a flat, triangular bone with two large surfaces, excluding the articular surface. The glenoid fossa is the bony articulating surface for the humerus, and the superoinferior inclination of the glenoid fossa, known as glenoid tilt, is an important contributor to GH stability [20, 21].

The inclination and version of the glenoid is based upon the medial border of the scapula where it intersects the scapular spine (Fig. 2.3). Normal version ranges from 3° to 11° of retroversion with an average of 7° of retroversion with respect to the scapular plane [22, 23]. Retroversion is associated with anteroposterior stability, while anteversion of more than 5° is found in the majority of unstable joints. However, retroversion of more than 15° is associated with posterior instability. Normal inclination ranges from 7° to 15.8° with an average of 4.2° of superior angulation [24]. This inclination angle is particularly important in preventing inferior translation of the adducted shoulder [11, 23, 25, 26].

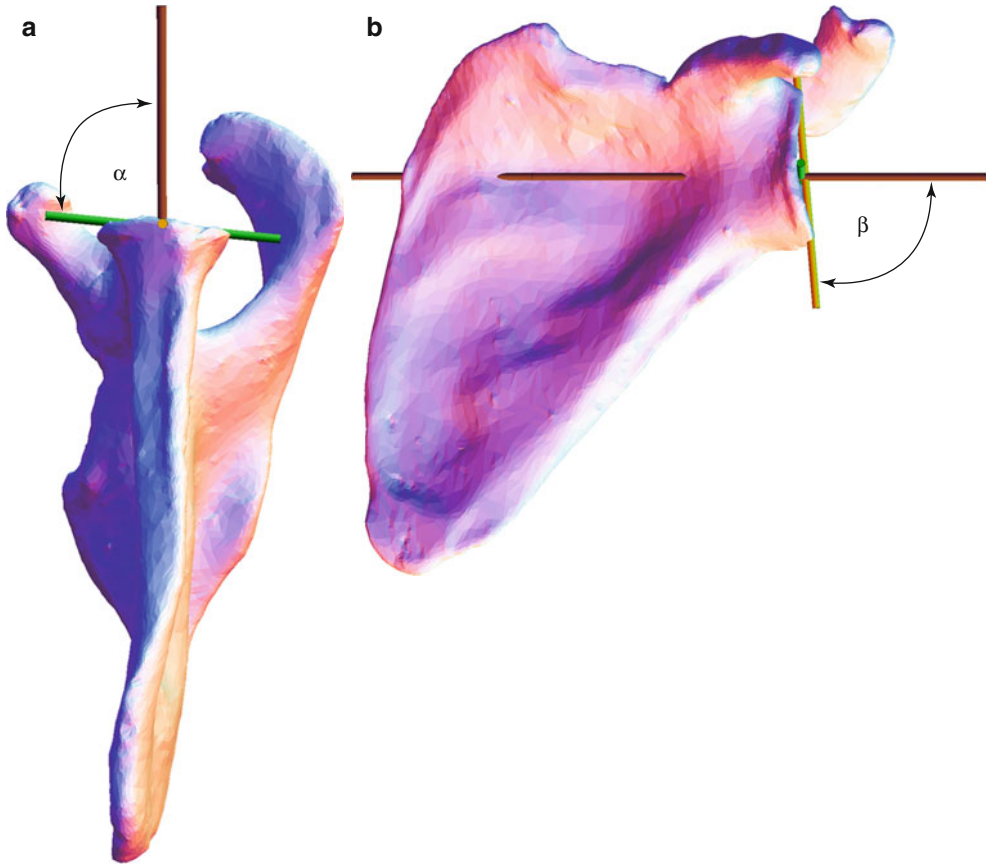


Fig. 2.3 The version and inclination of the scapula is 3–11° and −8° to 16°, respectively. Version is defined as the angle between the line formed by anteroposterior glenoid rim and the line perpendicular to the line formed by the center of the glenoid to the medial border of the scapula at its intersection with the scapular spine in the axial plane (**a**). Inclination is measured as the angle between the line formed by the superoinferior glenoid rim and the line perpendicular to the line formed by the center of the glenoid to the medial border of the scapula at its intersection with the scapular spine in the coronal plane (**b**)

Clavicle

The clavicle is a strut connecting the axial skeleton to the shoulder girdle via the sternoclavicular joint medially and the acromioclavicular joint laterally. The acromioclavicular joint is a diarthrodial joint between the lateral border of the clavicle and the medial edge of the acromion. The clavicle acts as a strut with the axial load transferred to this articulation, which may explain why this joint is often subject to early degenerative changes, especially in people consistently applying high loads.

Scapulothoracic Articulation

The scapulothoracic articulation increases effective arm elevation beyond the 120° of the GH joint. On average, there are 2° of GH elevation for every 1° of scapulothoracic elevation, although the ratio varies within different parts of the arc of motion. The serratus anterior, which maintains the medial angle against the chest wall, and the trapezius, which rotates and elevates the scapula in concert with GH motion, are the two most significant muscles that act upon the scapula [27–30].

Passive Stabilizers

Most investigators attribute a significant passive stabilizing effect to the joint capsule with its discrete ligamentous reinforcements, negative intra-articular pressure, elasticity of the rotator cuff tissue, and fibrous labrum. Indeed, many surgical procedures for the treatment of shoulder instability have been directed at repairing or reconstructing the chock-block of the glenoid labrum and GH joint capsule [31–38]. Differences in capsuloligamentous tension affect translation of the humerus on the glenoid in varying positions of the arm. The GH ligaments are lax in the mid-ranges of rotation. Instead, stability is afforded by a concavity-compression mechanism in which the convex humeral head is compressed into the matched concave articular surface of the glenoid by the shoulder musculature, negative intra-articular capsular pressure, and adhesion-cohesion forces [39, 40].

Articular Surface

The glenoid articular surface is pear-shaped with the anteroposterior width of the inferior half about 20 % larger than

the width of the superior half. The glenoid is narrower and approximately half as deep along its anteroposterior axis compared to its superoinferior axis [41]. The articulating surfaces of the normal humerus and glenoid are nearly spherical, and the two contacting cartilage surfaces have very similar radii of curvature. The GH joint can be modeled as a shallow ball and socket configuration with only small translations of the humeral center of rotation relative to the glenoid at the extremes of motion. Therefore, contact on the glenoid articular surface remains relatively constant, whereas contact on the humeral head is focal and changes according to arm position [13, 14, 41–47].

With its hemispheric humeral head and shallow glenoid articular surface, the GH joint is designed for mobility. At any position, only 25–30 % of the humeral head is in contact with the glenoid fossa. Despite the lack of articulating surface coverage, however, the humeral head moves only 1–2 mm within the center of the glenoid cavity throughout the GH range of motion [17, 48, 49].

Using radiographs to evaluate the radius of curvature of the articular surfaces of the glenoid and humerus does not reflect the true congruity of the GH joint, as the average difference of the bony radii of curvature is more than 30 % or 8 mm [40]. In fact, the articular cartilage at the periphery of the glenoid is thicker than it is centrally, which establishes a highly congruent GH joint surface [40, 50]. Generally, the glenoid and humeral radii of curvature differ by less than 10 %, or within about 2.5 mm of a 25.5 mm radius of curvature [43]. The resultant articular conformity is the foundation for the concavity-compression effect of the shoulder musculature and also serves to restrict translation under physiologic loads to within 2.5 mm in all directions [43, 44].

Negative Intra-articular Pressure

The normal GH joint is fully sealed by the capsule and contains less than 1 mL of synovial fluid. Adhesion and cohesion forces act upon the highly conforming GH joint to impart some resistance to the separation of the glenoid from the humerus [39]. Venting the capsule leads to a significant increase in inferior translation in the adducted shoulder, an effect more apparent in shoulders with a small superior glenohumeral ligament [51]. In experiments, venting the capsule also increases instability by decreasing the amount of force required to translate the humeral head by an average of 55 % for anterior forces, 43 % for posterior forces, and 57 % for inferior forces [39]. It has also been shown that in healthy, stable shoulders, intra-articular pressure decreases with increasing humeral translation, while in unstable shoulders, there is no correlation between intra-articular pressure and humeral head translation. In the unstable shoulders, a Bankart lesion—defined as an injury to the anterior glenoid labrum due to anterior shoulder dislocation—was present [38, 52, 53]. The association was made between an intact labrum and maintenance of intra-articular pressure [54]. However, the passive mechanisms of negative intra-articular pressure, articular congruity, and adhesion-cohesion cannot prevent GH instability at high loads by themselves.

Glenoid Labrum

The labrum is a fibrous structure, triangular in cross section, and firmly attached to the circumference of the glenoid rim. At its attachment on the superior portion of the glenoid, it is redundant and can appear loose, whereas the inferior attachment is tight and smoothly transitions from articular surface to labrum. Therefore, mobility of the labrum above the superoinferior midpoint of the glenoid is normal and variable, whereas mobility below the midway point on the glenoid is abnormal and pathologic [41, 55]. Effectively, the labrum deepens the glenoid socket an average of 9 and 5 mm in the superoinferior and anteroposterior planes, respectively, and the traumatic loss of the integrity of the labrum decreases resistance to translation by approximately 20 % [42, 45, 55]. Loss of labral integrity not only decreases the effective depth of the glenoid but loosens the anchor point of various capsuloligamentous structures. Given the labrum's direct and indirect contributions to stability, Bankart deemed the avulsion of the labrum from the anteroinferior glenoid rim, the "essential lesion" responsible for recurrent anterior dislocations [56]. Here, the labrum is separated from the glenoid rim, and the inferior and middle glenohumeral ligaments, which are firmly attached to the labrum at that point, are also avulsed. Surgical intervention is designed to repair this important structure [56, 57]. However, the superior labrum and its biceps origin should not be ignored. Their importance to stability has been shown, as increased anteroposterior and superoinferior translation in the lower and middle ranges of elevation occurs with injury to these structures [58–60].

Joint Capsule

The joint capsule allows for extensive range of motion and therefore has a much larger surface area than the humeral head. Because the resting position of the arm is next to the body, it is the inferior joint capsule that is usually described as redundant to allow for significant abduction and elevation [61]. At the extremes of motion, different parts of the capsule will become taut. For example, the inferior pouch tightens in abduction and external rotation, thus affording joint stability. Also, the anterior translation of the humeral head is minimal in extreme internal rotation, which seems to be an effect produced by tensioning the posterior capsule [3, 62, 63]. The varying tension on different parts of the capsule and the

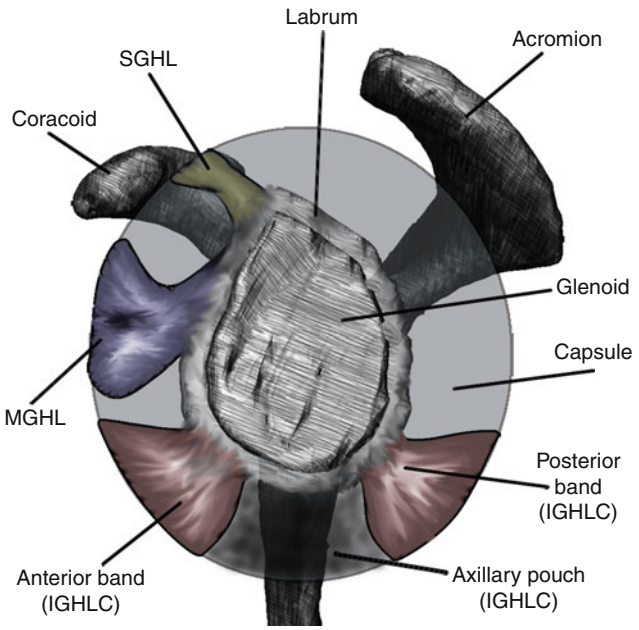


Fig. 2.4 The inferior glenohumeral ligamentous complex (*IGHLC*) is the most important static stabilizer at the extremes of motion. It is formed by the anterior band, axillary pouch, and posterior band. Contributions from the capsule, superior glenohumeral ligament (*SGHL*), and middle glenohumeral ligament (*MGHL*) are also important for anterior, posterior, and inferior stabilization

stabilizing ligaments are functions of their geometry and the position of the arm. In fact, the capsule and GH ligaments constitute a continuous fibrous membrane anatomically, and their mechanical properties are inherently associated [64–66]. In the mid-range of rotational motion, the capsuloligamentous structures are lax, and stability is achieved by other passive and dynamic mechanisms. At the end points of motion, the ligaments become taught and stabilize the joint, and are generally the most significant force preventing translation [1, 15, 43, 61, 67–72].

Glenohumeral Ligaments

The superior glenohumeral ligament (SGHL), middle glenohumeral ligament (MGHL), inferior glenohumeral ligament (IGHL), and coracohumeral ligament (CHL) are thickenings of the GH joint capsule. They are the predominant capsuloligamentous structures responsible for joint stability at the extremes of motion. No single one of these structures stabilizes the GH joint in all positions, and their importance in stability varies with arm position (Fig. 2.4) [1, 11, 15, 18, 19, 27, 42, 43, 49, 61–64, 68–70, 72–77].

The CHL is a thick band of capsular tissue which originates from the base of the lateral coracoid and inserts into the lesser and greater tuberosities. This ligament tightens with the arm in adduction. The CHL and SGHL prevent inferior translation in adduction and posterior translation in forward flexion, adduction, and internal rotation. Some studies suggest that the CHL is important in a suspensory role, but other studies claim that these findings may be inaccurate as the SGHL may have been inadvertently cut while sectioning the CHL. This could lead to the conclusion that the CHL is important in preventing inferior instability when it may not be [1, 15, 43, 78].

The SGHL has a similar function to the CHL and it runs a similar anatomic course. The SGHL extends from the anterosuperior edge of the glenoid to the top of the lesser tuberosity. Together, these ligaments define the rotator interval corresponding to the anterior border of the supraspinatus and the superior border of the subscapularis. While the function of the rotator interval is not clearly defined, it has been suggested that it is important in maintaining negative intra-articular pressure [54, 79, 80].

The structure and properties of the MGHL are the most inconsistent of the three GH ligaments, and it is absent in 8–30 % of people. When present, it originates from the superior labrum, supraglenoid tubercle, or scapular neck and inserts on the medial aspect of the lesser tuberosity. It limits anterior translation of the head in the 60–90° of abduction and inferior translation in adduction. The MGHL and SGHL also prevent anterior translation indirectly by limiting external rotation [4, 61, 81].

The IGHL is the most robust and consistent of the GH ligaments. It has three anatomically distinct regions—an anterior band, axillary pouch, and posterior band. The anterior band is the thickest of the three regions of the IGHL and extends from the anteroinferior labrum and glenoid lip to the lesser tuberosity of the humerus. With the arm in abduction and external rotation, the anterior band moves anterior to the GH joint, its tension increases, and it becomes the primary stabilizer against anterior translation [61, 67, 69, 76].

The anterior band and axillary pouch of the IGHL demonstrate viscoelastic behavior by being stiffer at higher strain rates than at lower strain rates. The proteoglycan content is higher in the anterior band than in the posterior band or axillary pouch. Other biochemical parameters are not statistically different, including water content, collagen, hydroxypyridium crosslinks, and sulfated glycosaminoglycan. The anterior band seems to have the most pronounced fiber bundle interweaving in the mid-substance and insertion sites as compared to the posterior band or axillary pouch [43, 67, 69].

The elasticity of the IGHL varies depending on anatomic region. The IGHL tends to behave elastically in the midsubstance of the ligament, and it behaves viscoelastically near its bony attachments. The anterior band has been shown to accommodate the most strain of the three regions of the IGHL, although all regions demonstrate the ability to sustain significant tensile strain prior to failure [67, 69, 76].

Ligament sectioning studies have shown that at 45° of abduction, the subscapularis muscle, MGHL, and IGHL are the primary GH stabilizers, while at 90° of abduction, the

IGHL is the primary stabilizer. The inferior half of the capsule seems to be more important than the superior half in terms of stability. Sectioning the posterior capsule increases anterior translation during the latter part of abduction. The IGHL and posteroinferior capsule are the primary restraints against anterior dislocation, and the MGHL, when present, is the secondary restraint [4, 61, 81].

In concordance with the ligament sectioning studies, biomechanical strain analysis experiments of the GH capsuloligamentous complex have shown that the IGHL and MGHL show the largest strain at about 45° of abduction, with maximum MGHL strain between 30 and 45°. Similarly, the IGHL distinctly shows the most strain at 90° of abduction. With the arm abducted, the anterior band of the IGHL shows the most strain in external rotation while the posterior band shows the most strain in internal rotation [75, 82]. Some experiments have shown that the SGHL is also important in preventing anterior translation in the abducted, neutrally rotated arm [70]. In all cases, the capsule is a secondary restraint to instability.

Posterior stability is provided by the posterior capsule and IGHL, which have their greatest effect with the arm in abduction, the position in which posterior dislocation usually occurs [83, 84]. Sectioning the posterior capsule, including the posterior band of the IGHL, results in significant posterior translation only with the arm in abduction. However, cadaveric experiments have shown that even with sectioning the posterior capsule, infraspinatus, and teres minor, posterior dislocation will not occur. Additional obliteration of the anterosuperior capsule, including the SGHL, results in posterior dislocation. Sectioning of the anterosuperior capsule and SGHL alone does not result in posterior dislocation. Therefore, it has been suggested that disruption of both the posterior and anterior capsules is necessary to accomplish posterior dislocation (Table 2.1) [3, 63, 70, 85, 86].

Dynamic Stabilizers

Dynamic stabilization is the phenomenon of providing stability to the GH joint through coordinated interactions between muscles that affect it. In general, muscles provide stability through four mechanisms: (1) bulk effect of the muscle itself, (2) contraction causing concavity-compression effect on the articular surfaces, (3) joint motion that secondarily tightens the passive ligamentous restraints, and (4) barrier effect of the contracted muscle [87, 88].

Rotator Cuff and Deltoid

The rotator cuff is a musculotendinous complex that provides stability to the GH joint by compressing the humeral head against the glenoid. Consisting of the supraspinatus, infraspinatus, subscapularis, and teres minor, the rotator cuff muscles originate from the scapula and insert on to the proximal humerus in a radial fashion on its own facet. Specifically, the supraspinatus originates from the supraspinous fossa and inserts on the superior and middle facet of the greater tuberosity. Innervated by the suprascapular nerve, the supraspinatus functions primarily to stabilize the GH joint during abduction of the shoulder, and it secondarily works synergistically with the deltoid as an abductor of the shoulder. The infraspinatus originates from the infraspinous fossa and inserts on the posterior facet of the greater tuberosity. Innervated by the suprascapular nerve, the infraspinatus works with the teres minor to externally rotate the humerus and stabilize the GH joint against posterior subluxation. The teres minor originates from the lateral border of the scapula and inserts on the inferior facet of the greater tuberosity. Innervated by the axillary nerve, it functions as an external rotator and GH stabilizer. Finally, the subscapularis originates from the subscapular fossa and inserts on the lesser tuberosity. Innervated by the upper and lower subscapular nerves, the subscapularis internally rotates the humerus and functions to stabilize the GH joint during abduction. The subscapularis is an important anterior barrier to resist anteroinferior displacement of the humeral head and therefore plays a critical role in GH stability [89, 90] (Fig. 2.5).

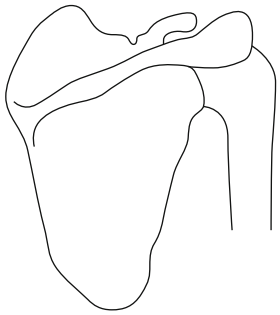
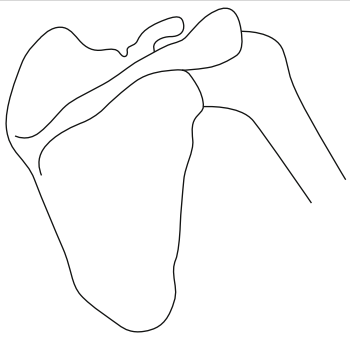
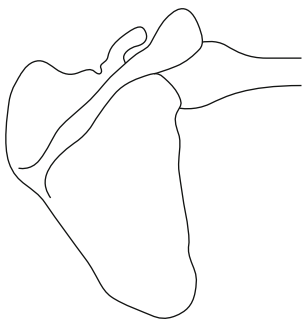
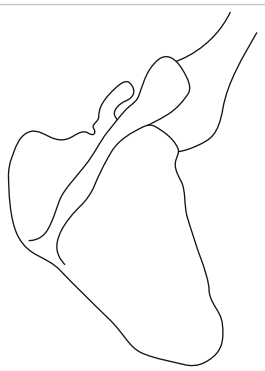
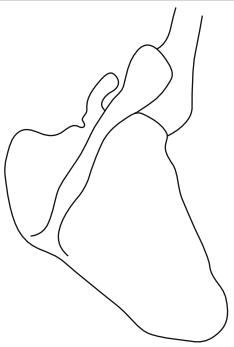
Although static and dynamic factors could potentially operate in all ranges of motion throughout the shoulder, it is thought that the static factors like the capsule and ligaments are primarily responsible at the end-ranges of the shoulder range of motion when under tension. Dynamic factors, like the rotator cuff and deltoid muscles, are primarily responsible in the mid-ranges of the shoulder, when the capsule and ligaments are lax and do not provide any support to the GH joint [11, 91, 92].

The rotator cuff muscles rotate and depress the humeral head during abduction, which is critical for GH stability. The mechanism by which the rotator cuff maintains the humeral head in the glenoid fossa is known as concavity-compression [45, 50]. This is a stabilizing mechanism in which the compression of the humeral head against the glenoid fossa allows for the GH joint to resist shear forces.

The muscle fibers of the rotator cuff primarily run transversely, and the tendons of the muscles form a cuff and surround the joint. They eventually blend intricately with the fibrous capsule. Through its attachments to the capsule, the rotator cuff reinforces the GH joint and functions as an active support structure [42]. The rotator cuff muscles have even been coined “true dynamic ligaments” [93]. Agonistic and antagonistic muscle groups must have coordinated muscle contractions to maintain a stable shoulder joint during movement [8, 11, 45, 94].

Each of the dynamic stabilizers contributes to GH stabilization at different angles of abduction (Table 2.1).

Table 2.1 The primary and secondary stabilizers of the glenohumeral joint during shoulder abduction with neutral rotation

		Anterior stability	Inferior stability	Posterior stability
Rest		Primary: supraspinatus Secondary: SGHL and MGHL (external rotation); posterior capsule (internal rotation)	Primary: superior capsule[a], CHL[a], SGHL[a], supraspinatus, biceps brachii Secondary: proximal 1/3 of anterior capsule (including SGHL and MGHL)	Primary: posterior capsule
0–45°		Primary: subscapularis, MGHL, IGHL	Primary: IGHL, MGHL	Primary: posterior capsule, IGHL
45–90°		Primary: IGHL, posterior-inferior capsule, biceps brachii (internal rotation), rotator cuff muscles, deltoid Secondary: MGHL	Primary: IGHL, MGHL	Primary: posterior capsule, IGHL, biceps brachii (external rotation)
90–135°		Primary: IGHL, axillary pouch, subscapularis, infraspinatus	Glenoid inclination	Primary: posterior capsule, IGHL
135–180°		Primary: IGHL, axillary pouch, Infraspinatus	Glenoid inclination	Primary: posterior capsule, IGHL

[a]The roles of the superior capsule, CHL, and SGHL in inferior stability are inconclusive

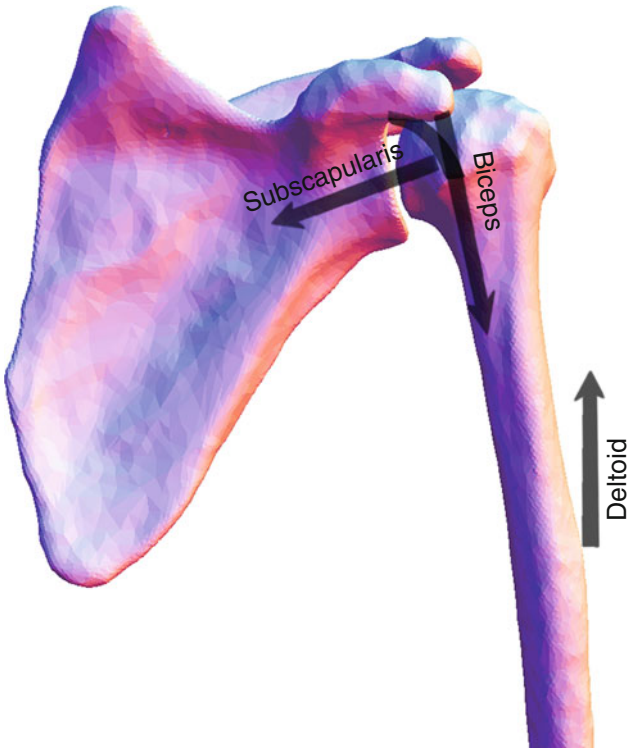


Fig. 2.5 The subscapularis is a major active stabilizer of the shoulder, and its location just anterior to the glenohumeral joint allows it to act as a major anterior stabilizing structure of the humeral head

At Rest

With the arm at the side, the weight of the arm pulling downward is counteracted, and the humeral head is sustained in the glenoid fossa by an isometric contraction by the supraspinatus muscle. This muscle produces the appropriate amount of tension by a spindle system which has motor and sensory fibers connected to the spinal cord [95].

Initial Movement

It has generally been accepted that the synergy of the rotator cuff and the deltoid is required for strong shoulder abduction. When its fibers contract simultaneously, the deltoid abducts the arm along the frontal plane. However, the deltoid does not function to abduct the arm at the initiation of the movement. When the humerus is at 0° of abduction, the deltoid's force of action is nearly vertical. This isolated force would cause upward translation of the humerus and impingement of the soft tissue between the humeral head and acromion [88, 96, 97]. The infraspinatus, subscapularis, and teres minor pull the humerus at the glenoid in a downward direction, which work to compress the humeral head and counterbalance the upward force produced by the deltoid (Fig. 2.6) [7, 98].

A study has shown that the deltoid muscle is still able to complete a full range of abduction despite a paralyzed supraspinatus muscle, but the power of abduction against resistance

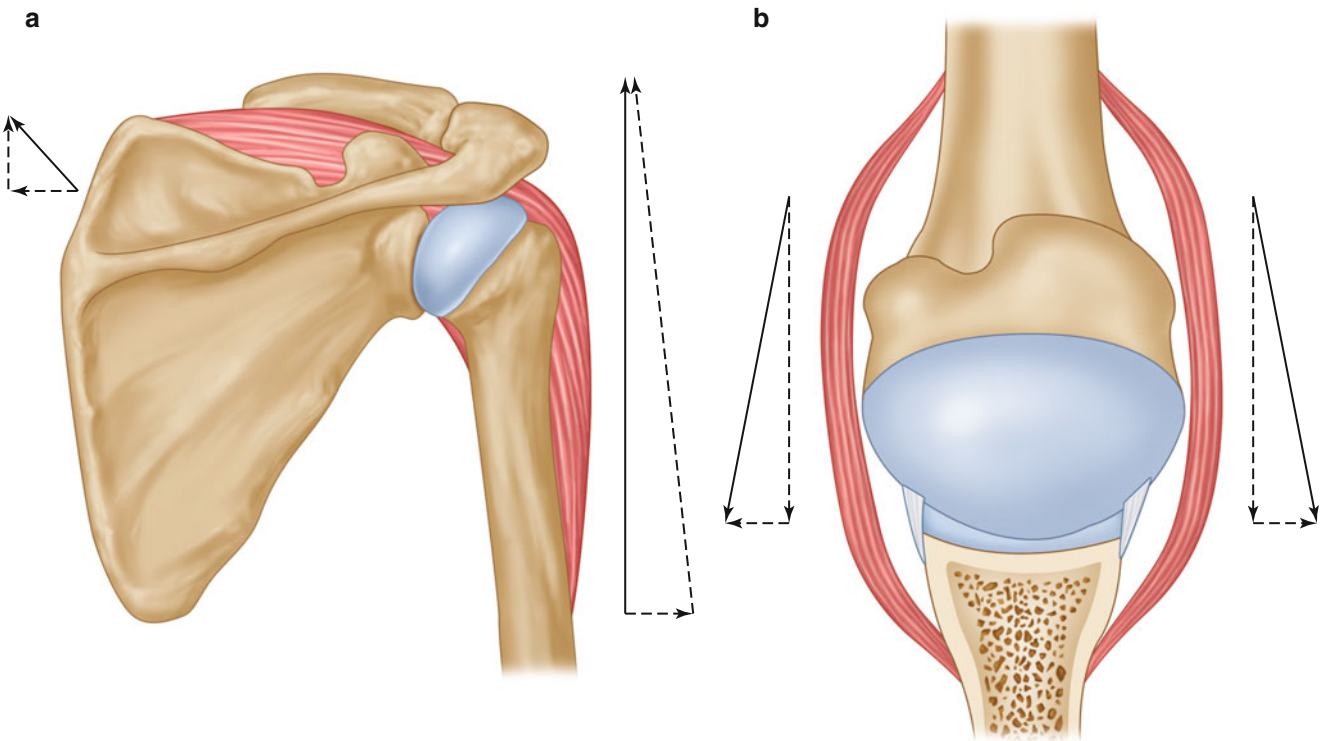


Fig. 2.6 (**a**) The resultant force vectors produced by the deltoid and supraspinatus muscles during abduction. The weight of the humerus is counterbalanced during shoulder abduction by the vertical forces produced by the deltoid and supraspinatus muscles. The supraspinatus also functions to pull the humeral head into the glenoid fossa—a phenomenon known as compressive effect. (**b**) The resultant force vectors produced by the subscapularis and infraspinatus muscles during abduction. Similar to the supraspinatus muscle, the subscapularis and infraspinatus muscles compress the humeral head into the glenoid fossa thereby stabilizing it

is consistently lower. The role of the supraspinatus muscle is to assist the deltoid in abduction to 90° and to stabilize the humerus to allow greater functional strength and stamina of the deltoid muscle [8, 9, 99].

At 0° of abduction, the subscapularis is largely responsible for shoulder joint stabilization, with smaller contributions from the infraspinatus and teres minor [61]. This counteracting force prevents the upward translation of the humeral head and secures it in place during the initiation of shoulder abduction. This phenomenon is an example of force coupling—a sum of forces produced by a group of muscles with differing force vectors resulting in a net moment distinct from the line of action of any one muscle [88, 93].

Midrange Movement

At 45° of abduction, the subscapularis muscle, along with the MGHL and IGHL, continues to bear the primary role of supporting the shoulder [61]. As abduction of the shoulder increases and approaches 90°, the role of the subscapularis and infraspinatus progressively increases. At 90°, the deltoid directs a large part of its force towards the glenoid, which results in the compression of the humeral head against the glenoid. In addition, the rotator cuff muscle fibers are oriented more transversely which, when combined with the forces produced by the deltoid, produce a tremendous compressive force on the humeral head through the glenoid. As abduction continues from 60° to 150°, the power of the subscapularis and the infraspinatus continues to rise [23].

Electromyographic (EMG) activity of the shoulder muscles in patients with generalized joint laxity shows that the activity of the subscapularis muscle is low and the activation speed is slow. The decreased subscapularis muscle activity presumably contributes to the joint instability [100]. Conversely, other studies have observed increased EMG activity of the subscapularis and supraspinatus in patients with generalized joint laxity. The increased subscapularis muscle activity is thought to compensate for capsuloligamentous laxity [101]. Overall, the role of the subscapularis muscle has generally been accepted to stabilize the shoulder anteriorly with the arm in abduction and neutral rotation. It becomes less important with external rotation, in which position the posterior cuff muscles reduce anterior strain.

End-Range Movement

As abduction of the humerus continues past 150°, the power of subscapularis shows a rapid decline, but the power of the infraspinatus continues to rise from 150° to 180° [23]. In the upper ranges of elevation, the axillary pouch of the IGHL stabilizes and supports the GH joint.

Biceps Brachii

The long and short heads of the biceps muscle are contributors to dynamic stability of the GH joint [9, 58, 102–104]. They are particularly important in the stabilization of both anterior and superior translation of the humeral head. The long head of the biceps originates from the superior glenoid labrum, and the tendon travels within the joint and anteriorly on the humerus through the bicipital groove. Like the rotator cuff, the long head of the biceps tendon lies in close proximity with the GH joint, making it anatomically ideal to act as a dynamic shoulder stabilizer. It is important to note the direction of the long head of biceps tendon force is considered two components—one perpendicular and the other transverse to the glenoid surface [58]. The effectiveness of the long head of the biceps in stabilizing the GH joint depends on arm position. The short head of the biceps originates from the coracoid process and travels along the humerus and joins the long head of the biceps to form the biceps brachii muscle.

The roles of the short and long heads of the biceps in anterior stability are particularly important when the arm is in abduction and external rotation [9, 58, 102–104]. The compressive effect and barrier effect of the long head of the biceps depend on joint orientation which determines the line of action of the biceps tendon. At neutral rotation, the tendon lies in a slightly anterior position. With internal rotation, the tendon lies anterior to the joint. With external rotation, it lies posteriorly. Therefore, the observed anterior stabilization offered by the biceps occurs when the arm is internally rotated and posterior stabilization occurs when the arm is externally rotated [58, 105]. The stabilizing effect of the biceps is largest in the lower and middle abduction angles. The short head of the biceps tendon, however, works as a GH stabilizer through a different mechanism—it functions primarily as a physical barrier to prevent anterior translation of the humeral head. The short head of the biceps always lies anterior to the humeral head and therefore prevents excessive translation of the humeral head when it moves anteriorly and comes into contact with the tendon (Fig. 2.7) [58, 105].

One study explored the relationship between the passive stabilization of the IGHL and the active stabilization by the biceps tendon. When the arm was placed in abduction and external rotation, the most vulnerable position for anterior dislocation, the transection of the long head of the biceps tendon resulted in an increase in IGHL strain. This increase in ligament strain could presumably contribute to instability. It was postulated that the long head of the biceps tendon maintains GH joint stability by resisting torsional forces on the humerus and that it does so by acting as an internal rotator in abduction and external rotation [106].

The role of the biceps in the shoulder is still controversial. Some studies claim that the long head of the biceps acts as a shoulder flexor and abductor, while others claim it works to externally and internally rotate the humerus [60]. It is thought that the biceps serves to dynamically stabilize the GH joint, particularly in humeral abduction and external rotation. The role of biceps in stabilization increases as GH joint stability decreases [60].

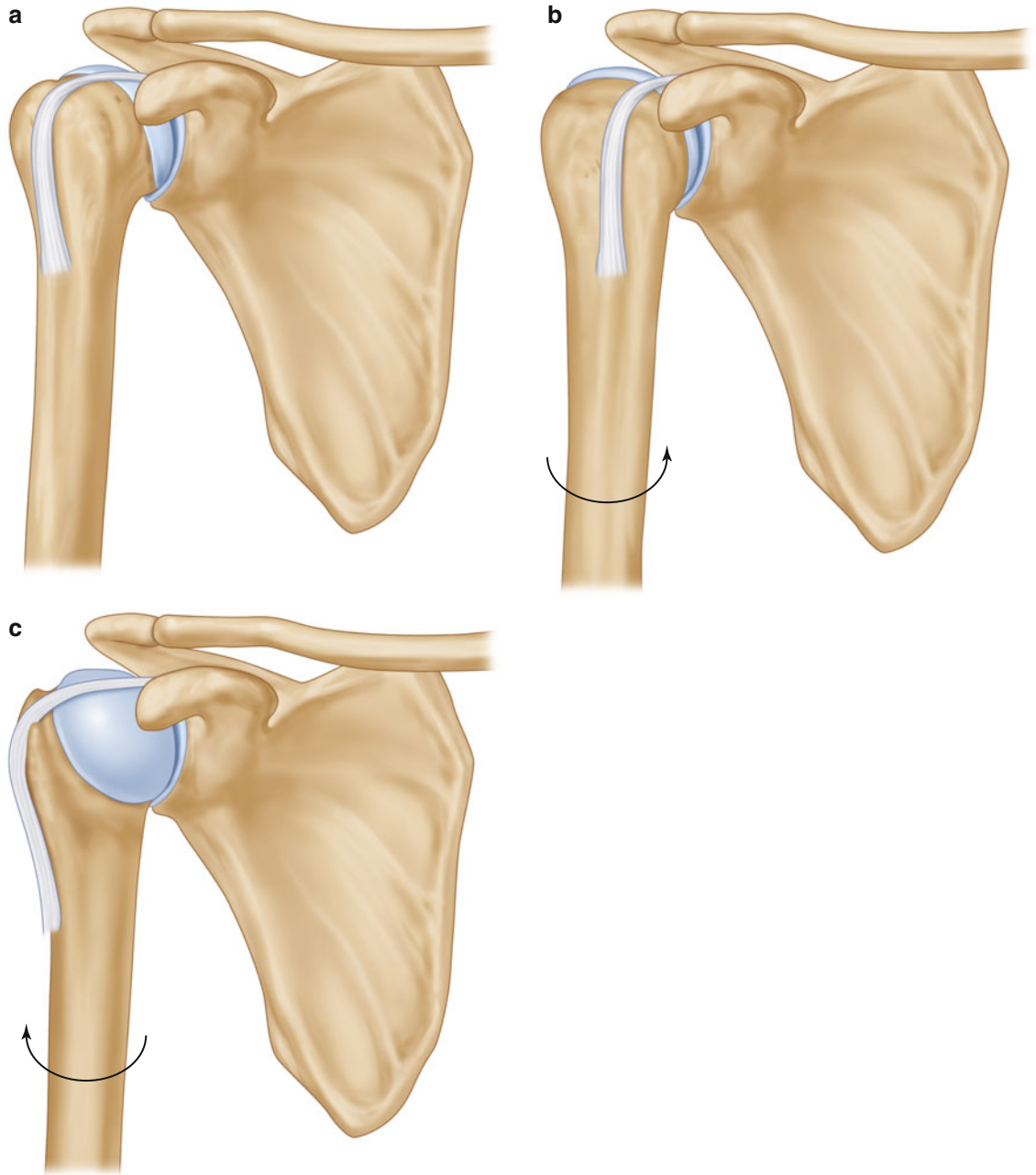


Fig. 2.7 The relationship between the long head of the biceps and the humeral head in neutral (**a**), internal (**b**), and external (**c**) rotations. Note as the humerus is internally rotated, the long head of the biceps lies in an anterior position, thereby restraining anterior humeral head translation. Conversely, the long head of the biceps acts as a posterior stabilizer when the humerus is externally rotated

Scapula

The scapula itself serves four functions: (1) provides a receptacle for the humeral head, (2) connects the body and the arm, (3) serves as a base for muscle attachment, and (4) orients the glenoid to increase range of motion available to the upper limb and thereby increases mobility [107]. The scapular rotators ensure the proper positioning of the scapula, which is crucial to optimize the length-tension relationship of the muscles for shoulder movement [107]. The scapular plane lies in approximately 35° of anteversion in relation to the coronal plane of the body [23]. This positioning of the scapula allows it to achieve proper balance of force couples and ensure dynamic stabilization of the shoulder throughout the entire range of motion. In essence, the scapula optimizes the contact between the humeral head and glenoid to ensure stability; mechanical stability is achieved by bringing the glenoid fossa directly under the head of the humerus [23, 96, 107].

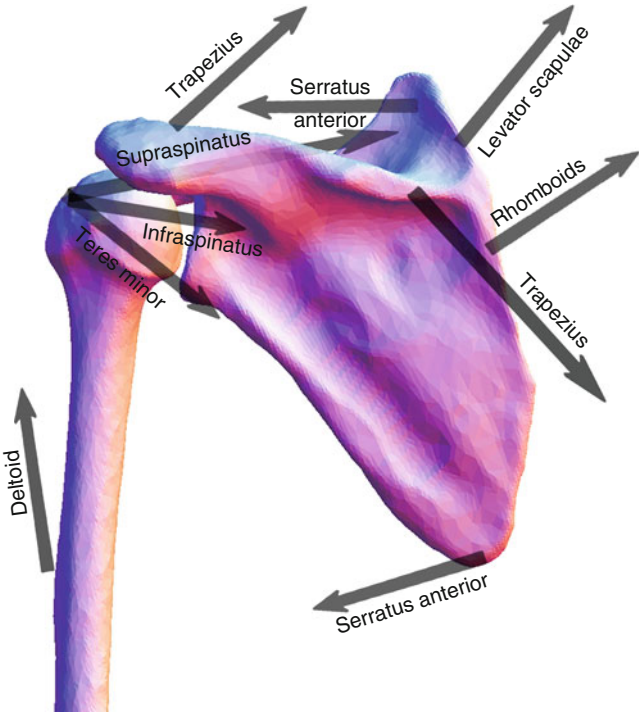


Fig. 2.8 The directions of force produced by the muscles acting on the humerus and scapula. The scapular rotators position the scapula to achieve motions with efficient biomechanics to allow for optimum shoulder function. The coordinated movements of the humerus and scapula are essential to provide stability to the glenohumeral joint by keeping the joint angle within a physiological range

Scapula Rotators

The scapular rotators are composed of the trapezius, rhomboids, latissimus dorsi, serratus anterior, and levator scapulae. Force coupling of these muscles is necessary to allow for active range of motion. The upper portion of the trapezius acts on the acromion in a medial direction while the serratus anterior produces a rotary force from the inferior angle of the scapula in a lateral direction [107]. The combination of these two forces rotates the scapula and is responsible for a significant part of total arm elevation . Rotation of the the scapula also allows for full abduction of the arm while avoiding impingement of the acromion upon the rotator cuff [108] (Fig. 2.8).

Glenoid Tilt

The glenoid articular surface primarily rotates in the coronal plane. Therefore, vertical stability does not depend as much on the vertical tilt of the glenoid surface. However, studies have shown the stability of the GH joint increases proportionally with the inclination of the glenoid and the relationship between the slope of the glenoid and the mechanical stability of the GH joint is linear in the posteroinferior direction [26, 109]. Inferior stability increases with a superior tilt of more than 10° [26]. In rare cases where excess inferior tilt of the glenoid leads to vertical instability, patients can often voluntarily dislocate their GH joint downward [23].

Scapulohumeral Rhythm

Scapular motion is particularly important during shoulder abduction and flexion. This motion is known as scapulohumeral rhythm [93]. Measurement of this motion shows that the ratio of GH movement to scapulothoracic movement is 2:1 during abduction. As the GH joint abducts, the scapula rotates upward to allow for full arm elevation and to maintain a position of stability.

The ability to control and coordinate the movement of the scapula in relation to the humerus is essential for stability of the GH joint. Improper movement of the scapula causes misalignment of the humeral head with the glenoid and contributes to shoulder instability [23, 96, 107].

Proprioception

Proprioception is the sense of the relative positions of parts of the body, and it helps to prevent excessive strain in capsuloligamentous structures of the shoulder. It is thought that damage to the soft tissue structures around the shoulder may also disrupt the proprioceptive capabilities of the ligaments, which may contribute to shoulder instability. Previous studies have shown that GH dislocation results in abnormal neuromuscular coordination and increases the likelihood of subsequent reinjury to the shoulder [110]. When comparing individuals with normal, unstable, and surgically repaired shoulders, proprioception is impaired in patients with GH instability. Interestingly, this feedback seems to be restored in surgically repaired shoulders [111].

The proprioceptive feedback mechanism is still not completely understood. Pacinian corpuscles, Ruffini endings, Golgi tendon endings, and other mechanoreceptors have been identified in the glenoid labrum and GH ligaments, which confirms the idea that capsuloligamentous structures of the shoulder have the potential to perceive relative positioning [112]. However, there have not been rigorous studies that qualify the role of proprioception in shoulder stability.

Summary

GH instability represents a broad range of pathology which can involve many anatomic structures. Static stabilizers, including the glenoid labrum, the GH capsule, the three GH

ligaments, intra-articular pressure, congruity of the joint surface, and adhesion-cohesion forces, are critical to providing passive stabilization. Dynamic stabilizers which include the rotator cuff, biceps brachii muscle, scapular rotators, and glenoid tilt are all important in active stabilization. The sophisticated movements of the shoulder require the delicate balance between the static and dynamic restraints to maintain stability during movement through a wide range of motion.

Shoulder dislocation is associated with disruption or permanent stretching of the GH capsular ligaments. In elderly patients, dislocation is frequently associated with rotator cuff tears. Muscular dysfunction may predispose to instability which is commonly seen in patients who perform repetitive throwing motions or overhead activities. Conversely, capsuloligamentous instability may result in muscular pathology as stabilizing musculature becomes unable to compensate for disrupted or loose static stabilizers. It is clear, however, that the disruption of the IGHL is the most commonly injured component of the capsule, with tissue disruption ranging from plastic deformation, mid-substance tear, or avulsion of the capsuloligamentous complex from its bony attachment site. GH capsular stretch and the resulting laxity are a key feature of shoulder instability and are a major contributor to recurrent dislocations [43, 74, 91].

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Shoulder Examination

3

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Introduction

Shoulder disease ranks among the most common musculoskeletal disorders, being the third cause of musculoskeletal disease (16 %), after the back (23 %) and knee (19 %) [1].

They represent one of the main reasons for orthopedic consult. Modern imaging studies help the specialists in the diagnosis, but an accurate history and physical examination should be the first step to determine which pathology is primarily responsible for the patient's complaints.

The shoulder is one of the most complex joints in the human body. Its evaluation is a challenge because different joints participate to the motion and direct observation of those simultaneous movements is obscured by muscles [2].

Physical examination consists of different phases: inspection and palpation, evaluation of joint motion, and specific tests. Depending on the suspected pathology, the clinician should perform tests to assess shoulder stability, rotator cuff deficiency, impingement, biceps tendon diseases, and SLAP lesion.

It is necessary to premise that many tests are eponymous, and several investigators have described more than one test leading to confusion [3]. Moreover, some tests are not sufficiently evaluated and rarely compared with a diagnostic gold standard (e.g., arthroscopic or open-shoulder surgery) [2]. Misquoting or misinterpretation of tests by subsequent investigators has compounded this problem. For these reasons, it is important to know the proper way to perform tests, as well as their sensibility and specificity, in order to rationally use them to assess shoulder diseases.

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Inspection and Palpation

Shoulder examination starts with the inspection and palpation. Both shoulders should be observed in order to identify deformity of the clavicle and difference in shoulder height. A prominent acromioclavicular joint is often secondary to osteoarthritis, while a prominent sternoclavicular joint can also be due to anterior dislocation, inflammation of the synovium, infection, or condensing osteitis. Difference in shoulder height can be correlated with scapulothoracic or glenohumeral problems. Scapular positioning is observed at rest, comparing the medial border and the inferior edge with that of the unaffected shoulder. Scapular asymmetry has been described in athletes (tennis shoulder or protracted scapula) [4–6], and some authors suggested it predisposes to rotator cuff impingement [7]. Scapular winging (prominence), resulting from lesion of the spinal accessory nerve (lateral) or lesion of the long thoracic nerve (medial), is accentuated by forward flexion of both arms to 90° [8, 9]. Evaluation of scapular motion is also important to screen for dyskinesis [10, 11].

Muscle inspection requires evaluation at rest and during motion. It will allow to better point out deficiencies in the posterior or middle deltoid. Atrophy of the muscle in the supraspinatus fossa or below the spine of the scapula is typical of patients with chronic rotator cuff tears. Palpation of the muscle belly is useful to distinguish a pathologic muscle contraction. It is needed especially for patients with large amount of subcutaneous tissue.

The sternoclavicular and acromioclavicular joints, the acromion, the greater tuberosity, the bicipital groove, the trapezius, the superior-medial tip of the scapula, and the posterior glenohumeral joint line are palpated for deformity and tenderness.

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Joint Motion

All joints around the shoulder girdle (glenohumeral, scapulothoracic, acromioclavicular, and sternoclavicular) participate to the shoulder motion. In measuring the ROM, the total motions are recorded. The unaffected shoulder should be examined at first to compare the deficit. Both passive and active motion should be valued in the cardinal planes: elevation in the scapular plane, external rotation with the elbow at the side, and internal rotation. In patients with isolated rotator cuff pathology, only active motion is affected.

Stability is assessed by external and internal rotation with the arm in 90° of abduction.

Stability Assessment

Specific tests can be used to diagnose pathologic instability. They include sulcus test, glenohumeral translation, anterior and posterior drawer tests [12], apprehension tests [13], relocation test [14], release test [15], and jerk test.

Sulcus Test

The *sulcus test* is performed with the arm adducted. The arm is pulled down by the examiner who measures the translation of the humeral head from the acromion (1+ for 1 cm, 2+ for 2 cm).

Glenohumeral Translation

The glenohumeral translation is assessed with the arm at rest and the examiner stabilizing the scapula. The examiner, with the other hand, applies an anterior and posterior translation force to the humeral head and defines the percentage of subluxation (Fig. 3.1). The test can be repeated for different degrees of internal and external rotation as well as different degrees of abduction.

Drawer Tests

The *drawer test* is performed with the patient supine and the arm abducted to 60° [12]. The examiner applies an axial force to hold the arm in neutral rotation and, with the other hand, translate the humeral head both anteriorly and posteriorly. Grade I is a translation over the rim, grade II is a translation that spontaneously reduces, and grade III is a dislocation without spontaneous reduction.

Fig. 3.1 Assessment of humeral head translation on the glenoid

Apprehension, Relocation, and Release Tests

The *apprehension test* is performed with the patient supine [13]. The examiner abducts the arm to 90° and slowly externally rotates the shoulder to 90° (Fig. 3.2). The test is positive if it causes any apprehension. From this position the *relocation test* can be performed [14]. The examiner applies a posterior force against the humeral head (Fig. 3.3). It moves from an anteriorly subluxed position to the center of the glenoid. The test is positive if the maneuver decreases patient's apprehension.

The sensitivity and specificity of these tests were reported to be 72 and 96 % for the apprehension test and 81 and 92 % for the relocation test [16].

The *release test*, or *surprise test*, consists in the sudden removal of the posteriorly directed force from the relocation test [15]. This maneuver stresses maximally the anterior structures. The test is positive when the patient feels again apprehension. This test has been reported as the most

Fig. 3.2 The position of apprehension for patients with anterior instability

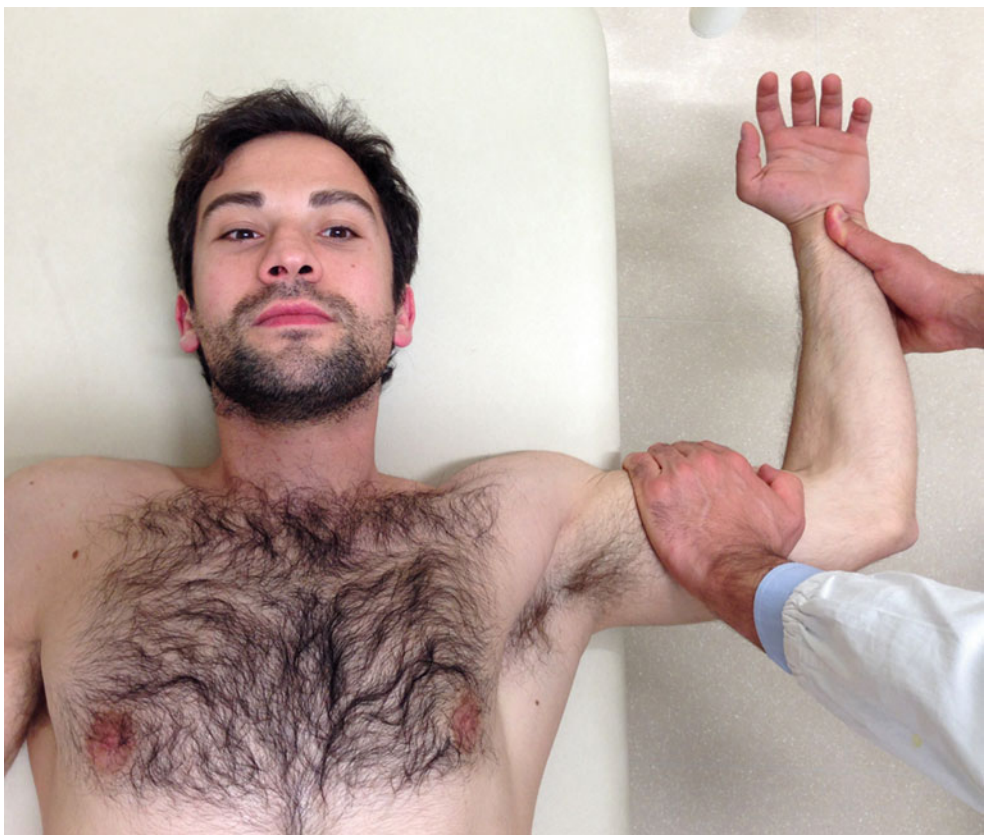


Fig. 3.3 The relocation test

accurate individual examination maneuver [17]. However, the predictability of anterior glenohumeral instability is higher when all three tests are positive.

Jerk Test

Posterior instability is valued by the *jerk test*, also known as *Jahnke test* or *posterior load test*. It is performed with the patient standing or sitting. The examiner holds patient's elbow with one hand and stabilizes the scapula with the other hand. The shoulder is flexed to 90°, internally rotated and adducted, applying a posterior load. The test is positive when the humeral head relocates into the glenoid determining a clunk.

Rotator Cuff Tests

Several clinical tests have been described to investigate rotator cuff deficiency. It is not feasible to use all of them at every examination. They should be used selectively and should be tailored to the clinical condition suspected [18]. However, there is lack of consensus from the available literature on the contribution of each test in the differential diagnosis of shoulder pain [3]. Only few tests show both high sensitivity and specificity. The reason for poor accuracy may be related to close relationships of structures in the shoulder [19], lack of understanding of the anatomical basis of the test [20], and lack of reproducibility.

The combination of information about mechanism of injury, pain behavior, and location of pain with conventional radiographic signs might provide a more accurate evaluation of clinical conditions.

Supraspinatus Tendon Tests

The supraspinatus is a long, thin muscle, whose function is to elevate the humerus. It arises from the dorsal surface of the scapula, in the supraspinatus fossa, and from the fascia covering the muscle. It passes over the top of the shoulder joint and its tendon inserts on the upper aspect of the greater tuberosity. It acts as an elevator of the shoulder, as the deltoid. For this reason, it is difficult to distinguish their activities. Moreover, physical examination can be hindered by patient's pain. In this case, subacromial lidocaine injection can help distinguish patient's weakness due to this factor.

The following are some of the main tests for the supraspinatus tendon.

Empty Can Test

The *empty can test* is also known as the *supraspinatus test* or the *Jobe test* [21]. Patient's shoulder is abducted of 90° in the scapular plane and internally rotated (thumb pointing downward). The examiner pushes patient's arm downward asking the patients to resist the pressure. The test is positive when pain or weakness arises (Fig. 3.4).

Fig. 3.4 The empty can test (or Jobe test) is performed placing patient arms in 90° abduction and 30° horizontal adduction (in the plane of the scapula) with thumbs pointing downward so as to produce internal rotation of the shoulder. The examiner then pushes the patient's arms downward while asking the patient to resist the pressure. Pain or weakness is indicative of a positive test

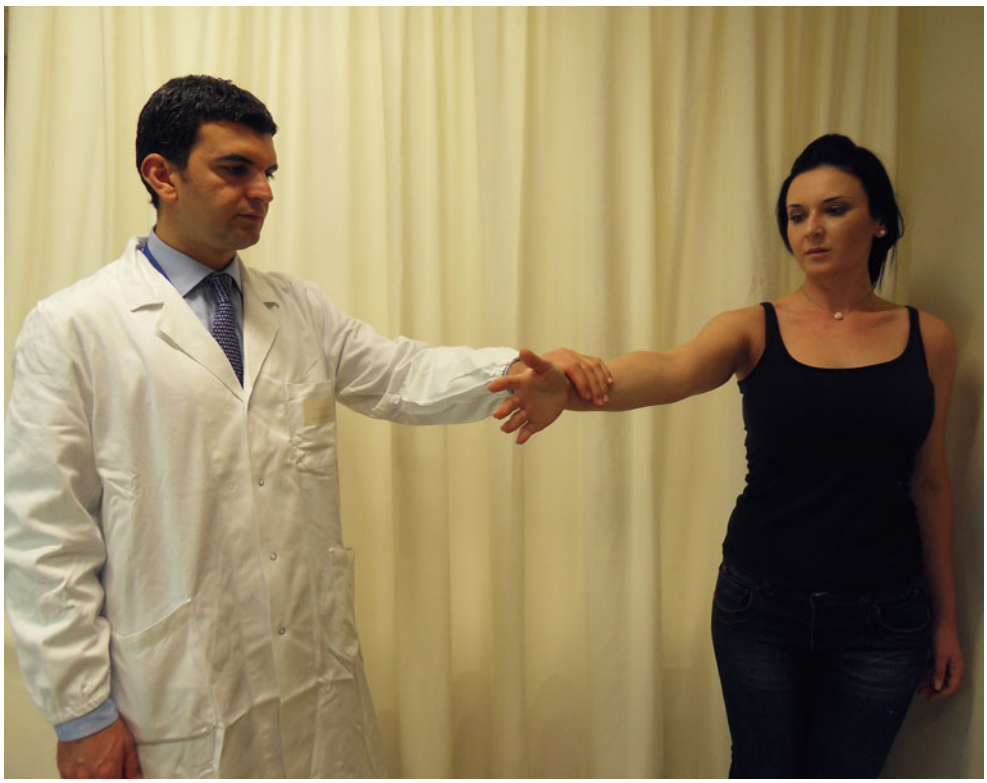


Fig. 3.5 The full can test is performed with patient's arms in 90° abduction in the scapular plane and rotated 45° externally, with the thumb pointing upwards. The sign is positive when there is pain or weakness at the downward pressure applied by the examiner

Sensitivity and specificity of the empty can test were reported to be greater than 80 % in different studies [22–30].

Full Can Test

The *full can test* is performed with patient's shoulder abducted to 90° in the scapular plane and externally rotated (thumb pointing upward). As for the empty can test, patient is asked to maintain the position while the examiner apply a downward force. The test is positive when pain or weakness arises (Fig. 3.5). Studies reported a sensitivity from 34.5 [23, 31] to 83 % [23] and a specificity from 30.8 [31] to 81 % [25].

Resisted Isometric Abduction

The resisted isometric abduction [32] is performed with patient's shoulder abducted to 90° and arm in neutral rotation. The examiner applies a downward force asking the patients to resist. Pain or weakness indicates a positive test. This test was reported among the most sensitive tests for the diagnosis of full-thickness tears [31].

Resistance Test

The *resistance test*, or the *gum-turn test* [33], is performed with the shoulder at 90° of abduction, 20–30° forward elevation and external rotation. Patient is asked to follow the path of a drawn spiral (width = 20 cm) for 20 times. If patient is not able to conclude it, because of weakness or pain, the test is positive. The resistance test was introduced and evaluated by Gumina et al. [33], who reported 55 % of sensitivity and 98 % of specificity.

Painful Arc Test

Patient is standing and is asked to abduct the arm with the shoulder externally rotated (palm facing up). If pain is experienced between 60° and 120°, the test is considered positive. Sensitivity ranges from 9.5 [34] to 97.7 % [35] and specificity from 9.9 [35] to 88.4 % [34].

Palpation of the Supraspinatus

With the elbow flexed to 90°, the shoulder is externally and internally rotated and then hyperextended. Palpating the top of the humeral head, a "sulcus" can be felt. The maneuver was first described by Codman [36]; subsequent studies reported good sensitivity and specificity (95.7 and 96.8 %, respectively) [34].

Drop-Arm Test for Supraspinatus

The *drop-arm sign* was described by Codman [37]. Patient elevates the arm fully and then slowly reverses the motion. A sudden drop or onset of pain is indicative of a positive test. Sensitivity was reported from 4.4 [38] to 73 % [39] and specificity from 77 [39] to 100 % [38].

Infraspinatus and Teres Minor Tendon Tests

The infraspinatus is a thick triangular muscle, with three pennate origins that arise from the infraspinatus fossa and insert on the greater tubercle, below the footprint of the supraspinatus.

The teres minor arises from the upper two-thirds of the dorsal surface of the lateral border of the scapula and from the septa between it and the infraspinatus. It inserts on the greater tuberosity below the insertion of the infraspinatus.

The infraspinatus and the teres minor externally rotate the shoulder. Their action is more easily differentiable from that of the deltoid that has a limited ability to externally rotate the humerus.

External Rotation Strength Test

The *external rotation strength test*, also known as *Patte test* [40], is performed with the examiner holding patient's elbow in 90° of forward elevation in the plane of the scapula. The patient is asked to externally rotate the arm against resistance.

External Rotation Lag Sign (ERLS)

The examiner externally rotates patient's arm as far as it will go passively, with the elbow flexed to 90° and the shoulder elevated to 20° in the scapular plane. Then, the examiner asks the patient to maintain the external rotation while supporting only the elbow. The test is positive when a lag or angular drop occurs. The external rotation lag sign has good specificity, from 91 [41] to 100 % [28].

Drop Sign

The *drop sign* is similar to the external rotation lag sign. They differ in the degrees of shoulder elevation. In this case the shoulder is placed at 90° of elevation in the scapular plane.

Dropping Sign

Patient's shoulder is adducted and the elbow is flexed to 90°. The examiner externally rotates patient's arm to 45° and then asks the patient to maintain the position. The test is positive when he fails to maintain external rotation and the forearm drops back to 0°. The sign was described as extremely sensible (100 %) and specific (100 %) [42].

Weakness with External Rotation

Patient's arms are alongside the body, elbows are flexed to 90° with thumbs up, and the shoulders are internally rotated to 20°. The examiner places his hands on the back of patient's hands and pushes the forearm internally (Fig. 3.6).

Subscapularis Tendon Tests

The subscapularis muscle arises from the subscapularis fossa and inserts on the lesser tuberosity of the humerus. It is an internal rotator of the shoulder, but its function is difficult to isolate with a single test because of the contribution of others muscles to this motion.

Lift-Off Test

The *lift-off test* was described in 1991 by Gerber and Krushell [43]. Patient places the hand on the back at waist level and then lifts the hand away from the body. The test is positive if he/she can perform this maneuver (Fig. 3.7). However,

Fig. 3.6 Weakness with external rotation is evaluated with the patient sitting or standing with the arms alongside the body. The elbows are flexed to 90° with the thumbs up, with the shoulders rotated internally 20°. The examiner places his hands outside those of the patient's and directs the patient to resist attempts at pushing the forearm internally

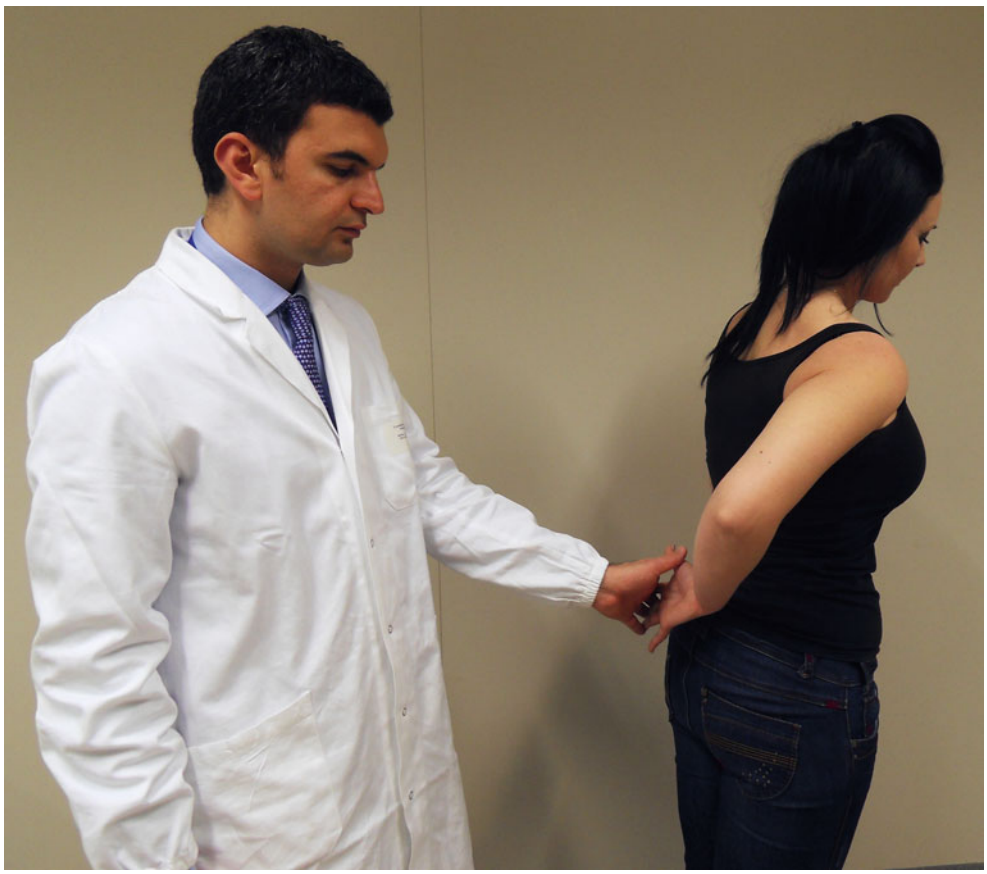


Fig. 3.7 The lift-off test is performed by placing the hand of the affected arm on the back (at the position of the midlumbar spine) and asking the patient to internally rotate the arm to lift the hand posteriorly off of the back. The test is considered positive if the patient is unable to lift the arm posteriorly off of the back or if he/she performs the lifting maneuver by extending the elbow or the shoulder

frequently patients have an internal rotation contracture that prevents from passively placing the hand behind his/her back.

The lift-off test reported a wide range of sensitivity, from 17.6 [35] to 94 [44], and good specificity, from 69 [35] to 100 [45].

Internal Rotation Lag Sign (IRLS)

The *internal rotation lag sign* is similar to the lift-off test. In this case the examiner lifts patient's hand from the back until full internal rotation. Then, the patient is asked to maintain this position while the examiner holds the elbow. The test is positive when a lag occurs.

Studies showed good sensitivity (97–100 %) and specificity (84–96 %) [28, 39, 44].

Belly Press Test

The *belly press test* was described by Gerber et al. in 1996 [46]. It requires less internal rotation than the lift-off test because the hand is not rotated behind the back. Patient's arm is at the side, the elbow is flexed to 90°, and the palm is placed on the belly. The patient should push the palm into his/her abdomen by internally rotating the shoulder. The test is positive if he/she shows weakness or needs to extend the elbow or the shoulder to exert force (Fig. 3.8).

This test seems to be a specific test for subscapularis muscle tendon tear [35, 44], even if its specificity is lower than the lift-off test.

Napoleon Test

The *Napoleon test* is a variation of the belly press test. Starting with the hand placed on the belly, patient should bring the elbow anteriorly without moving the entire shoulder girdle forward. The examiner can also test muscle strength by holding resistance against patient's elbow. The test is positive if the maneuver produces pain and/or weakness.

The sensitivity ranges from 25 to 98 %, while specificity is around 97 % [35, 44].

Bear-Hug Test

The bear-hug test was described by Barth et al. in 2006 [35]. Patient places the hand on the contralateral shoulder with the examiner holding the elbow anterior to the body. Patient should resist examiner's attempt to raise the hand. The test showed good specificity, even if lower than that of the lift-off test [35, 44].

Impingement Tests

Neer Impingement Sign and Test

The *Neer impingement sign* was first reported in 1972 and later fully described in 1983 [47]. It was originally performed with the examiner stabilizing the scapula with one

Fig. 3.8 Belly press test is performed with the arm at the side and the elbow flexed to 90°, by having the patient press the palm into his or her abdomen by internally rotating the shoulder. The test is considered positive (1) if the patient shows a weakness in comparison to the opposite shoulder or (2) if the patient pushes the hand against the abdomen by means of elbow extension or shoulder extension, indicating inability to exerting a force against the abdomen by active internal rotation produced by the subscapularis

hand and the other hand elevating patient's arm in the plane of the scapula. The maneuver was modified placing the patient supine to minimize scapular rotation. The arm is brought into full forward flexion and internal rotation to further accentuate supraspinatus impingement underneath the coracoacromial arch. Even if the sign has good sensitivity, its specificity is limited because other shoulder pathologies, especially Bankart lesions, SLAP lesions, and acromioclavicular joint arthritis, often cause pain with this maneuver [48].

The *Neer impingement test* is performed after a positive Neer sign by injecting 5 mL of 1 % lidocaine into the subacromial space. It will reduce or avoid pain when the Neer sign is repeated after few minutes.

Hawkins-Kennedy Impingement Test

The *Hawkins-Kennedy impingement test* was described in 1980 [49]. It is based on the same principle of the Neer sign [50]. In this case patient's shoulder is placed in 90° of forward flexion with the elbow bent 90°, and the examiner then forcibly internally rotates the arm. As for the Neer sign, it is sensitive but lacks specificity.

Internal Rotation Resistance Stress Test

Internal rotation resistance stress test should be performed in patients with a positive Neer impingement sign to differentiate subacromial and internal impingement [51]. Patient's arm is positioned in 90° of abduction in the coronal plane and 80° of external rotation. Then, isometric external and internal rotations are tested. Weakness in resisted external rotation indicates subacromial impingement, while weakness in resisted internal rotation indicates internal impingement.

Biceps Tendon Tests

There is a significant crossover between rotator cuff tears and pathology of the long head of the biceps. An accurate history and physical examination are fundamental, especially in the case of biceps tendinitis and instability. Several tests have been described for isolated lesions of the biceps tendon. However, little literature is available on sensitivity and specificity of these tests.

Yergason's Test

Patient's arm is place at side, with the elbow flexed to 90° and hand in pronation. It is asked to supinate the hand against resistance (Fig. 3.9). The test is indicative of biceps tendinopathy if pain rises anteriorly along the bicipital groove or in the anterior shoulder. As the shoulder is not moving, the test allows a more isolated examination of the biceps. In patients with biceps tendinopathy, it was found an incidence of 50 % positivity with this sign [52].

Speed's Test

The Speed's test was first described in 1966 [53]. It is performed with patient's arm placed in 60–90° of forward flexion, the elbow extended, and the hand in full supination. It is asked to resist a downward force at the wrist. The test is positive if it induces pain at the anterior shoulder or in the bicipital groove. Its specificity and sensitivity were reported to be 14 and 90 %, respectively [54].

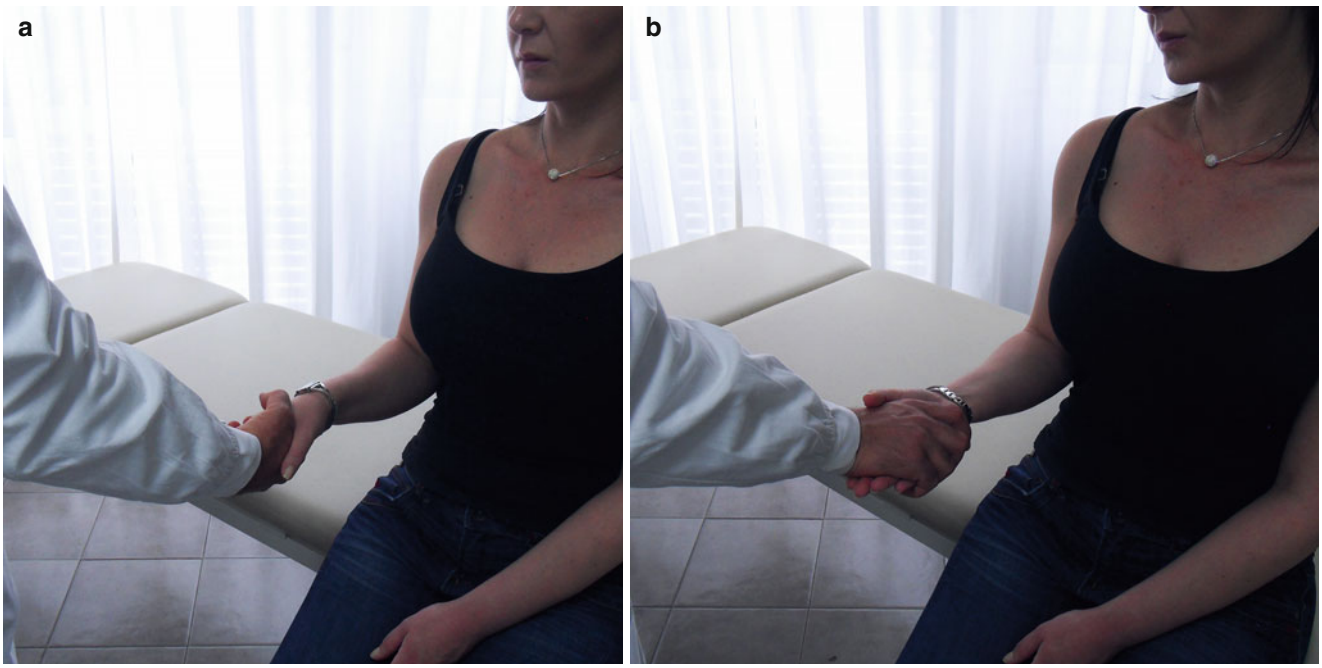


Fig. 3.9 The Yergason's test. Starting from full pronation (**a**), the patient is asked to supinate the hand against resistance (**b**)

DeAnquin's Test

The examiner rotates patient's arm while having his or her finger in the most tender spot in the bicipital groove. The test is indicative of biceps tendinitis if pain rises when tendon glides beneath the finger.

Biceps Instability Test

The *biceps instability test* was described by Abbott and Saunders to identify a complete or incomplete dislocation of the tendon [55]. Patient's shoulder, fully abducted, is slowly brought from complete external rotation to internal rotation. If the biceps tendon subluxates or dislocates from the groove, a painful click can be palpated or audible.

Lippmann's Test

The examiner displaces the tendon from one side to the other, with the elbow flexed. It is necessary to pay attention to roll the subluxed tendon and not the deltoid muscle [56].

Ludington's Test

The *Ludington's test* is performed with patient's hands on top of the head, with palms down and fingers interlocked. Biceps contraction can produce pain in the bicipital groove in the case of tendinitis, or subluxation can sometimes be palpated in the groove.

Superior Labrum Tests

O'Brien Test

The *O'Brien test*, or *active compression test*, was described in 1998 to differentiate acromioclavicular joint pathology and superior labral pathology. It is performed with the shoulder forward flexed to 90° and adducted 15° toward the midline. The examiner produces a downward force with the arm internally rotated (thumb pointing to the floor) and then with the arm in full supination and external rotation (Fig. 3.10). The test is indicative of superior labral pathology when anterior shoulder pain increases with the arm internally rotated and decreases when the arm is externally rotated. In their original study, O'Brien et al. reported sensitivity of 100 % and specificity of 99 % for this test [57].

SLAPprehension Test

The *SLAPprehension test* is a modification of the O'Brien test. In this case the maneuver is performed with patient's shoulder adducted to 45°.

Anterior Slide Test

The *anterior slide test* was introduced in 1995 by Kibler et al. [58]. It is performed with patient's arms akimbo. The examiner places one hand over the top of the acromion and the other hand on patient's elbow where an anterosuperior directed force is exerted. The test is positive if pain or a click is felt over the anterior shoulder. Kibler et al. reported higher specificity (91 %) than sensibility (71 %) for this test [58].

Fig. 3.10 The O'Brien test (active compression test)

Crank Test

The *crank test* is performed with the patient either supine or sitting, the arm axially loaded in 160° of flexion and then internally and externally rotated in an attempt to catch labrum tears between the two joint surfaces. The test is positive if it produces pain, catching, or a click. Sensitivity and specificity have been reported to be 91 and 93 %, respectively [59].

Pain Provocation Test

The *pain provocation test* is performed with the arm abducted to 90° and externally rotated. Patient is asked to place the hand in full supination and then in full pronation (Fig. 3.11). The test is positive if pain increases while the hand is in pronation. The sensitivity of the test is 100 % and its specificity is 90 % [60].

Biceps Load Test I

The *biceps load test I* was first described in 1999 for patients with history of recurrent anterior instability [61]. It is performed in the position of apprehension (90° of abduction and

Fig. 3.11 The pain provocation test. With the arm in 90° of abduction and full external rotation, the hand is placed in full supination (**a**) and then full pronation (**b**). Increased pain in full pronation suggests a SLAP lesion

Fig. 3.11 (continued)

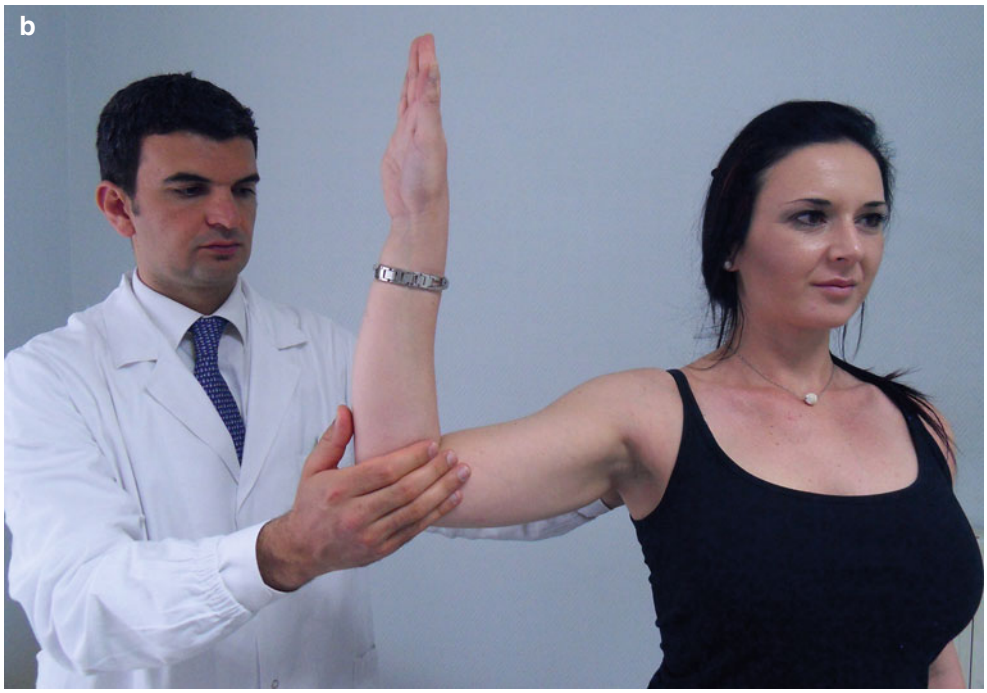
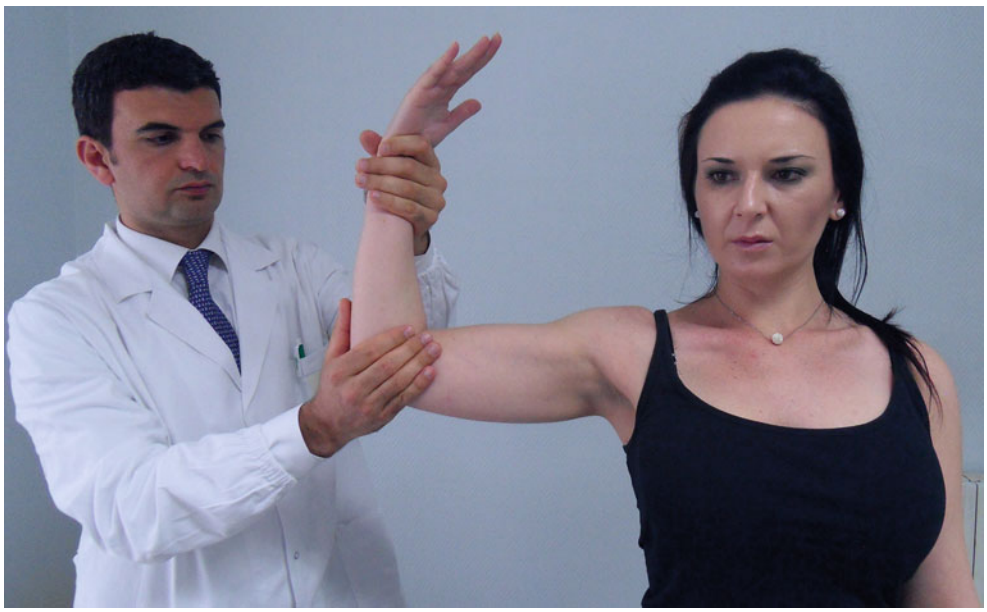


Fig. 3.12 The biceps load test I



full external rotation). The examiner holds patient's arm at that position and exerts a resistance against elbow flexion (Fig. 3.12). The test is suggestive of SLAP lesion if biceps contraction does not change patient's pain and apprehension. It has a sensibility of 91 % and a specificity of 97 % [61].

Biceps Load Test II

The *biceps load test II* was described in 2001 for patients without history of anterior instability. It is performed with patient supine, arm elevated to 120°, in full external rotation, elbow flexed to 90°, and forearm supinated. The patient is asked to flex the elbow against resistance. If the maneuver increases pain, the test is positive. The sensibility has been reported to be 90 % and the specificity 97 % [62].

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Imaging of the Shoulder

4

Nicola Magarelli, Chiara Carducci, and Claudia Dell'Atti

Introduction

Radiological examination of the shoulder currently employs different imaging techniques that are used separately or more often in combination, depending on the specific clinical issues. The task of imaging is not only to detect the presence of pathology but also to direct the surgeon towards the most suitable treatment (conservative, arthroscopic, or open surgery). The need to choose between different imaging modalities is in part due to the anatomical complexity of the shoulder and the adjacent soft tissues. The contribution of each method does not always answer the specific clinical question posed by the specialist. Conventional radiographic examination (CR) performed with standard and complementary views is often deemed crucial for the biplanar evaluation of bone structures of the glenohumeral and acromioclavicular joint, as well as to exclude calcifications or bone lesions; however, when no explanation is identified for the "pain" in the shoulder, given the absence of lesions detected by radiography, it becomes necessary to integrate second-level exams.

Ultrasound (US) examination of the shoulder is a technique used to study myotendinous components and other periarticular soft tissues with the aim of assessing the tendinous components at their tuberosity insertions, detecting the presence of articular and periarticular fluid (bursae and synovial recesses), and excluding traumatic and nontraumatic lesions of the rotator cuff. US examination has proved a reliable method to document the presence of calcifications that are undetectable on standard radiographical exams, as well as ultrastructural alterations of the tendon and the fibrochondral junction of the entheses. Moreover, in experienced hands, it can document any compression of the suprascapular nerve in the homonymous notch or in the spinoglenoid groove. US examination plays an important role as a guide in minimally invasive surgery, such as the drainage of superficial fluid collections, calcific tendonitis, space-occupying lesions, and in the symptomatic treatment of a painful shoulder (to guide the injection of hyaluronic acid and steroidal and nonsteroidal anti-inflammatory drugs).

Computed tomography (CT) is a radiological technique that employs ionizing radiation. It applies different reconstruction algorithms and the possibility to perform, by means of the latest technological devices (volumetric CTs), 2D multiplanar and 3D volumetric reconstructions in multiple planes. This exam is especially indicated in traumatic pathology. It is not normally used in cases of clinical suspicion of rotator cuff tears, given its low contrast resolution compared to MRI examination. CT finds another important application in the study of the glenoid bone in shoulder instability to exclude avulsion lesions of the glenoid bone itself (quantification of the glenoid bone defect).

Magnetic resonance imaging (MRI), a second-level methodology, is now an indispensable technique for the diagnosis of disorders of the shoulder. Since its clinical introduction, it has played an important role in neuro- and musculoskeletal radiology. Its constantly increasing use is currently justified in more complex diseases of the shoulder. MRI is considered a preliminary examination to the surgical/arthroscopical approach, since it allows traumatic and atraumatic myotendinous, capsuloligamentous, and fibrocartilaginous lesions, as well as alterations in the other peri-skeletal soft tissues to be documented.

Contrast medium imaging techniques (CT arthrography and MR arthrography) deserve a separate discussion; their questions are aimed at studying capsuloligamentous and fibrocartilaginous structures. Particularly, they are suitable for analysis of articular cartilage and anatomical structures that may be injured in cases of subluxation or dislocation. Such minimally invasive techniques are also employed to confirm the presence of subtle rotator cuff tears or in patients who have undergone rotator cuff repair, for whom MR-based examinations are difficult to interpret due to the presence of

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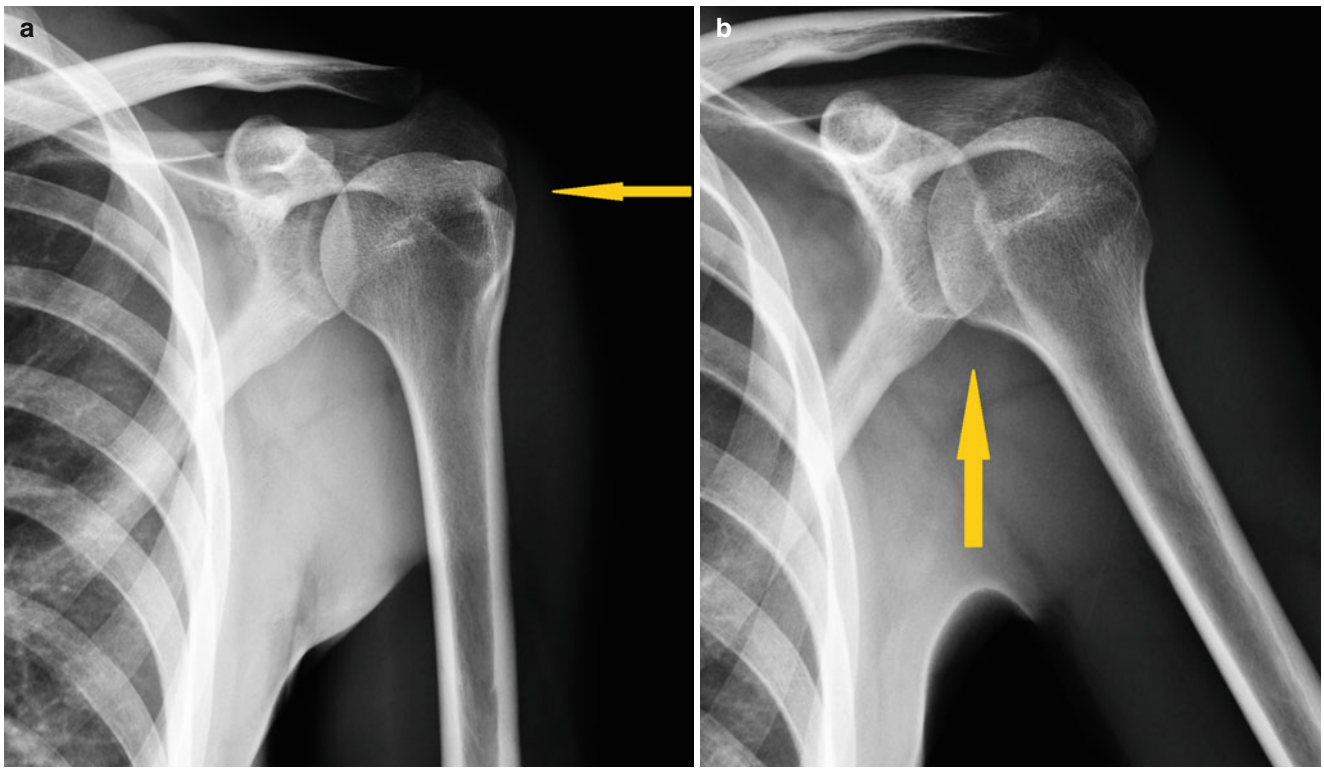

Fig. 4.1 (**a**) Radiographic anteroposterior (AP) view of the left shoulder performed with external rotation of the arm. The greater tuberosity of the humerus can be visualized (*arrow*). (**b**) Radiographic AP view of the same shoulder performed with internal rotation of the arm. On this view, the lesser tuberosity of the humerus can be visualized (*arrow*)

local ferromagnetic artifacts (metal anchors) or reactive subacromial-deltoid (SAD) bursitis. Moreover, clinical diagnosis of a frozen shoulder can be confirmed by MR arthrography, which assesses joint "capacity." Sometimes the individual methods are applied in combination when the clinical question or the result of a single method has identified benign or malignant space-occupying lesions. Only the combination of these methods allows a better characterization of the lesions and their relationships with surrounding neurovascular structures.

Conventional Radiology

Standard anterior-posterior (AP) views of the shoulder are obtained with internal and external rotation of the arm (Fig. 4.1). In the first case, the purpose is to evaluate the cortical bone of the greater tuberosity, while the second makes the lesser tuberosity visible. Moreover, standard AP views can be obtained by tilting the X-ray tube in a craniocaudal direction by 20° in order to better estimate the subacromial space. The external-rotation AP view identifies the presence of calcifications at the supraspinatus insertion, while internal-rotation AP view shows calcifications in the infraspinatus, teres minor, and subscapularis. As a normal finding, the external-rotation AP view can document the presence of an area poor in trabeculae at the level of the greater tuberosity called "pseudocyst of the humerus" [1]. Another normal finding is the identification in the external-rotation AP view of a hyper-diaphanous lamellar ridge projecting in correspondence with the joint cavity [2]. In addition to standard exams, complementary views can be performed according to the clinical question, each one with different purposes: (1) true AP view: this view is obtained by inclining the patient by approximately 40° in order to have the shoulder blade parallel to the sensitive plane and the beam angled medial to lateral by 45°; the purpose of this projection is to scan the joint line avoiding overlaps; (2) "outlet" view: a posterior-anterior view with the patient in the upright or supine position with the side under scrutiny on the sensitive surface inclined by about 60° without or with craniocaudal inclination of the X-ray tube at 20°; this view shows, inscribed in the glenoid, the humeral head which must project between the coracoid process anteriorly and the acromion posteriorly; (3) axillary views, including the West Point view: this exam is performed with the patient prone on the X-ray table with the limb to be examined raised by 8 cm, the head and neck are rotated in the opposite side, the radiographic cassette is maintained on the top portion of the shoulder, and the X-ray tube centered on the axilla with a 25° medial inclination; the

resulting image is a tangential view of the anteroinferior glenoid; (4) Stryker notch view: this is performed with the patient supine on the table with the radiographic cassette positioned under the shoulder, the palm of the hand of the side to be examined is placed on the head, the X-ray tube is tilted by 10° cranially and centered on the coracoid; (5) double oblique or apical oblique view, in which the patient is sitting so as to make the scapular blade parallel to the sensible plane with the X-ray tube tilted mediolaterally by 45° and craniocaudally by 45° in order to project the anteroinferior surface of the glenoid [3]. Complementary views are usually performed when the clinical question concerns the instability of the shoulder, as they identify lesions in the posterior cortex of the humeral head (Hill-Sachs fracture) (Stryker notch view), fragmented bony avulsions of the glenoid (bony Bankart lesion) (West Point and double oblique views), or humeral dislocations (outlet view). The outlet view is also used to provide further images of the functional acromioclavicular arch without bone overlaps. In this way, the presence of calcifications in the cuff structures, and in particular of the supraspinatus and infraspinatus tendons, is identified along with the calcifications in the SAD bursa. Furthermore, this view allows examination of possible osteophytes of the undersurface of the anterior part of the acromion and acromioclavicular joint responsible for subacromial impingement. Radiographic studies of the shoulder are used as preliminary step of minimally invasive examinations such as CT arthrography (CTA) and MR arthrography (MRA). Radiography of the shoulder is performed only in the AP view to evaluate the proper position of the needle into the joint cavity and the initial filling of the same.

Echotomography

Over the years, US examination of the shoulder has gained importance because of its ease of execution and because, according to some clinical indications, it could be the ultimate exam. This method has many advantages, including the fact that it does not use ionizing radiation, making it harmless and repeatable at any time.

The patient is seated in front of the operator and is informed of the different arm positions required during the examination of each tendon. The supraspinatus tendon is investigated by bringing the patient's arm behind her/his back, in order to prevent the shadow of the acromion from covering the greater tuberosity of the humerus (Fig. 4.2). The ultrasound probe explores the tendon in an oblique coronal plane. Then, the probe is oriented in a plane orthogonal to the previous one and shows, from front to back, the supraspinatus and the posterior rotator cuff (infraspinatus and teres minor). At this point, the patient is asked to bring her/his arm to the front, with the forearm at a 90° angle

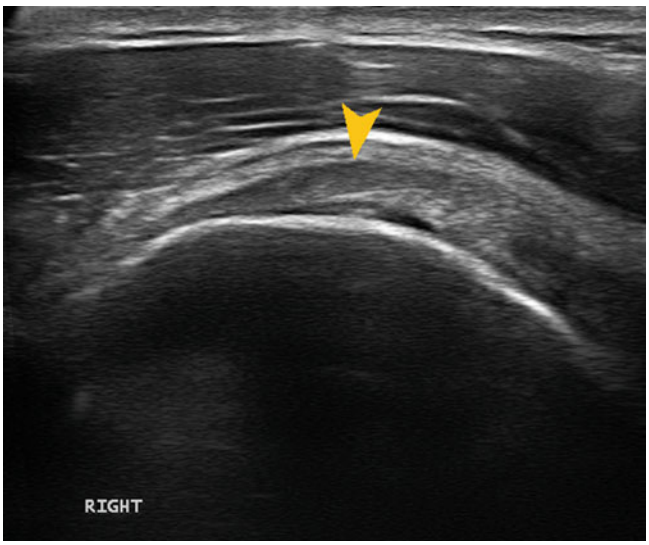

Fig. 4.2 Echotomographical oblique coronal scan for the evaluation of the tendon of the supraspinatus tendon (*arrowhead*)

with the upper arm and with the palm of the hand upwards. The front and upper surface of the humerus is imaged in axial planes, in order to show the subscapularis tendon, which is composed of approximately 4–5 tendon units and, more laterally, the intertuberosity groove of the humerus where the long head of the bicep (BLH) is visible. During this scan, it is possible to study the tip of the coracoid process, from which the coracoacromial originates, as well as the coracohumeral and coracoclavicular ligaments, the short head of the biceps brachii and, more medially, the coracobrachial muscle. Axial scans can document effusions in the subcoracoid bursa, in the synovial sheath of the LHB, and in the subscapularis recess. More caudal axial scans allow visualization of the pectoralis major tendon. Bringing the probe to longitudinal planes allows the examination of the tendon units of the subscapularis and LHB along their major axis.

Another important advantage of US is the ability to perform dynamic scans that are useful for assessing tendon's movement and possible bone or calcified formations involving tendons that limit or prevent their regular movement. On the axial plane, it is possible to assess the depth of the bicipital groove and the position of the LHB in the groove itself. The transverse ligament is identified, which closes the intertubercular sulcus anteriorly, thus preventing dislocation of the LHB. By instructing the patient to place her/his hand onto the contralateral shoulder and running a posterior oblique coronal plane scan, it is possible to evaluate the teres minor and infraspinatus tendons at the same time, which are frequent sites of calcification. On the contrary, a posterior medial vertical plane serves to distinguish the individual muscle bellies. Another crucial plane is the posterior axial one, which documents glenoid fibrocartilage and the

scapular notches where the neurovascular bundle can be identified (nerve, artery, and suprascapular veins). US examination is concluded with the evaluation of the acromioclavicular joint (ACJ). This joint is studied by placing the transducer on its upper surface and identifying the two bone heads separated by a fibrocartilaginous disc. In pathological conditions, this space can contain fluid expanding the capsule; ACJ may also appear swollen because of marginal osteophytes which alter bone profiles, narrow the joint, and compress the interposed articular disc (degenerative changes). Under physiological conditions, the subacromial bursa appears as a hyperechogenic structure between the deltoid muscle and the supraspinatus tendon (oblique coronal scan); this aspect depends on the attachment of the bursal wall: in the presence of fluid, the attached walls separate showing an anechogenic layer between the deltoid and the supraspinatus. US study of the rotator interval deserves a special mention. Located between the tendon of the supraspinatus and the most cranial fibers of the subscapularis, it contains the superior glenohumeral ligament (SGHL), the LHB, and the coracohumeral ligament (CHL). The SGHL is difficult to explore with an ultrasound probe, while the LHB and the CHL can be scanned by ultrasound probe without interpositions. This ligament is an important stabilizer of the LHB, which has a conical shape with its apex at the coracoid. Its base, consisting of a medial and a lateral heads, encloses the LHB together with the transverse ligament of the subscapularis, preventing its dislocation.

The presence of clinically evident swelling localized in the subcutaneous adipose tissue in a muscle, adjacent to a joint cavity, or in continuity with it, gives further indication for the execution of US examination. In these cases, thanks to the structural study of the lesion, it is possible to take into account space-occupying solid benign diseases, such as lipomas and fibro-lipomas, primary or secondary malignant lesions, or fluid-containing lesions, such as arthrogenous ganglia. Finally, US examination can document an alteration in the size and structure of muscular masses surrounding the shoulder girdle, such as neuromuscular disease, in which the muscle belly appears hyperechogenic due to fatty infiltration. In these cases, the examination can be useful for muscle biopsy.

In normal conditions, the anterior glenoid labrum cannot be explored due to the lack of an adequate acoustic window; therefore, US is not indicated in shoulder dislocation. However, an indirect sign of the occurred dislocation is the depression found on the posterolateral surface of the humeral head (Hill-Sachs lesion).

Computed Tomography

Computed tomography (CT) of the shoulder is performed in the axial planes with multi-detector devices. The choice between prone or supine position depends on the patient's comfort and on the clinician's specific indication: standard examination of the glenohumeral joint; CTA approach (in which the distribution of air or radiopaque contrast is affected by the patient's position); examination of multiple-trauma patients (where the position is mandatory); or CT-guided biopsy. In most CT examinations, the patient is positioned at the center of the gantry, but there are skeletal segments for which the use of small fields of view is required and the patient has to be positioned obliquely to avoid the acquisition of unnecessary volumes [4].

During the examination of the shoulder, the patient is positioned at the center of the gantry so that the two shoulders are scanned simultaneously, unless there are different indications. A volumetric acquisition is performed in order to limit motion artifacts and to ensure a better 2D and 3D reconstruction in multiple planes. In addition, two different reconstruction algorithms are chosen in order to highlight the bone structures (high resolution) or soft tissues (standard or soft algorithm). The technical parameters are summarized in the Table 4.1.

The images thus obtained are visualized in the bone and soft tissue window. Several studies have been published on the use of CT in patients with shoulder dislocation, and new methods for the evaluation of bone defects have been proposed [5–7]. In this case, volumetric acquisition is required in the axial plane, as well as subsequent 2D MPR and 3D reconstructions in oblique sagittal planes to obtain "en face" views of the glenoid. A comparison between 2D and 3D acquisitions in patients with shoulder instability was carried out to assess whether the 2D could replace the 3D technique, which is considered the reference method and, furthermore, to assess whether it would be sufficient to scan only the injured shoulder to avoid any unnecessary radiation exposure [8] (Fig. 4.3). The actual role of CT in the study of the shoulder has been recently reconsidered, since MRI replaced CT in many fields; a case in point is the study of rotator cuff, in which only CTA still has diagnostic value in those patients who cannot undergo MRI. The choice between MRI and CTA of the shoulder is controversial. They have the same sensitivity for the diagnosis of full-thickness rotator cuff tears, but MRI appears to have greater sensitivity than CTA in the diagnosis of partial-thickness cuff tears [9]. CT is often used in neuromuscular diseases, because it allows the

Table 4.1 CT technical parameters of the shoulder

Speed (slice-ms)	Interval (mm)	FOV	Acquisition technique	kV	mA	Acquisition mode	Reconstruction (mm)
1.25	0.6	Large	Elicoidal	140	400–600	Standard	0.6–0.6 bone

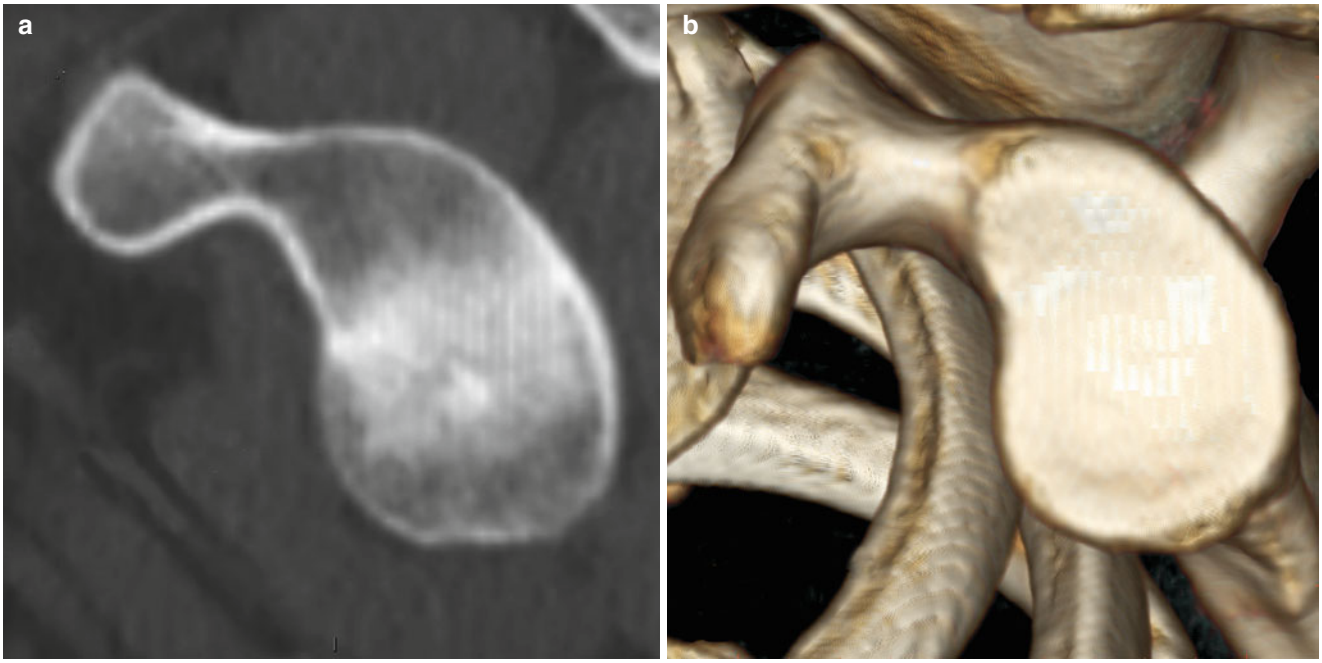

Fig. 4.3 (**a**) MPR 2D reconstruction in oblique sagittal planes for the evaluation of the articular surface of the left shoulder glenoid ("en face" view). (**b**) Volume rendering reconstruction in oblique sagittal planes for the evaluation of the articular/joint surface of the left shoulder glenoid ("en face" view)

densitometric analysis of shoulder girdle muscles; in fact, a reduction of muscle tissue density and a gradual replacement with adipose tissue can occur.

The choice of a specific contrast medium depends on the clinical indication; in the different scenarios of shoulder disorders, the radiologist can perform CTA using a single dose of iodinated water-soluble contrast or a mixture of iodine contrast and air in order to obtain the double contrast. If the purpose is to adequately "contrast" the glenoid rim or the articular surface of the cuff tendons, or when the clinical suspicion is an inflammatory, degenerative, or pseudotumoral synovial disease, such as rheumatoid arthritis (RA), pigmented villonodular synovitis, and synovial chondromatosis, the single-contrast technique may be indicated [10]. The pathology of the ACJ also takes advantage of CT study, as it identifies articular surfaces and periarticular soft tissues. This joint is often affected by degenerative changes with the presence of subchondral cysts and osteophytes which alter the profile of the undersurface of the joint and imping against the rotator cuff tendons during arm elevation; in addition, subacromial space can be reduced by joint effusion and swelling.

In case of chronic inflammatory diseases, CT can document periarticular osteopenia up to the erosion phenomena typical of the most aggressive forms or erosion of the distal end of the clavicle as in chronic renal failure (secondary hyperparathyroidism). Moreover, joint pain exacerbated by pressure on the distal end of the clavicle can be associated with a fuzzy osteopenic area of the clavicle that can be related to reflex sympathetic dystrophy syndrome (Fig. 4.4). The window of visualization of bone tissue can show calcification of the fibrocartilaginous disc of the ACJ, which is typically observed in metabolic diseases such as hydroxyapatite crystal deposition disease [11].

CT is also useful in the evaluation of coracoid impingement. Distance between the apex of the coracoid process and the lesser tuberosity of the humerus can be measured in the axial planes. This distance changes depending on the rotation of the arm (internal or external rotation). The coracohumeral distance can also be subjected to change resulting from fractures of the coracoid process and lesser tuberosity, calcification of the subscapularis tendon, or surgical procedures (transposition of the coracoid process).

A CT scan is performed when bone lesions undetected at standard examinations lead to disabilities. These include acute fractures of the humeral tuberosities; osteochondritis dissecans of the glenoid or humeral head; stress fractures of the clavicle, scapula, and ribs; and post-traumatic osteolysis of the distal end of the clavicle [12]. Finally, CT also plays an important role in benign or malignant tumors affecting the scapular-humeral girdle. An example of this is differential diagnosis between osteochondroma and peripheral chondrosarcoma; in fact, CT can accurately assess the nature of the lesion by detecting the mineralization pattern and thickness of the cartilaginous cap, even though MRI has a more significant role in the last case due to its inherently higher contrast resolution.

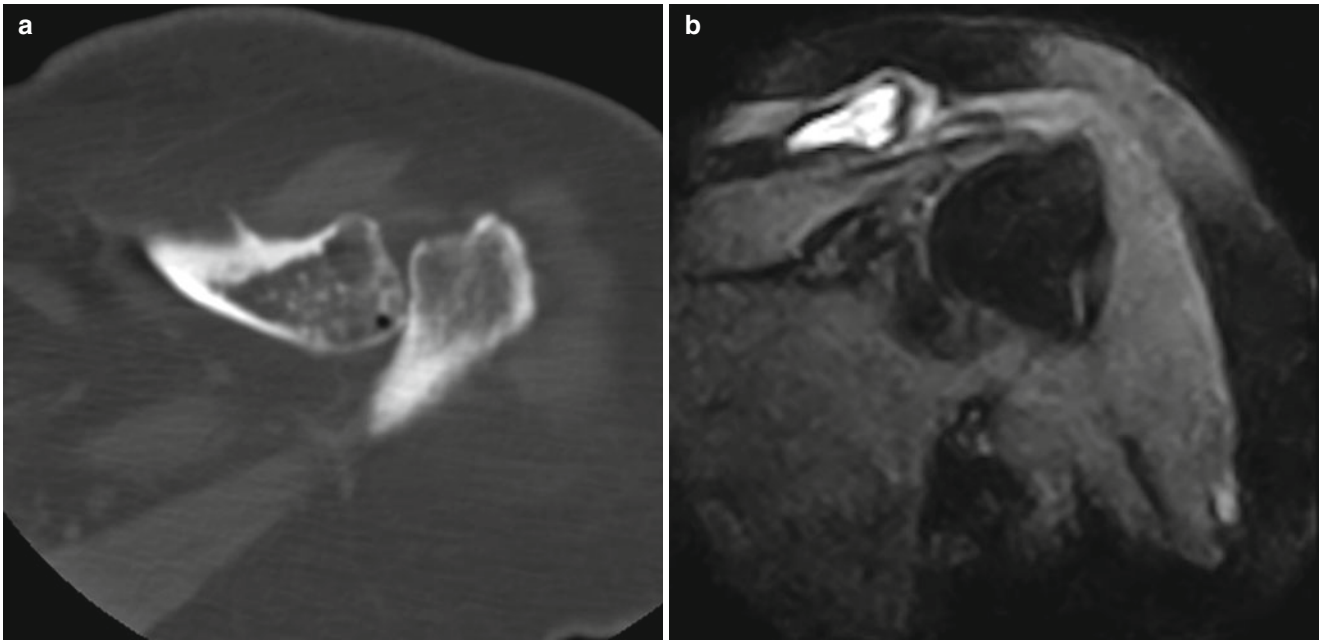

Fig. 4.4 (**a**) Algodystrophic focus in a 63-year-old woman with pain at finger pressure of the distal end of the left clavicle. Axial CT scan of the acromioclavicular joint. (**b**) Same patient: MR image performed with suppressed signal from adipose tissue in a coronal oblique plane. It documents widespread edema of the distal end of the clavicle

Magnetic Resonance Imaging

The ability of MRI to obtain multiplanar acquisitions and soft tissue contrast with multiple parameters allows a detailed study of the shoulder and helps to identify the complex anatomical structures and variants as well as pathologies of these structures. Among the characteristics that make MRI an important diagnostic method for musculoskeletal system are the high sensitivity to physical differences between tissues and fluids, the ability to show these differences as tissue contrast, the ability to highlight these differences working with specific parameters, and the ability to highlight vascular and neural structures without the administration of a contrast medium (CM) [13]. Since MRI does not use ionizing radiation, it has an important value in the study of pediatric patients.

Since its introduction, industries improved the performance of MRI devices and dedicated coils over years. Updated software for fast sequences have been recently introduced, which allow the evaluation of the shoulder in about 30 min, thereby reducing motion artifacts due to patient's fatigue from maintaining the same position for a long time. Dedicated phased array coils with high spatial resolution are now used which allow optimal visualization of the different structures to be evaluated: tendons, muscles, capsuloligamentous and bone structures, and neurovascular bundles.

Injures affecting the shoulder can be traumatic and non-traumatic. Indications for MRI include tendon (rotator cuff/LHB), bone (tumors, systemic diseases), capsuloligamentous, and articular cartilage pathologies. Thanks to the signal intensity and operator's choice of the sequences to be used, the radiologist is directed towards any pathology. The sequences used in a standard MRI exam of the shoulder are shown in Tables 4.2 and 4.3.

The images are acquired on three anatomical planes: axial, oblique coronal, and oblique sagittal, with 3–4 mm sections for 2D acquisitions and 0.6–1 mm sections for 3D acquisitions. The study on these three planes allows to evaluate the different structures of the shoulder (Table 4.4).

Technical parameters are variable and are chosen by the operator depending on his skills and experience; however, acquisition planes are dictated by the different anatomical structures to be evaluated. For the study of the rotator cuff, all three acquisition planes are necessary so that it is possible to visualize not only the tendons at their bony insertions but also the myotendinous junction and the capsuloligamentous and bony cartilaginous structures.

In high-field closed systems, the patient lies supine on the table with the limb under examination in a neutral position along the body; the external rotation of the arm would impinge the visualization of the LHB, while internal rotation would result in capsular redundancy and difficult discernment between supraspinatus and infraspinatus tendons in the axial plane. Another position which is extremely useful to complement the standard positions, especially in MRA, is the abduction external rotation (ABER) position, since it assesses the inferior capsule and centering of the humeral head in the glenoid cavity [14].

Table 4.2 Sequences for standard MRI of the shoulder (GE)

MRI sequences (1.5 T GE)	TR/TE (ms)	Thickness (mm)	Matrix	ETL	NEX	FOV (cm)
Axial PD	2,000/26	3	224×256	4	3	12–14
Axial T2 FS	3,000/50	3	224×256	8	3	12–14
Coronal T1	600/20	4	224×256	4	3	12–14
Coronal T2 FS	3,000/50	4	224×256	8	3	12–14
Sagittal T2 FS	3,000/50	4	224×256	8	3	12–14
Sagittal PD FS	2,000/26	4	224×256	4	3	12–14

Table 4.3 Sequences for standard MRI of the shoulder (Philips)

MRI sequences (1.5 T PHILIPS)	TR/TE (ms)	Thickness (mm)	Matrix	TEF	NEX	FOV (cm)
Axial T2 FFE	463/14	3	292×164	–	2	16
Sagittal TSE T1w	1,205/20	3	256×160	5	2	14
Coronal DPw TSE	1,207/30	3	256×140	5	3	14
Coronal PDw SPAIR	3,000/30	3.5	352×224	5	2	14.3
Axial 3D WATS C3	20/8.1	0.6	160×162	–	2	9.7

Table 4.4 Scan planes and anatomical structures detectable

Coronal oblique plane	Sagittal oblique plane	Axial plane
Longitudinal	Transverse	Long head biceps tendon (bicipital groove)
Infraspinatus muscle and tendon	Supraspinatus muscle and tendon	
Supraspinatus muscle and tendon	Teres minor muscle and tendon	
	Long head biceps tendon (proximal portion)	
Acromioclavicular joint	Subscapularis tendon and muscle	Longitudinal
		Subscapularis tendon and muscle
Acromion	Rotator interval	Glenoid labrum (anterior and posterior portion)
Glenohumeral joint	Acromion	Joint
Subacromial-subdeltoid bursa	Coracoacromial ligament	Glenohumeral joint
Glenoid labrum (superior and inferior portion)	Coracoacromial arch	Glenohumeral ligaments
	Glenohumeral ligaments	

MRI is very accurate in the study of full-thickness rotator cuff tears, which are associated with indirect signs, such as the elastic retraction of the tendinous stump and the presence of fluid which extends in the subacromial-deltoid bursa. Partial-thickness rotator cuff tears are slightly more difficult to visualize because they can affect the articular, bursal, or interstitial aspect of the tendons without tendon retraction. In addition, the "false" hyperintensity signal at the distal third of the supraspinatus tendon due to the "magic angle" artifact is responsible for a linear signal alteration involving the structures located at 55° from the static magnetic field (B0). A radiologist skilled in musculoskeletal MRI will of course recognize the artifact and avoid it using sequences that are not subjected to it (sequences with TE >30 ms). On the axial plane it is possible to follow the rotator cuff tendons inserting onto the greater tuberosity posteriorly to the supraspinatus, the infraspinatus, and the teres minor. On the axial plane, it is also possible to identify complete or partial lesions of the subscapularis tendon and its relationship with the tip of the coracoid process.

The sagittal plane allows to evaluate the supraspinatus and infraspinatus fossae occupied by the supraspinatus, infraspinatus, and teres minor muscles, respectively; the morphology of the acromion is also assessed, which can contribute to rotator cuff tear by subacromial impingement. Furthermore, in the sagittal plane it is possible to identify anteriorly the rotator interval (RI) (the space between the supraspinatus and subscapularis tendon), occupied by the superior glenohumeral ligament, the LHB, and the coracohumeral ligament.

MRI has an undisputed role in the study of diseases involving the glenoid labrum and capsuloligamentous structures. In this way, when the clinical suspicion of shoulder instability is supported by capsulo-labral-ligamentous or bone injures, MRI becomes necessary as its high contrast resolution allows complete assessment of the individual stabilizing structures. However, some structures, such as the superior glenohumeral ligament – whose role in the stabilization of the shoulder is not very clear – and some anatomical variations cannot be seen at basic examination; therefore, one must refer to the MRI examination after articular injection of CM (MRA).

Intrinsic power of contrast resolution of MRI, higher than in any other radiological techniques, makes it essential in the study of tumors and cysts involving the shoulder girdle (Figs. 4.5 and 4.6). Determination of the extension of the tumor enables the surgeon to choose the most adequate

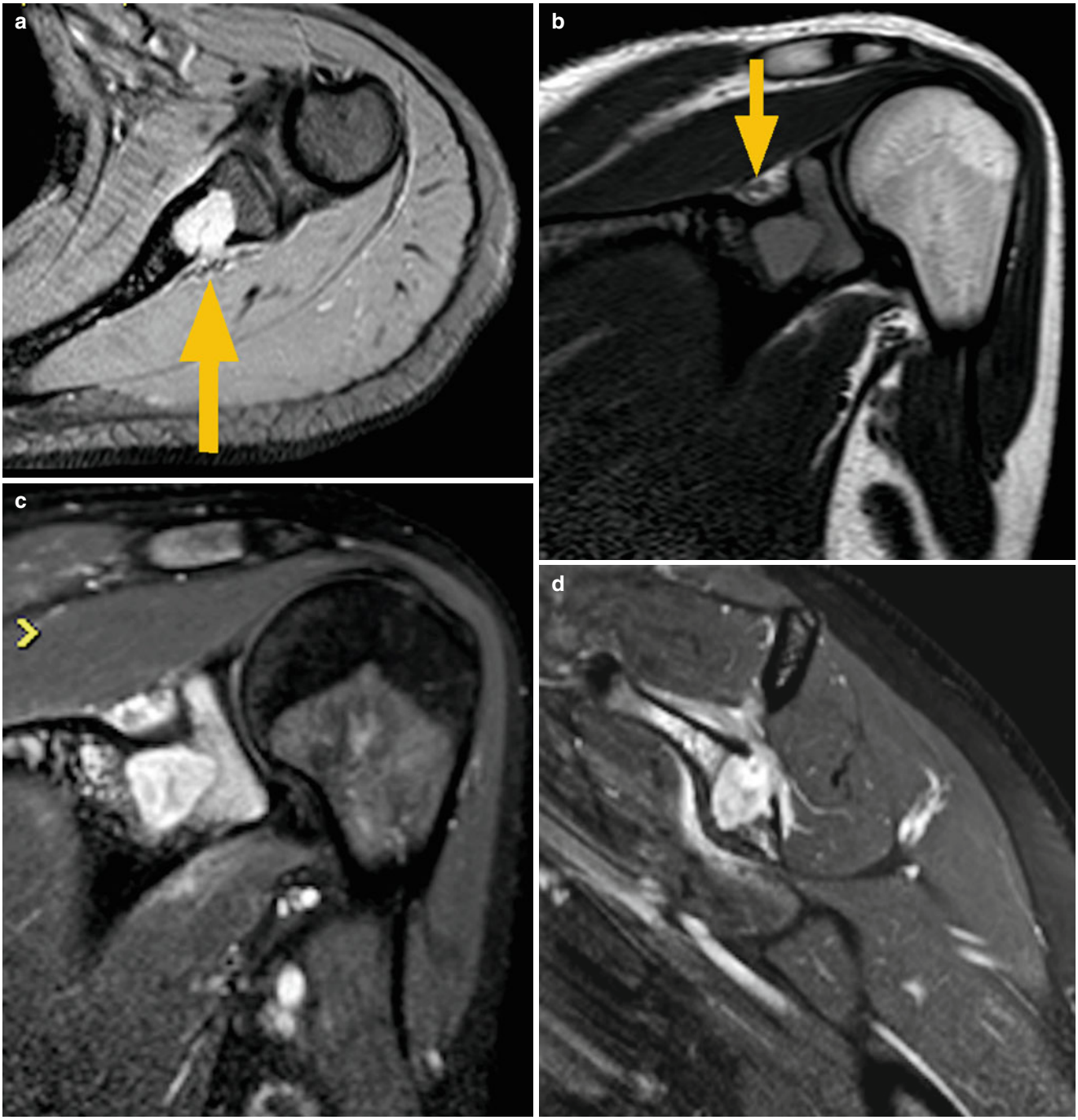

Fig. 4.5 18-year-old patient with painful symptomatology in the left shoulder. (**a**) Axial T2* GRE. Lytic lesion (*arrow*) at the body of the scapula with interruption of the posterior cortex. (**b**) T1w coronal scan, documenting lytic lesion with sclerotic rim and peripheral edema. Immediately above the lesion, the suprascapular notch and nerve are shown (*arrow*). (**c**) Coronal oblique FS T2 scan. Lytic lesion with hyperintense contents. (**d**) Oblique sagittal T2 FS scan. The scan confirms pathological interruption of the posterior cortex of the glenoid neck and edema of the surrounding soft tissue

surgical procedure, such as limb preservation surgery [13]. With regard to systemic diseases, MRI allows the evaluation of the replacement of the normal signal intensity of the bone marrow (medullary shift) by metabolically active tissue; in fact the signal is nonspecific as it could be related to a reactivation (e.g., physiological conditions of hypoxemia) or changes in the hematopoietic system (anemia, leukemia, multiple myeloma, lymphomas, etc.).

In the shoulder, as in other joints, sequences with fat tissue saturation are routinely used in order to enhance the

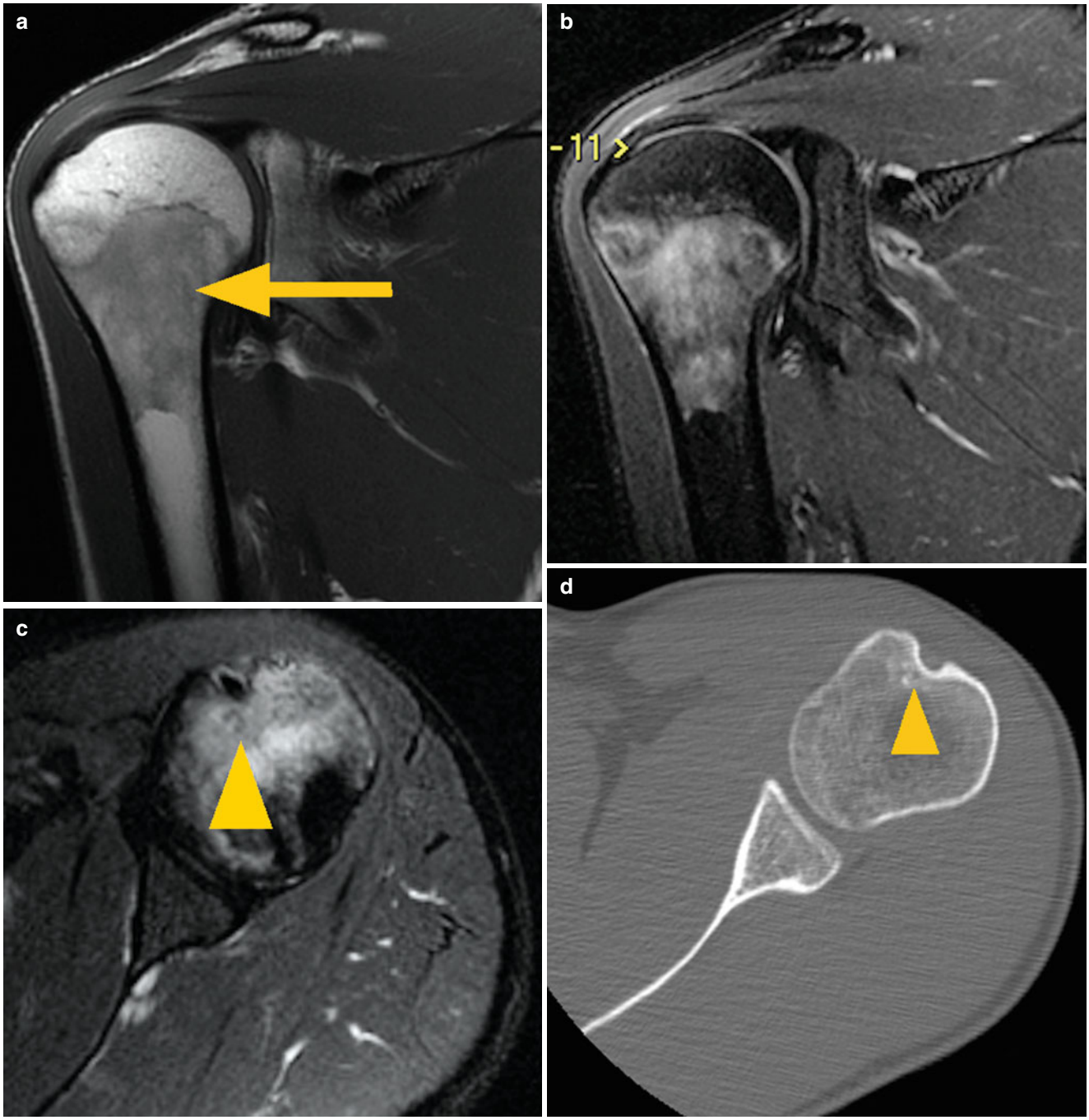

Fig. 4.6 24-year-old patient with predominantly nocturnal pain that lessens with the use of salicylates: clinical suspicion of osteoid osteoma. (**a**) Coronal oblique T1 image shows slight hypointensity at the left proximal humeral metaphysis (*arrow*). (**b**) Oblique coronal T2 FS scan confirms the signal alteration in the proximal humeral metaphyseal region. (**c**) Axial T2 FS. Edema of the spongy bone is documented in the metaphyseal region with small focal hypointense alteration immediately below the bicipital groove (*arrowhead*). (**d**) In order to complete diagnosis, a thin-layer CT scan was performed of the lesion described at MRI. The CT examination documented the presence of "nidus" with central calcification at the same site as the hypointense lesion shown on MRI (*arrowhead*). Examinations confirm the clinical suspicion of osteoid osteoma

contrast of different structures and eliminate the hyperintense signal from the subcutaneous and bone marrow fat. This choice lies mainly in the case of detection of lesions associated with the presence of cellularity or fluid in the form of joint or para-articular effusion or blood serum fluid. Thus the presence of fluid-type signal within a tendon or glenoid fibrocartilage allows the diagnosis of lesions. Moreover, thanks to the contrast between bone and joint

fluid, the presence of the latter in the joint cavity allows to evaluate the cartilaginous coating of the joint surfaces, excluding edematous or abrasive-erosive changes. The injection of articular CM facilitates the diagnosis of chondral injures since it increases the sensitivity of the method allowing the early stages of chondromalacia to be identified.

Chronic inflammatory joint diseases, such as RA, are accompanied by joint and bursal effusions and associated with destructive bone erosions. In this case, intravenous (iv) administration of CM during MRI exam allows the radiologist to evaluate the activity of the disease, documenting the hyperintense thickening of the joint and bursal synovial walls and the hyperintensity of the erosive cavities, or the presence of granulomatous tissue, even when subcutaneous. Glenoid labrum undergoes degenerative changes that appear in the form of focal signal abnormalities on long TR sequences and with globular or lamellar morphology; these alterations may lead to the formation of space-occupying mucinous cysts that may extend medially up to the suprascapular notch causing compression on the neurovascular bundle. MRI can document the presence and extension of this cyst, its nature (mucinous tissue: T2 hyperintense signal), and related signs of neurovascular compromise: venous-vascular ectasia and/or atrophy of the shoulder girdle muscles shown by hyperintense signal on T1 and T2 sequences (fibro-fatty replacement of muscle mass and edema). Finally, T1 morphological study of the shoulder allows the documentation of morphological degenerative bone changes ranging from an "inflammatory" chondral phase up to far more serious phases when the joint capacity reduces, resulting in subchondral sclerosis, geodiform cystic lesions, and foci of necrosis.

Another huge advantage of MRI lies in the evaluation of the postoperative shoulder as it can show many potential causes of recurrent symptoms. However, the evaluation of a postoperative shoulder is not always easy, since the magnetic susceptibility artifacts can alter the homogeneity of the field and "disturb" the evaluation of those structures that are normally assessable at presurgical MRI examination. Artifacts in MR depend on many factors such as the type of metal and the size and complexity of the surface (the higher the complexity the greater the artifact). Moreover, material is just as important, because titanium induces a lower local dishomogeneity in the magnetic field, while steel and cobalt-chromium are responsible for a greater dishomogeneity in the field. Metal anchors determine local artifacts that usually do not alter the assessment of the structures to be examined; in addition, the operator knows that sequences affected by field dishomogeneity, such as GRE sequences, are to be avoided in the study protocol. The use of SE, with low TE, a wider bandwidth, a larger FOV, and a larger matrix, can reduce ferromagnetic artifacts compared to the classical protocol. Biodegradable radiolucent implants commonly used in shoulder surgery induce few or no artifacts [15].

Magnetic Resonance Arthrography

MR arthrography (MRA) of the shoulder is generally indicated in clinical suspicions of shoulder instability, SLAP (superior labrum – anterior to posterior) lesions, intra-articular chondral loose bodies, pathology of the rotator interval (RI), adhesive capsulitis, in the postoperative assessment of surgical procedures, and, to a lesser extent, in case of differential diagnosis between full-thickness and partial-thickness tears of the rotator cuff unaddressed at previous ultrasound and/or standard MRI examination [16].

MRA is generally required after the completion of first-level examinations (standard X-ray and ultrasound) and standard MRI exams, following clinical-specialist evaluation. The completion of the investigation with CT is often recommended in patients with suspected osseous lesion of the glenoid (bony Bankart lesion). In addition, MRA carefully studies the RI (Fig. 4.7). RI structures are intracapsular and may be affected by a variety of inflammatory synovial diseases, such as adhesive capsulitis, rheumatic diseases, and septic arthritis. Adhesive capsulitis is a clinical syndrome characterized by pain and severe functional limitations. It can be idiopathic or secondary to trauma, surgery, osteoarthritis, inflammatory diseases, metabolic diseases (diabetes mellitus), and pathology of the cuff/LHB. Characteristic MRA signs of this disease are the reduced capacity of the joint, difficulty in the introduction of contrast medium into the joint, and thickening of the axillary recess and of the coracohumeral ligament. Another anatomic structure of the RI difficult to detect except with MRA is the pulley of the biceps tendon, in proximity to the intertubercular groove. Its injury may cause medial dislocation of the LHB causing injury to the articular surface of the subscapularis tendon and the anterior superior translation of the humeral head. The coracohumeral ligament constitutes the upper edge of the pulley, while the superior glenohumeral ligament constitutes the inferior edge. The coracohumeral and superior glenohumeral connect the supraspinatus and subscapularis with the pulley. The knowledge of this region helps to explain why the lesions of the upper fibers of the subscapularis tendon involve the superior glenohumeral joint and the medial band of the coracohumeral, whereas lesions of the anterior supraspinatus may involve the lateral band of the coracohumeral ligament. MRA is the only examination that can shed light on the anomalies of the biceps pulley in the RI and shows subluxation or dislocation of the LHB and lesions of the subscapularis and supraspinatus tendons around the pulley [14, 17].

The execution of MRA entails two phases: the first is the intra-articular glenohumeral injection of paramagnetic CM under fluoroscopic guidance and the second consists in the execution of MRI with 1.5 T using arthro-sequences (Table 4.5).

During the intra-articular injection of paramagnetic CM, the patient is supine, positioned on the table of

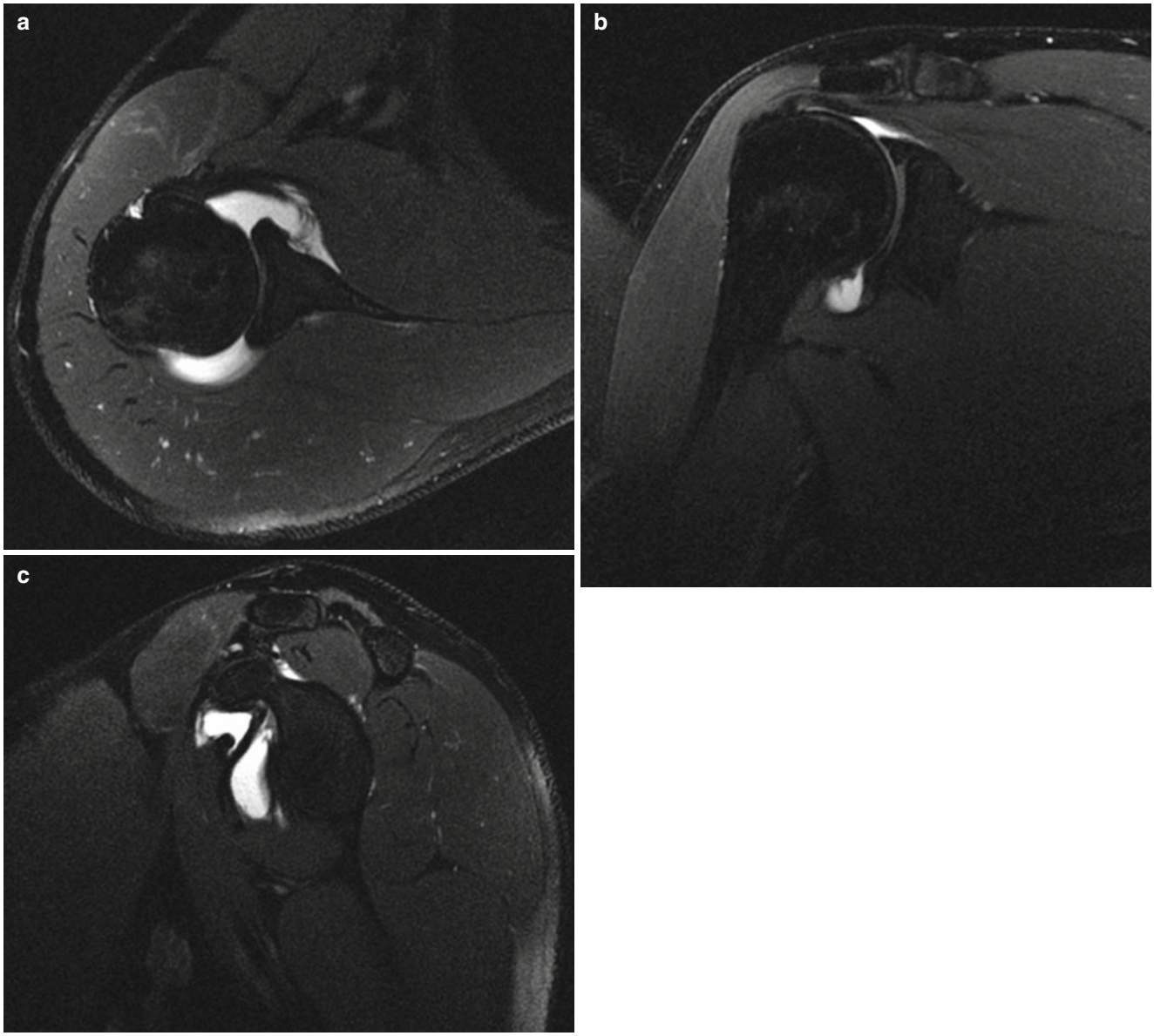

Fig. 4.7 T1w sequences with suppression of fat signal, in the axial (**a**), coronal (**b**), and sagittal (**c**) planes, respectively, after administration of intra-articular paramagnetic contrast medium under fluoroscopic guidance. Good capsular distension can be assessed with involvement of recesses by contrast, relative distension of the LHB sheath and correct view of the capsuloligamentous glenohumeral structures and glenoid labrum

Table 4.5 Sequences for MRA of the shoulder (GE)

MRA sequences (1.5 T GE)	TR/TE (ms)	Thickness (mm)	Matrix	ETL	NEX	FOV(cm)
Axial T1 FS	600/20	4	224×256	4	3	12–14
Coronal oblique T1 FS	600/20	4	224×256	3	3	12–14
Coronal oblique T1	600/20	4	224×256	4	3	12–14
Sagittal oblique T1 FS	600/20	4	224×256	4	3	12–14
ABER T1 FS	600/20	4	224×256	3	4	14

fluoroscopic unit, with her/his arm slightly rotated externally or along the body, in order to make the anterior joint approach quick and easy. The skin is disinfected and the joint is approached under fluoroscopic guidance. A small dose of local anesthetic can be administered (1 % carbocaine) to make the procedure painless. A spinal needle is positioned within the glenohumeral joint having as a radiographic marker the articular margin at the middle-superior

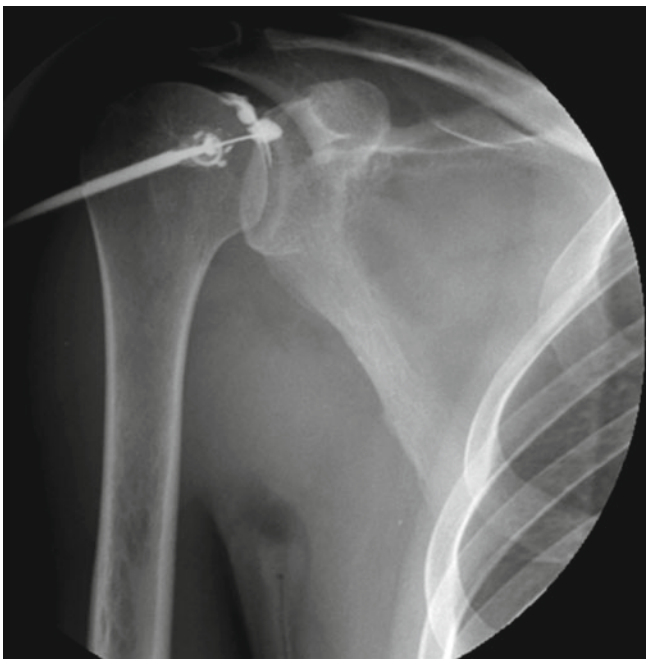

Fig. 4.8 Anteroposterior radiograph of the right shoulder with a spinal needle placement in the intra-articular glenohumeral joint. The ideal entry point of the spinal needle is the superior third of the glenohumeral joint line: linear disposition of radiopaque contrast medium is an X-ray sign of the correct needle placement

third, below and laterally to the coracoid process. About 2 cc radiopaque CM are administered in order to assess the correct intra-articular positioning of the needle, as the joint rim will appear as a radiopaque image between the glenoid and humeral head (Fig. 4.8). We proceed to the administration of 18–20 cc of paramagnetic CM (20:1 dilution). After the procedure, the spinal needle is removed and the skin is disinfected. Finally, the patient is asked to make small movements of the shoulder, in internal and external rotation to facilitate the optimal distribution of the CM introduced. At the end of the procedure, the patient generally refers to a sensation of heaviness and rigidity of the shoulder, in relation to the joint filling of CM. Furthermore, she/he may feel a slight tenderness along the front side of the arm, due to the distension of the LHB sheath, or posteriorly, in correspondence of the scapular region.

In the second phase, the actual implementation of MRI, fat-suppressed T1-weighted sequences are used in the three spatial planes and the investigation can be completed with an oblique scan plane in position of abduction and external rotation (ABER) position. It is useful to complete the MRA using a standard T1-weighted sequence with morphological value.

After the examination, neither functional limitation nor shoulder rest is necessary. In the hours after the examination, mild discomfort in the articulation may persist, which rapidly disappears within 6 h.

The entire procedure must use sterile specialist tools and devices. There is no contraindication to any type of intra-articular injection of paramagnetic CM. There is no need for fasting, nor are blood tests or special precautions required before the procedure. It is sufficient to make sure that the patient is not allergic to iodine, given the albeit minimum administration of iodine contrast agent during the correct positioning of the spinal needle within the intra-articular site.

MRA complications are rare. They include infections, bleeding, allergy, synovitis, and post-procedure pain. Allergic reactions to Gadolinium are rare, although mild/severe reactions have been reported in the literature. A history of adverse reaction to iodine contrast or to anesthetic imposes a premedication or the removal of the substance from the injection. Vasovagal reactions and nausea often occur during the intra-articular injection, whereas bleeding and infection are very rare [18].

In patients carrying pacemaker, MRA can be replaced by CTA examination, which is equally valid for rotator cuff tears but much less sensitive for lesions of the glenoid labrum.

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Biology of Injury and Repair of Soft Tissues of the Shoulder

5

Michael O. Schär and Scott A. Rodeo

Introduction

Pathologies of the shoulder, especially rotator cuff (RC) tears, are increasing in the ageing population. The biology of the different shoulder pathologies is complex and remains poorly defined. However, the understanding of the mechanisms that lead to pathologic changes and healing can lead us to better techniques for treating pathologies of the shoulder. Many factors have been implicated in the development of degenerative changes in the shoulder but also in the healing process. This chapter will outline what we currently understand about the biology of different shoulder pathologies including those of the rotator cuff (RC), the long head of the biceps, and the glenoid labrum.

Rotator Cuff

Biology of the Intact Rotator Cuff

Four Zones of the Intact Tendon-Bone Interface

The tendon-bone attachment in the RC represents a biomechanical challenge due to the fact that the force has to be transferred from the soft tendon tissue with low stiffness to the relatively stiff bone. In the RC, this problem is solved with a special transitional area between the tendon and the bone called the "enthesis" (Fig. 5.1). In this area, the stiffness increases gradually from the tendon to the bone and enables an effective transfer of the mechanical load from the tendon to the bone. The fully formed enthesis is generally described as having four zones:

(I) Tendon
(II) Uncalcified fibrocartilage
(III) Calcified fibrocartilage
(IV) Bone

In these zones, the different collagens and proteoglycans, such as aggrecan, decorin, and biglycan, are not distributed uniformly. There are no sharp boundaries between these zones [1]. This continuous change from bone to tendon enables a transfer of the load between the bone and the tendon. The transition of these four zones occurs over a distance of approximately 1 mm in length. The mechanisms that regulate development of the complex series of tissue gradations are not yet clear.

During tendon-bone healing, the normal enthesis with its four distinctive zones is not restored properly, which results in a relatively high incidence of repair failure. Understanding the natural development of the tendon and its enthesis during embryogenesis may help to improve the healing rate and to reestablish the four zones that connect the bone with the tendon.

Fetal Development of the Native Tendon and the Tendon-Bone Junction

The mechanisms that govern formation of the four zones of the enthesis remain unclear. Little data exists concerning this topic. The tendon and bone of the RC is formed 15.5 days postconception during fetal development. The transitional zone does not develop until 7 days postnatally and turns into a mature fibrocartilaginous enthesis after 21 days postnatally [2].

Fibroblasts of the tendon (zone I) are active during the entire developmental time. Collagen I is expressed by fibroblasts in zone I and II, whereas collagen II is expressed in zone III and IV by chondrocytes. In zone IV, it is expressed up to 14 days postnatally. Close to the insertion, chondrocytes become hypertrophic and start expressing collagen X beginning 14 days postnatally. These hypertrophic chon-

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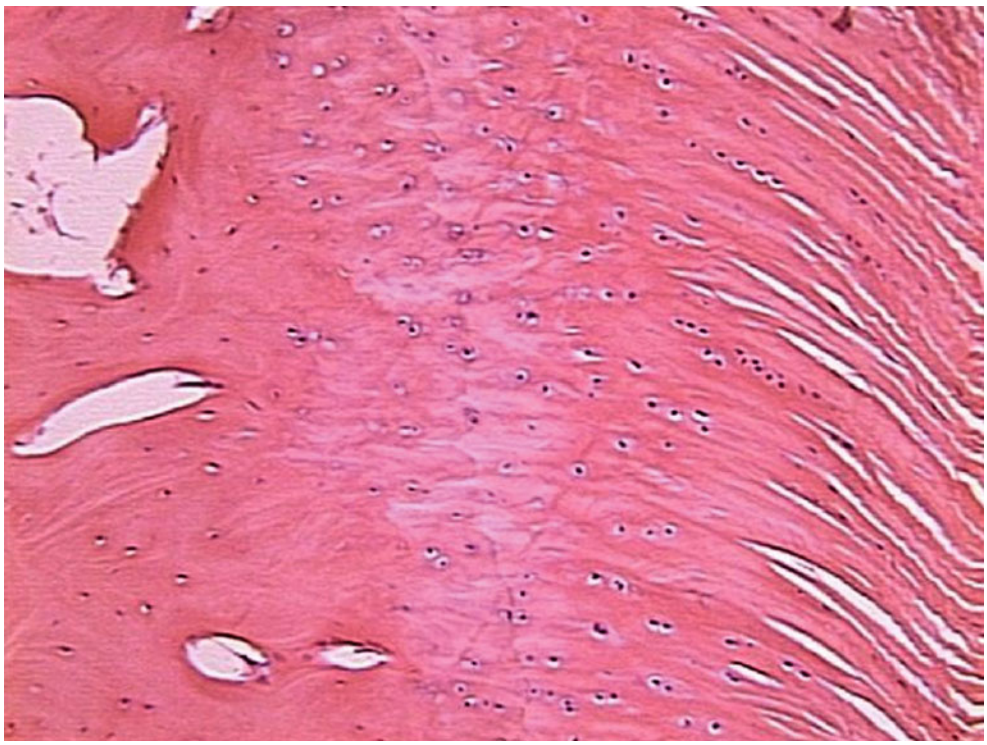

Fig. 5.1 Normal ACL insertion with bone blending into tendon substance via a calcified and an uncalcified fibrocartilage zone (hematoxylin-eosin stain)

drocytes possibly mature into the fibrocartilaginous transition zone. It is hypothesized that collagen X might sustain the transition between the unmineralized and mineralized zones [3].

Several crucial transcription factors are involved in the development of the tendon-bone junction in the RC. Scleraxis (Scx) is a basic helix-loop-helix protein which is a key transcription factor in tenogenesis [4–6], while chondrocyte differentiation is induced by the transcription factor Sox-9 [7]. The size of the developing tendon is regulated by how many cells are expressing Scx. Scleraxis and Sox-9 [8] also appear to play an important role in enthesis formation [4]. The expression of Scx has been detected in the developing insertion site, but Sox9 might also be expressed in the developing enthesis [9]. Both transcription factors are expressed during fetal development as early as 10.5 days postconception [4]. At 15.5 days postconception, scleraxis was more restricted to tendons. Lastly, proteins such as parathyroid hormone-related protein (PTHrP) and Indian hedgehog (Ihh) protein might not only play an important role in the development of the growth plate but also in the development of the tendon-bone enthesis by regulating chondrocyte differentiation [10, 11].

Various members of the bone morphogenetic protein (BMP) family of proteins, in particular BMP-12, BMP-13, and BMP-14 (GDF-7, GDF-6, and GDF-5), also have an influence on tendon development. Mice that are deficient in BMP-12, BMP-13, or BMP-14 show biomechanical, biological, and/or structural abnormalities in the tendon [12, 13]. BMP-4 seems to be another important factor. Its expression in tendons is necessary for the initiation of bone ridges. Blockade of BMP-4 during embryologic development did not abrogate insertion site formation, suggesting that other pathways seem to play an important role [14].

Other factors include members of the transforming growth factor beta (TGF-β (beta)) protein superfamily. These proteins have been shown to have a positive effect on the expression of Scx. For example, in the TGF-β (beta) 2/TGF-β (beta) 3 double mutant or type II TGF-β (beta) receptor null mice, most of the tendons were lost [15]. However, the induction of Scx-expressing tendon progenitors was not affected in these embryos. Fibroblast growth factors (FGF) also influence gene expression in early development. Scleraxis was highly upregulated when an FGF-8-soaked bead was applied in somites [16, 17]. The inhibition of FGF signaling on the other hand caused loss of Scx expression. However, the precise mechanism(s) of the interaction between members of the FGF family and Scx is currently unclear.

Tendon and cartilage emerge from the same precursor cells, and BMP and FGF appear to have antagonistic roles in regulation of the process of differentiation. BMP-2 stimulated chondrogenesis in chicken embryos and inhibits tendon development [5, 18, 19]. Inhibition of BMP lead to tendon differentiation, whereas the inhibition of FGF induces chondrogenesis [5].

Signals from muscle may also play a role in RC tendon. Several studies suggest that signals from myogenic cells are not necessary to initiate the expression of Scx. In a mouse knockout model, in which mice without muscle were bred,

Scx was expressed in the progenitor cells in similar amounts as in normal mice [11, 20]. However, the expression of Scx was not sustained in the setting of a continuous absence of myogenic cells [20, 21]. Scx seems to be regulated by ectodermal signals [5].

Mechanical cues may also play a role in enthesis formation, although little is known about its mechanism [3, 22]. For example, the expression of Scx has been shown to be mechanosensitive, as mesenchymal stem cells that were cyclically loaded responded with an upregulation of the expression of Scx [23]. Further studies are necessary to understand the complex pathways involved in tendon and insertion site development.

Biology of Rotator Cuff Degeneration in Adults

The pathogenesis of the RC tendon degeneration remains an area of controversy. The etiology for degeneration can be divided into intrinsic and extrinsic causes.

Extrinsic Causes for Rotator Cuff Degeneration

Subacromial impingement, resulting in damage of the supraspinatus tendon, was first published by Neer [24] as being an extrinsic cause for RC tendon degeneration. In this situation the tendon impinges on the overlying coracoacromial arch, leading to degeneration and eventual rupture of the tendon. This model is supported by the fact that 72 % of the patients with stage I and II impingement syndrome in one study showed good to excellent results after subacromial decompression [25].

Intrinsic Causes for Rotator Cuff Degeneration

A widely accepted intrinsic model is the degenerative microtrauma model [26], where age-related degeneration leads to increased mucoid deposition, fatty infiltration, a shift from collagen type I to III, and hydroxyapatite microcalcifications [27, 28]. The basic cellular and molecular processes that lead to these changes are currently not well defined. All of these changes adversely affect the material properties of the tendon, leading eventually to a partial- or full-thickness RC tear. Current data suggests that metabolic dysfunction and genetic predisposition may accelerate this process [29, 30].

Tendon hypovascularity is another intrinsic factor that likely contributes to RC degeneration. Two studies show that there is a critical hypovascular zone within 10 mm of the supraspinatus tendon insertion [31, 32]. Interestingly, in the infraspinatus tendon, this zone was only found in the superior portion. This hypovascularity could lead to cumulative degeneration and impaired tendon healing. A recent study reveals evidence that hypoxic damage in the RC may lead to a loss of cells by apoptosis [33].

It has been shown that cytokines such as IL-1β (beta) and enzymes such as MMP-3 are important contributors to the pathology of tendinopathies [34]. Cytokines also play a key role in oxidative stress-induced cellular apoptosis. The role of apoptosis in degeneration of tendon cells has been highlighted. Excessive apoptosis has been noted in the torn edges of the RC tendons. Yuan et al. [35] reported that the number of apoptotic cells in degenerative RC tendon was significantly higher than that in the control group. These apoptotic cells are also found in the perivascular areas. This oxidative stress-induced apoptosis is mediated by the release of caspase 3 and the cytochrome C pathway [36]. Conversely, heat shock proteins seem to protect tendon cells from the cytotoxic effects of apoptotic mediators and cytokines [37].

Biology of the Rotator Cuff Tear

That degenerative processes likely predispose to RC tears as we age is suggested by natural history data. However, high-energy trauma in patients without degenerative changes in the RC can lead to RC tears. After an RC tear, the histopathological changes in the tendon are not restricted to the end of the tendon. A study compared the histopathological changes after RC tears in the intact part of the supraspinatus tendon with an intact tendon control group [38]. The tendon in the tear group showed changes of the collagen with an increased collagen type III content and a loss collagen alignment. A decrease in tenocyte number with associated alterations of the shape of the nucleoli was also reported. Furthermore, increased vascularization was observed. Due to the fact that these changes were not only limited to the tendon end but also to the intact third of the tendon, the authors advise against a debridement of the tendon end during repair. The authors did not correlate the pathological changes with the tear size [38].

Matthews et al. [29] found a decrease in the fibroblast number with an increasing tear size. The number of newly formed vessels and inflammatory cells which are important for the healing process was also reduced in massive tears. In smaller RC ruptures, they found a thickened synovia, which normally indicates an attempt at healing. In massive tears, these findings were absent [29].

Biology of the Rotator Cuff Healing

The healing process can be roughly divided into three phases. In the initial inflammatory phase, there is infiltration of inflammatory cells to the injury site. Within the first 24 h, macrophages and monocytes remove necrotic tissue and release cytokines that have an influence on vascularization, cell migration, proliferation, and differentiation. This phase lasts for several days. After several days, the proliferative phase begins. Cells, including tenocytes and fibroblasts, are recruited to the repair site. Early collagen type III production

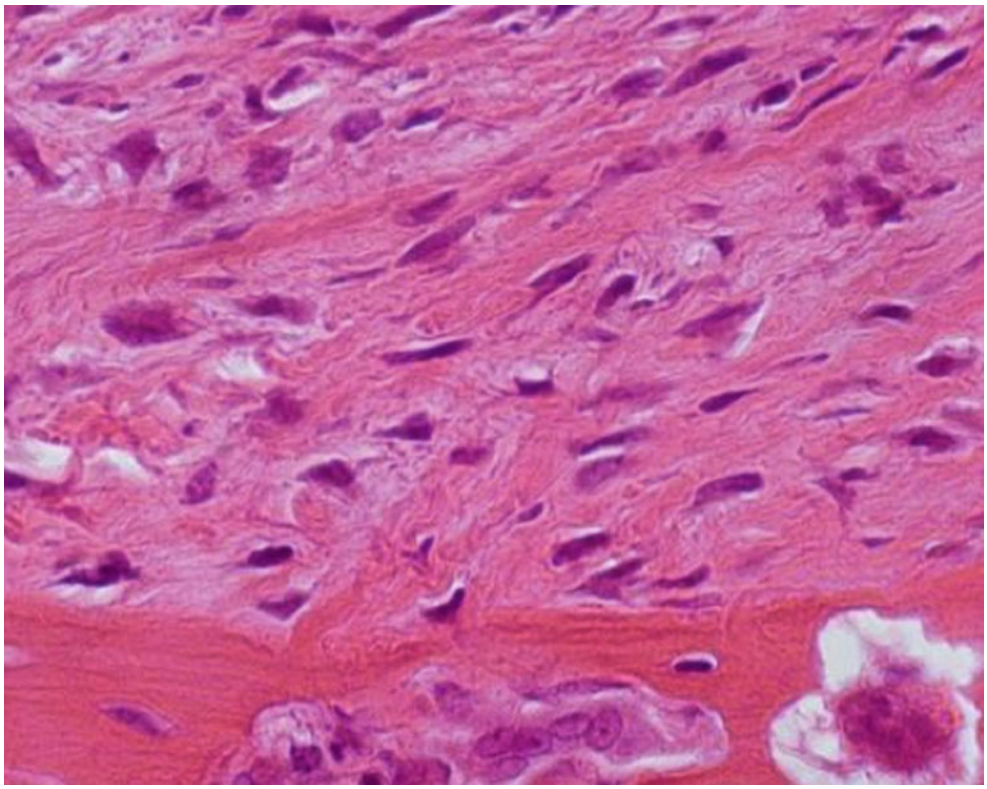

Fig. 5.2 Histology of the healing enthesis after a rotator cuff repair containing interposed fibrovascular tissue. This tissue is gradually remodeled (hematoxylin-eosin stain)

begins in this phase. This phase lasts several weeks and is then followed by the remodeling phase. During the remodeling phase, collagen III is replaced by collagen type I, and the cellularity of the tissue gradually decreases.

In this process, the tendon-bone interface is repaired by reactive scar formation (Fig. 5.2). A recreation of the four zones of the native insertion site does not occur. This scar tissue has inferior material properties compared to the native insertion site. In order to reach a physiological reconstruction of the enthesis, new biological treatment strategies are required. These strategies should address all the requisites that are necessary for healing. Such requisites are (1) intrinsic and extrinsic cells, (2) different growth factors released in the optimal concentration at the right time, (3) ECM proteins, and (4) the optimal amount of load and mobilization. It is also becoming evident that the status of the muscle affects healing capacity.

Influence of Cells on Healing

The hypocellular nature of the tendon and the enthesis may contribute to the poor healing potential of the repaired rotator cuff. Two different cell sources are likely important for healing:

- Intrinsic cells including tenocytes and osteoblasts
- Extrinsic cells including inflammatory cells and mesenchymal stem cells

Hirose et al. [39] reported that intrinsic cells derived from the epitenon of the bursal surface of the tendon migrate into the repair site. Tenocytes synthesize and secrete extracellular matrix proteins [40].

Because these cells exhibit very low proliferative capacities [41], one possible approach to improve tendon healing is to augment the enthesis with extrinsic cells, for example, mesenchymal stem cells (MSCs). These cells have the unique properties of pluripotency and self-renewal. Furthermore, they can be harvested in high numbers from the bone marrow. However, preliminary work in a rat RC repair model found that mesenchymal stem cells did not improve the biomechanical and histological properties [42]. One reason could be that the repair site may lack the signals necessary to induce appropriate differentiation of the transplanted MSCs. This is supported by further work in this model in which MSCs that were transduced with the transcription factor scleraxis before implantation led to improved tendon healing (Fig. 5.3) [43]. Similarly, MSCs genetically modified to overexpress the developmental gene MT1-MMP have also been shown to improve early RC healing by the production of more fibrocartilage at the insertion and improved biomechanical strength [44].

Leucocytes, including lymphocytes, neutrophils, and macrophages, represent another extrinsic cell source that is involved in the early cellular events following tendon repair. These inflammatory cells act in the initial stage of the tendon repair process by secreting growth factors and other soluble mediators, which initiate the repair cascade [45].

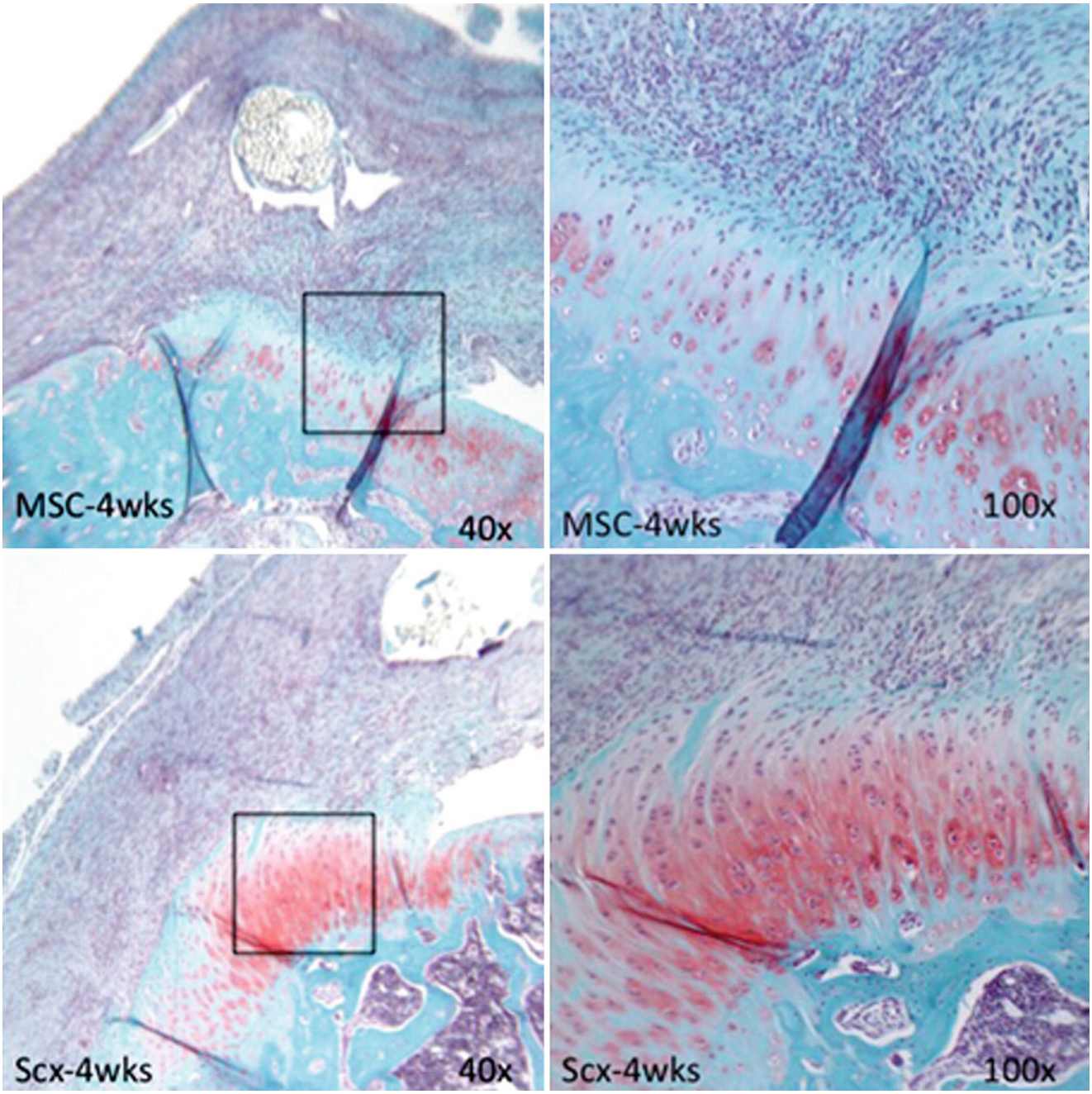

Fig. 5.3 Histology images of cartilage at the insertions site. Slides were prepared with safranin-O/fast green stains that stain the proteoglycans in cartilage a magenta color. There was a greater area of metachromasia found in the Ad-Scx as compared with the mesenchymal stem cell (*MSC*) group at 4 weeks. *Scx* scleraxis (magnification×100) (From Gulotta et al. [43]. Copyright: Dr Scott A. Rodeo. Reproduced with permission)

Influence of Growth Factors on Rotator Cuff Healing

The healing response in RC tears is highly dependent on a coordinated sequence of growth factor expression. Migration of cells into the defect is mediated by growth factors and is important for the healing of the tendon. In the proximal site of the myotendinous insertion, growth factors appear earlier than at the distal defect of the tendon, although they are detectable in the lesion for a longer period [46]. Several studies show that most growth factors return to normal levels 3–8 weeks after the injury [46, 47]. Cytokines involved in tendon healing include transforming growth factor-β (TGF β1), fibroblast growth factor (FGF), bone morphogenetic proteins (BMP), interleukins (IL), platelet-derived growth factors (PDGF), and vascular endothelial growth factors (VEGF). The interplay of these factors is complex.

Table 5.1 Type I and type III collagen ratio

Time point	Group	COLI:COLIII
2 weeks	Control	0.4
	Ca-P	1.1
	TGF-β_3	0.8[a]
4 weeks	Control	0.5
	Ca-P	0.9
	TGF-β_3	1.1[a]

COLI:COLIII type I to type III collagen ratio, *Ca-P* calcium phosphate matrix, *TGF-β_3* transforming growth factor–beta 3
[a]Denotes significance

Transforming Growth Factor-β (Beta)

Transforming growth factor beta TGF-β (beta) is produced by all the cells that are involved in the healing process, whereas normal tendons show low concentrations of TGF-β (beta) [48, 49]. The TGF-β (beta) 1 isoform is involved in adult wound healing and leads to the formation of scar tissue. In contrast, in early fetal "scarless" wound healing, TGF-β (beta) 3 is increased. TGF-β (beta) 1 remains highly upregulated for the first 8 weeks following tendon injury and repair in the adult wound [47, 49]. It is active in the healing process during all phases [46, 49]. The negative effect of the TGF-β (beta) 1 isoform was shown in a study where TGF-β (beta) 1 was delivered via an osmotic pump in a rat supraspinatus tendon tear model. An increase in type III collagen production was seen, indicative of a scar-mediated response [50]. Kovacevic et al. [51] reported that RC reconstructions that were augmented with an osteoconductive calcium phosphate matrix containing TGF-β (beta) 3 showed a significantly improved strength of the repair at 4 weeks postoperatively and resulted in a more favorable collagen I/collagen III ratio, when compared to the group where the augmentation was performed only with calcium phosphate matrix (Table 5.1). Manning et al. [52] reported similar results.

Fibroblast Growth Factor

Members of the fibroblast growth factor family may affect tendon-bone healing. FGF-2, which is also known as bFGF, has a role in the formation of granulation tissue. bFGF is produced not only by leukocytes but also by tenocytes and fibroblasts [48, 49]. It remains highly upregulated during the entire healing process but has its peak between the seventh and the ninth day. This factor stimulates the proliferation of RC tendon cells (RCTC) in a dose-dependent manner and suppresses the secretion of collagens from RCTC in vitro [53]. Several authors reported improved tendon healing after the addition of bFGF [54, 55]. Local application of bFGF using an acellular dermal matrix graft led to a significant increase in strength and tendon maturity at 6 and 12 weeks postoperatively [56, 57]. On the other hand, Thomopoulos et al. [58] showed in an intrasynovial flexor tendon canine model that the administration of bFGF failed to produce improvements in either the mechanical or the functional properties of the repair. They also found an increased vascularity, cellularity, and adhesion with an increase in peritendinous scar formation.

Bone Morphogenetic Protein

It has been proposed that the formation of the enthesis is similar to that of the enchondral ossification [59]. It has been shown that in the tendon-bone healing, the ingrowth of bone into the tendon is of utmost importance [60]. For this reason, the augmentation of RC reconstructions using osteoinductive growth factors has been proposed. The prototypical osteoinductive factors are members of the BMP family. Except for bone morphogenetic protein-1 (BMP-1), which is a metalloprotease, all BMPs belong to the TGF-β (beta) superfamily.

BMP-2, for example, increased the collagen I production in an in vitro study on tenocyte-like cells isolated from human RC tissue samples [61]. An increase in collagen type I production and expression, as well as increased cell activity, was observed for BMP-7. The combination of BMP-2 and BMP-7 resulted in smaller changes compared to the use of BMP-7 alone [61]. The application of BMP-2 in an injectable hydrogel into a ruptured RC resulted in a significantly higher maximum pullout load at 4 and 8 weeks postoperatively [62].

BMP-14 can be localized to the bursal side of the tendon and the tendon edge in the histologic examination of full-thickness RC tears in humans [63]. When adipose-derived or bone-marrow-derived MSC were treated with BMP-14 in an in vitro model, a higher proliferation rate was observed, along with an increased differentiation towards a tenocyte phenotype [64, 65]. In contrast, MSCs that were treated with BMP-13 differentiated into chondrocytes [66]. The addition of rhBMP-12, rhBMP-13, and rhBMP-14 induced neotendon formation when implanted at ectopic sites in vivo [67]. In an animal model, the application of rhBMP-12 and rhBMP-13 enhanced RC tendon-bone healing [68, 69].

Matrix Metalloproteinases and Tissue Inhibitor Metalloproteinases

Matrix metalloproteinases (MMPs) belong to the family of proteases and are catabolic enzymes. They are capable of degrading all the components of the extracellular matrix (ECM) such as collagen. In normal healthy tissue, there is a balance between the MMPs and the tissue inhibitors of metalloproteinases (TIMPs). An imbalance between these two factors produces a collagen dysregulation with subsequent adverse effects on matrix material properties. Data from unloaded healing flexor tendons of rats suggest that

MMP-9 and MMP-13 mediate tissue degradation during the early phase of healing, whereas MMP-2, MMP-3, and MMP-14 (MT1-MMP) mediate tissue degradation and later remodeling [70]. The time course of the expression of MMPs and TIMPs during RC healing was first reported by Choi et al. [71].

In a clinical study performed with patients that had undergone recent RC reconstruction, a significantly higher expression of MMP-1 and MMP-9 was found in the supraspinatus tendon in the non-healed group compared to the healed group [72]. Also MMP-13 protein levels were increased in torn RC tendons, and they showed a proportional correlation with the patients' pain score [73]. It has been shown previously that MMP-13 plays a role in diseases characterized by excessive degradation of the ECM, including osteoarthritis and rheumatoid arthritis [74]. However, the balance between MMPs and their inhibitors is complex and requires further study. Specific MMPs delivered at a certain time point may have a positive effect on healing. For example, MSCs that were genetically manipulated to overexpress MT1-MMP (MMP-14) improved the RC healing in a rat model [44].

There are many unanswered questions about the optimal doses and combinations of various cytokines, timing of delivery, and the ideal delivery vehicle. Furthermore, the complexity of wound healing, including inflammation, cell proliferation, matrix synthesis, and remodeling, suggests that healing may be best optimized by a combination of factors.

Influence of the Extracellular Matrix on Healing

Proteoglycans and collagens are the structural components of the ECM in tendon and enthesis. 95 % of all the collagen in tendons is type I collagen. In the rest of the enthesis (zone II–III), the collagen organization is less parallel than in the tendon itself [75]. Type I collagen is found mainly in zones I and IV (bone and tendon) [2]. It is also found temporarily in the stage of remodeling [49]. Type II collagen is found during fetal development in zone IV and postnatally in zones II and III (fibrocartilage zones) [2, 76]. Type III collagen is found in zones I and IV [77]. It is present during the early stages of the healing process [78]. In the healing of tendons and bone, collagen type III is associated with the early scar tissue formation [78, 79]. It is not yet clear to what extent excessive levels of collagen III can impair insertion quality. The fibril diameter of collagen type I is regulated by type V collagen [78], and therefore, type V collagen can be found predominantly in zones I and IV [80, 81]. The fibril-related type IX collagen is located predominantly on the osseous side of the insertion and connects mainly with type II collagen.

Collagen type X is produced during human development by hypertrophic chondrocytes in the mineralized fibrocartilaginous transitional zone III [2, 80]. Collagen X seems to play an important role in the conversion of unmineralized to mineralized tissue due to the fact that it persists in zone III (mineralized fibrocartilage region), even if the hypertrophic chondrocytes are no longer present.

It is interesting to note that collagen X is not present in adult enthesis healing [2, 80]. It is in fact not produced until zones II and III have been developed. The role of mechanical stimulus in the expression and production of collagen X is supported by the fact that in tendons of patients with paralyzed shoulders, the formation of a fibrocartilaginous transitional zone is disturbed [82]. Collagen XII is situated on both sides of the insertion [83] and belongs, like collagen type IX, to the fibril-associated group. It binds to collagen type I [80].

Proteoglycans also contribute to the tissue regulation of the ECM. Biglycan, for example, is a tendon-specific ECM protein. It is only found in zone I of the enthesis area [77, 80, 84]. Biglycan forms bridges between the collagen fibrils and increases their stability. By influencing fibrillogenesis, it also determines their structure [83]. Biglycan is involved in organizing the niche for tendon stem/progenitor cells in mammalian tendon. In double null mice lacking both biglycan and fibromodulin, a local increase of BMP signaling was detected, which favors local chondrocytic/osteoblastic differentiation. It also promotes ectopic endochondral bone formation and impairs tendon formation in the young adult [85].

Aggrecan is a cartilage-specific ECM protein analogous to versican, which is only found in the unmineralized fibrocartilage region of the enthesis [76, 77, 80, 84]. The production of aggrecan is triggered by compressive stress [2] which is why this protein is predominant in zone III [80]. Aggrecan strongly binds water [80].

Decorin is another important proteoglycan. It regulates the collagen fibril diameter, similar to type V collagen. It is found mainly in zone I and II of the intact insertion [78, 80, 83]. During the healing process, decorin is found at reduced levels but can still be detected. It contributes to the stabilization of the collagen structure by building bridges between collagen fibrils and by influencing fibrillogenesis through inhibition of collagen type I formation [80, 83]. It also regulates the activity of TGF-β (beta) [80].

Influence of Load and Mobilization on Enthesis Healing

Several studies show that load has a profound effect on tendon and enthesis healing. It is well established that mechanical stress improves the tensile strength, stiffness, and cross-sectional area of tendons [86, 87]. This is most

probably caused by an increase in collagen and ECM synthesis by tenocytes [87]. If collagen is not being stressed during the proliferative and remodeling phase, it is weaker and less organized compared to collagen under tensile load. Repetitive motion increases DNA content and protein synthesis in human tenocytes [88]. At the same time, application of strain to tenocytes produces stress-activated protein kinases, which in turn triggers apoptosis, demonstrating the complexity of the tenocyte response to load [89, 90].

Influence of Muscle Changes on Rotator Cuff Healing

After an RC tear, the muscle retracts, atrophies, and is infiltrated by fat. These muscle changes have significant effects on rotator cuff tendon healing and represent important prognostic factors for the success of an RC reconstruction.

Retraction

The retraction of the musculotendinous unit is generally considered to be an important pathophysiological consequence after an RC tear. Several studies show that a pronounced retraction leads to a higher retear rate [91–93]. It has been shown that continuous elongation of a retracted, fatty infiltrated, and atrophied musculotendinous unit is technically feasible in the sheep infraspinatus model [94]. There is evidence that up to Goutallier stage III, retraction of the supraspinatus muscle is caused mainly by the muscle. In advanced stages, the retraction results also from shortening of the tendon tissue itself [95]. Due to this shortened tendon stump, the muscle has to be stretched beyond its physiological length in order to compensate for the tendon retraction. When the muscle retracts, the pennation angle increases and therefore gaps form between the single muscle fibers. As a result, fat is deposited into these gaps.

Fatty Degeneration

It is currently unclear if fatty infiltration is part of the normal age-related degenerative process of tendinopathy or appears due to the failed biological repair mechanism. Fatty infiltration is found after RC tears as well as after a neurogenic lesion. It is found in different areas of the musculotendinous unit, including the intramuscular compartments [96]. Here the accumulation of fat leads to a reduction of the mechanical properties [97]. Fatty infiltration is also found in the extramuscular space and in the torn tendon [98]. It is notable that fat is deposited not only around the muscle fibers but accumulates in type 1 muscle also within the sarcoplasm [96]. Even though fatty infiltration is an important factor for the outcome of RC repairs, little is known about the etiology of the process. Understanding the pathophysiological mechanisms that lead to fatty degeneration is mandatory in order to determine potential strategies that may ultimately be able to reverse these adverse changes.

Several theories have been proposed which try to explain the mechanism of the formation of fatty infiltration. Peroxisome proliferator-activated receptor gamma (PPARγ (gamma)) is one of the central regulators of adipogenesis. PPARγ (gamma) is a ligand-activated transcription factor that not only plays an important and central role in adipose cell differentiation [99] but also in the control of macrophage function, immunity, and cell proliferation [100]. Studies show that PPARγ (gamma) is not only necessary but also sufficient for fat cell differentiation [101]. The adipogenic transcription factor CAAT/enhancer-binding protein β (C/EBPβ (beta)) also seems to play an important role in the formation of fatty infiltration. It is induced during early adipocyte differentiation and is able to transactivate adipocyte genes. Furthermore, like PPARγ (gamma), it is able to inhibit Myf-5, a myogenic transcription factor [99].

A neurogenic cause for fatty degeneration is also discussed in the literature. Vad et al. showed that 25 % of the 28 patients with a complete RC tear had an abnormal electromyogram, which is suggestive for a peripheral neuropathy [102]. The tensile stress on the suprascapular nerve after an RC tear may promote atrophy and fatty degeneration. This is supported by the fact that in tears of the supraspinatus tendon, the intact infraspinatus muscle also showed fatty degeneration [103].

Other authors suggest that the change in the muscle architecture after an RC tear may make the muscle vulnerable for fatty changes. These changes were documented in animal studies. When the muscle retracts, the muscle fibers shorten, the pennation angle increases, and as a result interstices are formed between the muscle fibers. This enlargement of the space between the muscle fibers could be perceived as an injury with the result that fat is deposited into these interstices [97].

The force of the supraspinatus muscle is directly proportional to the degree of fatty infiltration [95]. This suggests that the loss of the contractile force is not only caused by the muscle atrophy but also by the degree of fatty infiltration. The change of the pennation angle supports this theory. An increase of this angle and subsequent integration of fat into the gaps reduces the load-transfer ability due to the fact that the force vector is almost perpendicular to the muscle fiber axis.

Atrophy

After an RC rupture, in addition to fatty infiltration, asymmetric muscle atrophy also develops [104]. The reduction of the muscle diameter after tenotomy is established as a prognostically important factor in therapy of an RC lesion [105]. However, the slow-twitch type 1 muscle fibers are more affected than the fast-twitch fibers (type 2A), whereas the type 2B fibers atrophy the least [97, 106]. Furthermore, the intramuscular fibrotic tissue also increases [106]. After a

short time period, the muscle shortens and starts to lose the capability to build up tension [107]. Sixteen weeks after having performed a tenotomy in the sheep, the infraspinatus muscle was retracted 29 mm, corresponding to the physiological range of motion in the muscle. In doing so, the muscle fibers were shortened and the pennation angle increased from 30° to 55°. The muscle diameter decreased to 57 % compared to the healthy opposite side. Due to the chronic retraction, the muscle fibers become shorter up to 50 % by the breakdown of serially arranged sarcomeres. Only the muscle length decreases while the diameter remains unchanged because the amount of fibers remains the same. The degeneration of the muscle is in reality more a reduction of healthy functional muscle tissue and not a degeneration in the proper sense [97]. This was confirmed by another study where it could be shown that the cause for atrophy in RC tears greater than 3 cm is not caused by muscle fiber death but by decrease of the absolute myofibril volume [96].

At the gene expression level, key regulators like muscle RING-finger protein-1 (MuRF1) and Atrogin-1, which are able to induce muscle atrophy, are upregulated shortly after tenotomy and returned to normal shortly after [108, 109]. On the other hand, several genes that are involved in muscle atrophy are massively upregulated in large RC tears compared to smaller tears, for instance, cathepsin B (CTSB), calpain, ubiquitin-conjugating enzyme-E2B (UBE2B) and ubiquitin-conjugating enzyme-E3A (UBE3A), and Forkhead box protein O1A (FOXO1A). This upregulation could be one of the reasons why massive tears have a poorer healing rate than smaller tears.

Exogenous Factors That Affect RC Healing

This section investigates some of the exogenous factors that impair or improve the healing of the RC.

Nonsteroidal Anti-inflammatory Drugs

Nonsteroidal anti-inflammatory drugs are commonly prescribed after RC repair. Although the inflammatory process contributes to healing by formation of reactive scar tissue, inflammation is a fundamental response to injury. Thus, blockade of the inflammatory process may have an adverse effect on healing. Indomethacin and celecoxib both significantly inhibited tendon-bone healing in a rat supraspinatus repair model [110]. There were significant differences in collagen organization and load to failure between the nonsteroidal anti-inflammatory and the control groups [110].

MMP Inhibition with Doxycycline

Matrix metalloproteinases may adversely affect development of new tissue at the healing tendon-bone junction. In one study, doxycycline, which is a broad-spectrum inhibitor of MMPs, was administered orally in rats after RC repair. Rats treated with doxycycline showed a reduced MMP-13 activity 8 days postoperatively, improved collagen fiber organization, and an increased load to failure after 2 weeks [111]. These data suggest the possibility of modulation of MMP activity to improve tendon healing.

Diabetes

Bedi et al. found in 2010 [112] that sustained hyperglycemia impairs tendon-bone healing after RC repair in a rodent model. Diabetic animals demonstrated significantly less-organized collagen and less fibrocartilage with a decreased ultimate load to failure (Fig. 5.4) [112].

Steroids

Local steroids are widely used in treatment of shoulder pain. A meta-analysis showed that subacromial injections of corticosteroids are safe and effective for improvement of RC tendonitis. They also seem to be more effective than NSAID medication [113]. However, it is established that corticosteroids can have adverse effects on collagenous tissues. For example, studies in rats show that local corticosteroids weaken both intact and injured RC tendons [114].

Nicotine

Nicotine has also been documented to impair tendon healing. In a rat model, the systemic application of nicotine using an osmotic pump caused a delay in tendon-to-bone healing in the supraspinatus tendon. The inflammatory response lasted longer in the nicotine group. There was increased early cell proliferation in the saline-treated controls, and type I collagen expression was higher in the controls compared to nicotine-treated animals. Biomechanical properties increased in both groups over time, but in the nicotine group, they lagged behind those of the control group [115].

Long Head of the Biceps Tendon

The most common pathology affecting the long head of the biceps (LHB) is tendinopathy (Fig. 5.5), leading to partial or complete tears. Hypertrophic tendinopathy may result in entrapment of the tendon within the glenohumeral joint. An "hourglass biceps" may result in an inability to slide through the bicipital groove, causing locking and pain of the shoulder. In up to 90 % of cases, these disorders are associated with RC tears [116, 117]. Glenohumeral arthritis may also be associated with pathologies of the LHB [116].

Very few studies have investigated the histopathological and biochemical changes in the LHB.

In LHB tendinopathies, chronic inflammation is found with a decrease in the number of fibrils in the distal portion of the tendon [117]. This decrease did not correlate with

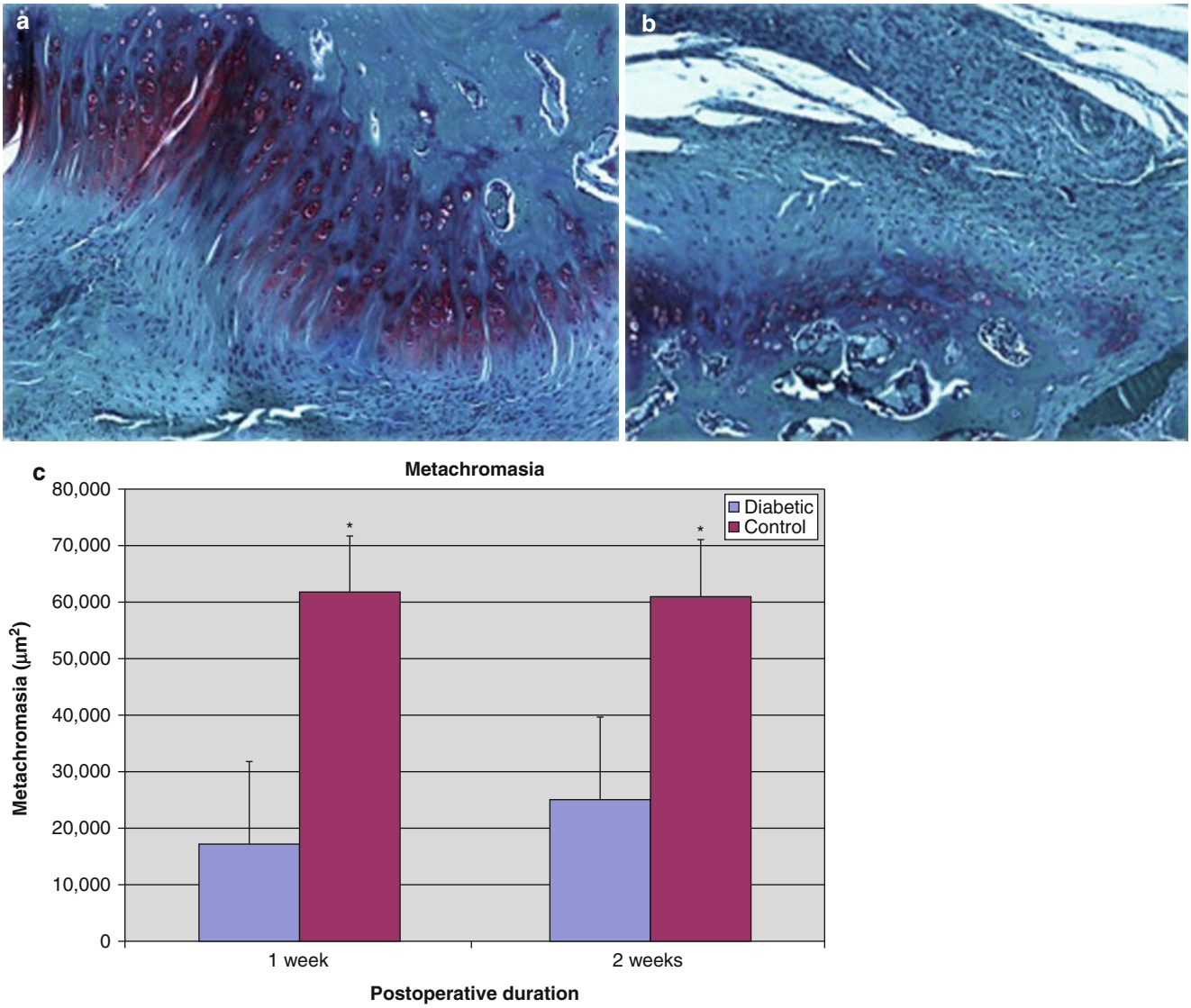

Fig. 5.4 Fibrocartilage formation. (**a**) Control enthesis (2 weeks, 40× magnification). (**b**) Diabetic enthesis (2 weeks, 40× magnification). (**c**) Quantitative histomorphometry revealed that the diabetic animals had significantly reduced fibrocartilage at the healing enthesis compared to control animals at both 1 and 2 weeks postoperatively (17,254 ± 14,957 μm^2 versus 61,724 ± 10,493 μm^2 and 25,025 ± 14,705 μm^2versus 61,000 ± 9,175 $\mu m^2$for 1 and 2 weeks, respectively) ($P < .05$) (From Bedi et al. [112]. Copyright: *Journal of Shoulder and Elbow Surgery*; Published by Mosby, Inc. 2010. Reproduced with permission)

clinical examination findings including pain. In another study, the presence of sensory and autonomic neuropeptides more in the proximal end of the LHB tendon was reported [118]. This suggests that the proximal end of the LHB tendon is innervated by a network of sensory sympathetic fibers, which may play a role in the pathogenesis of shoulder pain by regulating the blood flow and also participating indirectly in the inflammatory reaction.

Looking at patients with partial or full-thickness RC tears, Murthi et al. found a high incidence of microscopic chronic inflammation and gross degeneration in the LHB tendon [116]. These pathologic changes were directly proportional to the extent of RC disease. LHB tendon ruptures typically occur in the hypovascular zone 1.2–3 cm from the tendon origin [119]. Kannus and Józsa found in spontaneous ruptured LHB tendons preexisting hypoxic degeneration in 49 %, mucoid degeneration in 22 %, and tendolipomatosis only in 4 %, either alone or in combination [120].

Glenoid Labrum

Pathologies of the glenoid labrum are most commonly seen in overhead throwing athletes such as baseball players. The labrum consists of a narrow band of tissue. This tissue is distinct from the fibrous shoulder capsule in adults and the

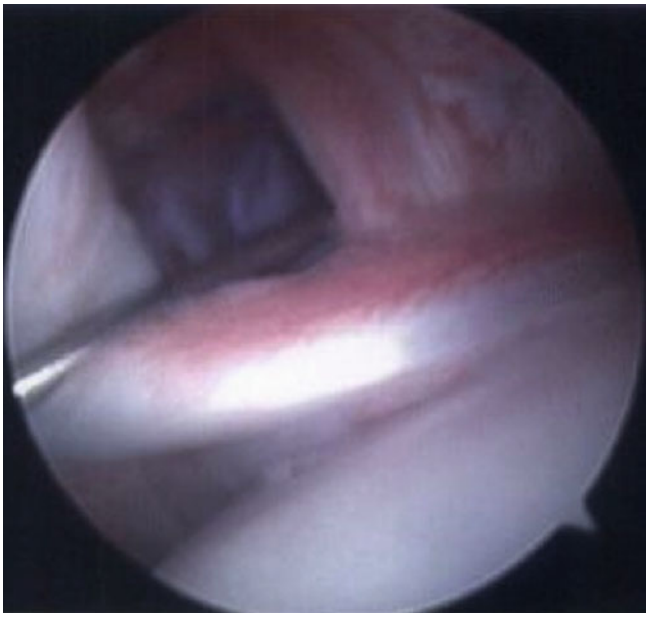

Fig. 5.5 Arthroscopic image shows LHB tendinosis with tendon thickening and flattened appearance

hyaline articular cartilage on the glenoid. It contains a fibrocartilaginous transition zone between the hyaline cartilage of the glenoid cavity and the fibrous labral tissue. The superior part of the labrum is comparable to the meniscus of the knee containing an attachment that stretches easily. The inferior part contains more inelastic fibrous tissue and acts therefore as a rounded extension of the articular cartilage [121].

The vascular supply of the labrum arises from the suprascapular artery, the circumflex scapular branch of the subscapular artery, and posterior humeral circumflex artery. Vessels penetrate the labrum in a radial and circumferential pattern. The inner third of the labrum though is avascular, similar to meniscus in the knee. Vascularity decreases with increasing age [122]. No metabolic data exists regarding the glenoid labrum. The glenoid labrum is comparable with the acetabular labrum. The fibrochondrocyte-like cells in the acetabular labrum are uniquely highly active in patients who underwent total hip replacement due to degenerative joint disease. These cells were shown to express and release cytokines and inflammatory enzymes like MMP-1/-2/-9, ADAMTS-4, and IL-6 and react to a pro-inflammatory stimulus [123].

Summary

The biology of injury and repair of the soft tissue in the shoulder is complex, and many unanswered questions remain. Due to the fact that our clinical success is dependent on a comprehensive understanding of the biology of healing, it is critical to develop an understanding of these processes. This will help in discovering new pathways to augment tissue healing and maybe even ultimately allow tissue regeneration and "scarless" healing, with restoration of biomechanical properties comparable to those of native soft tissue.

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Principles of Shoulder Rehabilitation

6

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Introduction

It is well known that the shoulder, by combining the actions across the glenohumeral, scapulothoracic, acromioclavicular, and sternoclavicular joints, provides a unique wide range of functional versatility to the upper extremity, making it prone to injuries and dysfunctions.

The strategies of shoulder rehabilitation have changed dramatically over the past two decades: thanks to a better knowledge of its complex arthrokinematics, as well as dealing with selective and well-known conditions (we are at last abandoning the term scapulohumeral periarthritis), physical therapy has reached elite standards in quality and effectiveness, thus allowing better, safer, and earlier restoring of function and painless motion. Nevertheless, all conservative and postsurgical approaches have been fine-tuned, and a good team approach is now a reality almost everywhere. For instance, physical management of shoulder pain has progressed from addressing single structures, such as the supraspinatus tendon with cross-frictions [1], to a multistructural approach, considering not only potential sources of symptoms but all possible contributing factors towards the etiology of injuries, maintenance of symptoms, and recurrence. So it is important to remember that, as experts at restoration, we need to be reminded of the importance of prevention: anticipated complications need to be expected and prevented. This is the golden concept of preventive rehabilitation. Never forget that we have to rehabilitate a patient, not her/his lesions, dysfunction, or MRI.

It is imperative to remember that all the guidelines and rehabilitative strategies have to be tailored to the single subject, taking into account a lot of variables such as age, gender, level of activity, pathoanatomy, and surgical procedures. A thorough knowledge of anatomy and biomechanics as well as the correct application of manual therapy, exercise, and modalities will lead to better outcomes.

In this chapter we decided to focus on those shoulder conditions more susceptible to arthroscopic treatment, such as conflicting and unstable shoulder, disorders of the acromioclavicular joint and long head of biceps, rotator cuff repair, and stiff and frozen shoulder.

General Principles

Early motion, to the extent allowable, is crucial. Strengthening should be gradual and progressive. Repetitions should be increased first, followed by increases in resistance. Exercises for strength, endurance, and power must be balanced. Stabilization activities and exercises with weight bearing (closed kinetic chain) promote effective and functional strengthening (Fig. 6.1). The deltoid, scapular stabilizers, and rotator cuff muscles each have unique mechanisms, and the exercises designed to strengthen them must take these into account. Strengthening for the serratus anterior can be accomplished through many different techniques, each of which has a different impact on the scapula and the percent of maximal voluntary contraction of the muscle. Isometric exercises are usually tolerated better by arthritic joints. Age affects muscle and endurance. Aerobic exercise benefits nearly everyone recovering from shoulder surgery, and patients should be encouraged to be as active as possible. Exercises should be designed to address impairments and progressed to approximate closely the desired level of function.

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Fig. 6.1 Closed kinetic chain exercise on balance board

During an upper limb movement, corticospinal system generates a motor program that activates muscles in coordinated sequences to create joints motion. This motor program must create the optimal conditions of stability at the proximal joint of the upper limb aimed at generating and transferring forces to the distal segments in an efficient manner. If these stable conditions are achieved, the rapid upper limb movements will not disturb body equilibrium during overhead activities. The pathological conditions of the shoulder may impair this motor program producing subtle compensatory changes in the normal muscle activation patterns exerted during the upper limb movements, especially those rapid and overhead. Therefore, correction of abnormal motor control and restoration of correct muscle activation pattern is imperative in the functional reeducation of shoulder impairments. Setting goals and providing feedback on progress towards those goals assist with motivation and compliance with the exercise program. Graphs of range of motion and tracking repetitions are helpful, especially when targets for these relate to desired functions.

Small increments in flexibility may be difficult to appreciate functionally: goniometric measures provide feedback on those gains. Modalities may be helpful to deal with pain, inflammation, and swelling especially in the early phases of physical therapy. During the rehabilitation process, the patient should be instructed to produce and control the movement in a painless functional range, in which the motion is performed in a coordinated manner.

Many systems ore scores are available to measure outcome, but this topic will be discussed elsewhere in this book.

Subacromial Impingement

Subacromial impingement is a common shoulder impairment. It occurs when the area between the superior humerus and inferior acromion process is diminished, resulting in compression of the interposed tissues including supraspinatus and long head of biceps (LHB) tendons, subacromial bursa, and shoulder capsule [2, 3]. This encroachment results in shoulder pain which is exacerbated by forward elevation and rotation of the upper extremity. Subacromial impingement can result in various stages of rotator cuff disease which range from mild tendon irritation to complete tendon tears. Causes can be subdivided into structural and functional mechanisms often referred to as primary or secondary impingement. Two primary mechanistic theories suggest specific fatigue-related kinematic changes that reduce the subacromial space: superior head migration and altered scapular kinematics [4, 5]. A poorly functioning rotator cuff, alterations in the position of the scapula due to weakness of scapular stabilizer muscles, impaired scapulothoracic mobility, and tight pectoralis minor may increase anterior tilting of the scapula leading to effectively reduce the subacromial space and so producing a functional impingement.

It is important to point out that shoulder impingement is not a sport-specific disease. Awkward working postures,

specifically working with the arms above shoulder level, can cause or worsen shoulder disorders. Indeed, working overhead has been linked to a lot of negative physiological and biomechanical consequences, with increased intramuscular pressure, impaired circulation, increased muscle activity, and fatigue development. So a good rehabilitation program has to reduce or modify those factors within overhead work which can enhance the risk of musculoskeletal injury.

Nonoperative Treatment

Acute Phase

The main goals of the acute phase of rehabilitation program are to relieve pain and inflammation, prevent muscle atrophy, reestablish painless range of motion, and normalize arthrokinematics of the shoulder complex. This phase may include a brief period of active rest, eliminating any activity that may cause increase in symptoms. Range of motion (ROM) exercises may include pendulum Codman's and active assisted exercises.

Relative rest may also be important in the reactive stage of rotator cuff tendinopathy [6]. Joint mobilization may be included with inferior, anterior, and posterior glides in the scapular plane. Neuromuscular control exercises are recommended with particular emphasis on scapular stabilizers, then isometric exercises for the external and internal rotators and biceps. Modalities such as cryotherapy, transcutaneous electrical nerve stimulation, and phonophoresis may be useful as adjunctive treatment. It is important to remember that use and diffusion of modalities may vary from country to country. To further reduce upward humeral head translation and tendon compression, avoidance of internal rotation in the early stages of rehabilitation may be appropriate. Patient education is particularly important in this acute phase: overhead activities, lifting, and reaching have to be avoided. Criteria for progression to the recovery phase are decreased pain or symptoms, increased ROM, and improved muscular function.

Recovery Phase

The initial goals of this phase are to normalize ROM and shoulder arthrokinematics, perform pain-free activities of daily living, and improve neuromuscular control and muscle strength. ROM exercises should be progressed to active work in all planes and self-stretches focused on the posterior joint capsule.

Strengthening should include isotonic resistance exercises for the supraspinatus (dumbbell or tubing), internal and external rotators, prone extension, horizontal abduction, forward flexion to 90°, upright abduction to 90°, shoulder shrugs, rows, push-ups, and pull-downs to strengthen the scapular stabilizers. It may be possible to enhance the effect of exercise by including manual therapy in the treatment package [7].

Upper extremity ergometer exercises for endurance, trunk exercises, and general cardiovascular conditioning should be maintained. When full painless ROM is achieved and muscle strength is approximately 70 % of contralateral side, patient may progress to the next phase, whose goal is to get the athlete back to throwing and nonathletes back to overhead activities. This phase should include improving strength, power, and endurance and sport-specific neuromuscular control. Emphasis is placed on high-speed, high-energy strengthening exercises and eccentric work in diagonal patterns. Plyometric, sport-specific exercises and proprioceptive neuromuscular facilitation and isokinetic exercises are initiated.

Maintenance Phase

The goal of this phase is to maintain a high level of training and prevent repeat injury. Emphasis is placed on intense workouts, proper arthrokinematics, and analysis and modification of techniques that may re-exacerbate symptoms. It is important for the patient to perform a home exercise program and clear understanding of the warning signs of impingement.

Generally, conservative treatment continues for 3–6 months. If the patient remains significantly disabled and has no improvement after 3 months of conservative treatment, the clinician must refer for surgical treatment (see Chap. 21).

Postoperative Care

After subacromial decompression, the patient is placed in a sling for a very brief period and is encouraged to remove it when comfortable and begin active and passive ROM exercises. When pain has decreased significantly and ROM has returned near to normal, a program of strengthening similar to conservative management is instituted.

Reports of 80–90 % success following subacromial decompression for impingement have been published. When acromioplasty was compared with conservative care, surgery appeared to be no more beneficial clinically at 6, 12, or 48 months [8].

Rotator Cuff Repair

Rotator cuff tears may result in significant shoulder dysfunction and functional impairments. Patients can have various clinical presentations due to different factors, including the characteristics of the rotator cuff defect.

The goal of rotator cuff repair is to restore the damaged tendon, eliminate pain, and improve function with increased

range of motion and shoulder strength. In addition to adequate surgical repair, outcomes are dependent on proper rehabilitation. Successful postoperative management following rotator cuff repair is dependent on several variables that have been shown to highly correlate with improved function, in particular integrity of the repaired rotator cuff and strength [9, 10].

The primary goal of the postoperative program is to protect the repair, promote healing, and gradually restore passive motion and muscular strength. It is imperative for the patient to be educated regarding protection of the repair site and the delayed nature of the healing process.

Thus, following rotator cuff repair, a postoperative abduction pillow brace supporting the shoulder at 30–45° of abduction may be necessary to decrease strain on the supraspinatus tendon repair site. It is important to underline that all rehabilitation professionals must be aware of the several factors that significantly affect the postoperative rehabilitation program. Two factors to consider are the surgical approach and the size of the tear. The rate of progression following rotator cuff repair is often determined by the amount of retraction present prior to repair, with the more retracted tendon requiring a slower rehabilitation course because of a higher postoperative failure rate. Tissue quality is also to be considered in determining the rate of postoperative progression, and another critical factor is the fixation method utilized.

Location and type of tear may require greater protection and slower progression depending on the tendon involved (infraspinatus and teres minor/subscapularis). The mechanism of failure of the rotator cuff and the timing of the repair have to be considered as well [11].

Another critical factor is the surrounding tissue quality. When it is fair to poor, the physical therapist should be cautious and more conservative postoperatively.

Nevertheless, the patient's characteristics, such as age, level of activity, lifestyle habits, and work situations, should be considered. The rehabilitation specialist should also consider the patient's goal for return to work and sport.

Postoperative Rehabilitation

The rehabilitation program following rotator cuff repair may be broken down into three main phases: (1) maximum protection, (2) moderate protection, and (3) functional phase. The goal of the first phase for rotator cuff repair is to prevent postoperative stiffness and scar tissue adherence while allowing for tendon to bone healing [12].

Maximum Protection Phase

Shoulder immobilization is recommended in this phase following rotator cuff repair regardless of the tear characteristics or method of surgical fixation. An abductor pillow brace with the shoulder supported between 30° and 45° of abduction in the scapular plane is recommended for about 3 weeks and then discontinued as determined by the surgeon or physical therapist. Active movements of the hand, wrist, and elbow are encouraged, and pendulum exercise, also known as Codman's pendulum exercise, is typically performed in this phase. Passive range of motion (PROM) and active assisted exercises are also prescribed during this phase. The use of a continuous passive motion (CPM) machine is not very frequent after this surgery and may complement the passive ROM by a therapist. Anyway, caution should be taken to avoid aggressive PROM into internal rotation with repairs of the infraspinatus and into external rotation with repairs of the subscapularis tendons.

Exercises for the scapular stabilizers may be initiated in this phase of rehabilitation as they have a synergistic relationship with the glenohumeral rotators. Early activation of the scapular stabilizers promotes improved scapulohumeral rhythm and functional use of the postsurgical shoulder [13].

Aquatic therapy is an appropriate option in the maximum protection phase, and it should begin as soon as the arthroscopic portals or surgical incisions are completely healed or otherwise covered with a waterproof bandage to allow for accelerated restoration of motion [14].

Closed chain activities are appropriate during this phase of rehabilitation as minimal activity of the rotator cuff and deltoid is exhibited.

Moderate Protection Phase

Progression to the moderate protection phase involves several factors, as described above. Generally, the patient is ready for active exercises between the sixth and seventh postoperative week. Common exercises in this phase of rehabilitation include progression of scapular stabilizers, isometric exercises for the rotator cuff musculature, and active range of motion of the glenohumeral joint. Physical therapist needs to be aware, at this point of the healing process, of the importance of exercises at force couples rather than isolated muscle. Mirror feedback may be helpful in retraining the patients' proprioception with these exercises.

Isometrics of the shoulder musculature are commonly performed during this phase of rehabilitation. Submaximal isometric contractions should be supervised in repairs of the infraspinatus and subscapularis in external rotation and internal rotation, respectively.

Neuromuscular electrical stimulation (NMES) may be used as an adjunct treatment to enhance force production and muscle recruitment and improve muscle function, but it is not well accepted by patients. Physical modalities (laser therapy, diathermy, etc.) may be prescribed to deal with

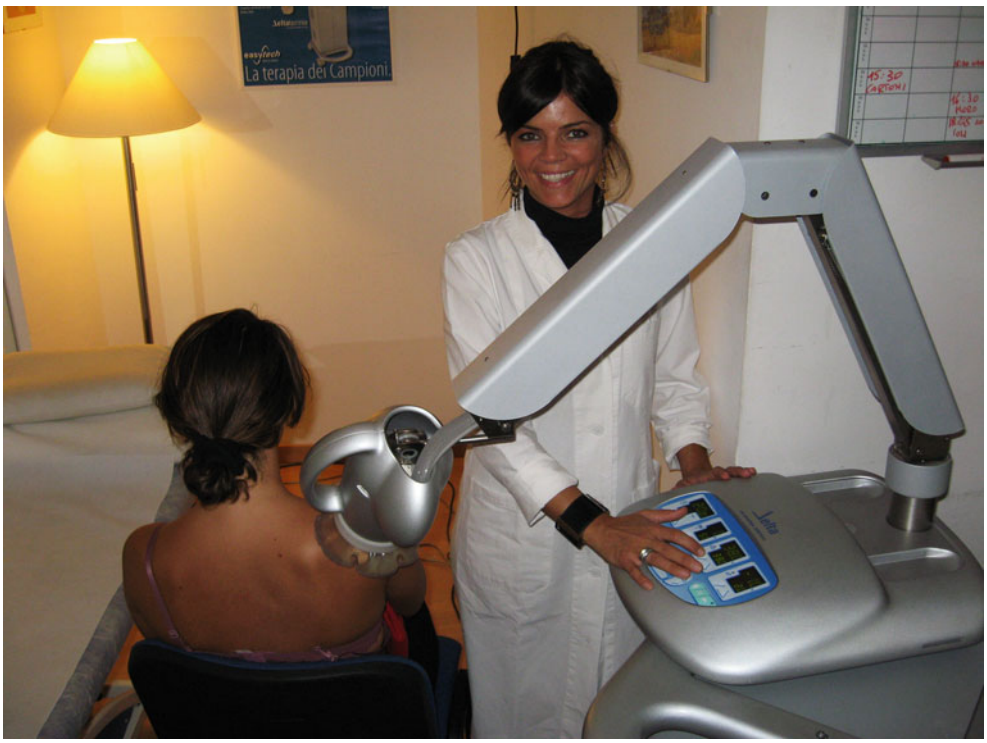

Fig. 6.2 Microwave diathermy for the treatment of pain and muscle spasm

pain or muscle spasm (Fig. 6.2). Active range of motion (AROM) exercises of the glenohumeral joint are typically performed during this phase of rehabilitation. It is suggested that upright elevation begins with elbow bent and then progressed to elbow straight as increased activity of the rotator cuff muscles is seen. Aquatic therapy can also be proposed, and exercises should include resisted forward flexion with paddles, ball proprioception exercises and resistance, and wall push-ups [15]. Closed chain activities are appropriate in this phase of rehabilitation (pointer and/or tripod positions).

Minimum Protection Phase

Transition to the minimal protection phase typically occurs 12–14 weeks postoperatively. In this period, strengthening of the rotator cuff begins, and progression to functional lifting and sports activities is allowed.

Strengthening exercises may utilize elastic resistive bands (tubing) or dumbbells with the glenohumeral joint in various positions to strengthen the rotator cuff muscles. Plyometric and isokinetic exercises may be useful adjuncts for athletic population, and proprioception exercises should be increased as well.

It is important for rehabilitation professionals to recognize evidence-based tactics to restore the impairments after rotator cuff repair and utilize them properly with consideration of the numerous variables that can impact patient recovery.

Disorders of the Long Head of the Biceps

The long head of the biceps (LHB) tendon has been recognized as a potential source of clinically significant pathology. When it is determined to be a significant contributor to patients' symptoms, the treatment options include different conservative interventions and various surgical procedures, such as tenotomy, transfer, or tenodesis. The ultimate management decision is based upon a variety of factors including the patient's overall medical condition, severity and duration of symptoms, expectations, associated shoulder pathology, and surgeon's preference.

The most important factors in selecting a surgical treatment are the primary cause of the condition, the integrity of the tendon, the extent of tendon involvement, and any related pathology that also needs to be addressed [16].

Due to the variety of surgical techniques proposed, it is imperative that the rehabilitation professionals communicate frequently with the physician to ascertain the type of surgery performed and fixation, the patient's tissue and repair quality, concomitant procedures performed, and any special instructions specific to the patients' rehabilitation. Successful biceps rehabilitation requires the therapist to create a good healing environment based on soft tissue healing properties. This concept involves controlling pain, swelling, irritation, and the load placed on the healing tissue.

Although little research specifically relating to the rehabilitation of LHB is present, therapists are aware that there

are differences in the management, for instance, of biceps tenotomy compared to tenodesis. Tenotomy rehabilitation will be more aggressive and advance more quickly. Conversely, rehabilitation following tenodesis will progress more slowly over the first 6 weeks to protect the healing biceps tendon.

Postoperative Rehabilitation

The rehabilitation program following surgical management of LHB pathology may be divided into four phases.

Immediate Postoperative Phase

Rehabilitation begins the day after surgery. A standard sling is used as needed. An elastic wrap is placed over the upper arm to provide support to the healing biceps. The goals are to decrease pain and swelling, initiate gentle rhythmic stabilization exercises and scapular control, and restore full PROM. Full passive motion is expected 1–2 weeks postoperatively with patients post-tenotomy typically achieving full motion slightly ahead of those post-tenodesis. Manual therapy treatments and modalities are prescribed as needed to decrease pain and improve ROM. Particular attention is placed on rhythmic stabilization and scapular exercises during this phase to improve neuromuscular control. As patient progresses, manual interventions subside in favor of active work.

Moderate Protection Phase

In this phase patients are typically out of the sling and experiencing minimal or no pain or swelling. The goals in this phase are to increase AROM, activity tolerance, and muscle strength and endurance. A key rehabilitation regimen proposed in this phase is the "lawn chair progression," which involves transitioning from supine AROM to more functional active exercises sitting upright. This phase lasts approximately 2 weeks for tenotomy compared to 6 weeks for tenodesis.

Functional Phase

The goals in this phase are increased endurance and strength. Biceps strengthening should include both supination and elbow flexion work. Exercise selection is based on patient goal and activity demands. Proprioception and neuromuscular reeducation exercise are crucial to counteract the inhibitory effects that pain and inflammation have on the rotator cuff and scapular stabilizers [17]. Bodyblade rhythmic stabilization exercises and multiplanar and multijoint patterns are important for a complete neuromuscular reeducation. Strengthening exercises focus on incorporation of the entire kinetic chain. Rotator cuff exercises begin with Thera-Band or tubing in external and internal rotation performed with the arm supported at 30° of abduction. Patients with tenotomy usually progress to the next phase from 4 to 6 weeks postoperatively, whereas those post-tenodesis wait until weeks 8–12.

Return to Sport

The goals for this phase are to increase muscle strength and power, complete an interval throwing program, and return to previous level of sport participation. Plyometric exercises are appropriate to enhance dynamic stability and proprioception. A safe and effective progression for plyometrics could begin with a chest pass exercise and progress to a proprioceptive neuromuscular facilitation (PNF) D2 pattern exercise. Athletes are able to return to sport when painless full motion and full strength are regained.

Disorders of the Acromioclavicular Joint

The acromioclavicular joint (ACJ) is a frequent source of shoulder pain. Its subcutaneous location makes it highly susceptible to trauma. Injuries such as AC separations are common in people who participate in contact sports. This joint is also predisposed to degenerative changes because of aging and the reliance on the arm for function. Another condition called "atraumatic osteolysis of the distal clavicle" has been recognized increasingly and coincides with the popularity of strength training. While dealing with any AC joint disorder, physical therapists must not forget that changes in structure and function of this joint because of injury or degeneration can result in a compromise to the "suprahumeral" space and that a high incidence of coexisting pathological conditions with symptomatic ACJ problems, such as biceps tendon pathology, full- or partial-thickness rotator cuff tears, and tears of the glenoid labrum, has been demonstrated.

Acromioclavicular Separation

Sprains and dislocations of ACJ are seen commonly with contact and high velocity sports, but other common causes of AC injury include motor vehicle accidents and falls. AC injuries occur five to ten times more frequently in males compared with females and are seen most commonly in people in their teens through their 30s. Incomplete injuries are more common than complete dislocations. The mechanism for AC joint injury is generally represented by a fall on the point of the shoulder with the arm in an adducted position.

Classification

The most commonly used grading system for AC injuries is the Rockwood classification [18]. This system consists of six different types of AC injuries. Types I and II are considered

incomplete injuries. Types III to VI are complete dislocations of the AC joint. By distinguishing the different types of complete dislocations, this system helps to determine the need for surgical intervention.

Nonoperative Treatment

Type I AC injuries are treated with ice, nonsteroidal anti-inflammatory drugs (NSAIDs) or analgesic medications, and immobilization with a sling as needed for pain relief. ROM is advanced as tolerated. Pendulum exercises and gentle strengthening exercises may begin when pain-free and when ROM is near to normal. Return to sports can generally occur by 2 weeks. Cross chest adduction, wide-grip bench press, and dips should be avoided for 5–6 weeks.

Type II AC injuries are treated conservatively as well. Most patients have a full recovery. A sling is typically worn for 10–14 days or until symptoms are improved. Gentle early ROM can begin as tolerated. The arm can be used for activities of daily living when tolerated, which is typically by day 7. Gentle strengthening exercises may begin as described for type I injuries. Taping techniques may be proposed. No heavy lifting or contact sports should be undertaken for 5–7 weeks.

The treatment of type III AC injuries is controversial. Nonoperative and operative treatments have been used in the past with successful outcomes. More recently, conservative treatment has gained favor. Harnesses and braces such as the Kenny-Howard or "figure-8" brace are sometimes used to depress clavicle and reduce the dislocation. A sling should be worn for comfort for 4 weeks.

Gentle ROM activities can begin as the pain subsides and can advance as tolerated. Light strengthening exercises can start when ROM is near to normal and when the patient is not experiencing significant pain. Because the articular disc is so poorly developed and the surfaces of this joint have such poor congruency, it is difficult for the ACJ to dissipate forces in the manner seen in other joints.

Rehabilitation professionals must remember as well that a complete neurovascular examination is imperative because the brachial plexus and subclavian vessels traverse the area between the clavicle and the first rib. Grade III sprains of the ACJ can result in traction to the suprascapular nerve as a result of downward displacement of the scapula and the resultant compression and traction to the nerve by the overlying transverse scapular ligaments. Patients being treated conservatively should be made aware of the resultant cosmetic deformity that will be present. If the patient has persistent pain or functional deficits after conservative treatment, surgical options may be warranted and should be considered for those patients who participate in throwing or overhead sports, heavy laborers, or patients who are unwilling to accept cosmetic deformity.

Types IV to VI dislocations are generally treated surgically. Many surgical procedures have been proposed (Weaver-Dunn procedure, dynamic muscle transfer, etc.) (see Chap. 45). After these procedures, patients are kept in a sling for approximately 8 weeks. Codman's pendulum and gentle ROM exercises may begin in the early phase of rehabilitation. Modalities may be used to deal with pain and inflammation. After the sling is discontinued, the arm can be used actively without weights. Progressive resistance exercises can start at 11–12 weeks. Return to heavy labor and sport generally requires 4–6 months.

Acromioclavicular Degenerative Arthritis

ACJ osteoarthritis is the most common cause of AC pain. This condition may be seen as a result of age or as a consequence of chronic overhead use of the extremity. Symptoms of degenerative changes of the ACJ include anterior and superior shoulder pain. Tenderness over the ACJ is also present. Treatment of ACJ osteoarthritis includes modifying activities, physical modalities, NSAIDs, and joint injection with steroids. If conservative treatment fails, surgery can be considered. The Mumford procedure, in which the distal clavicle is resected, has shown good result.

Atraumatic Osteolysis of the Distal Clavicle

Osteolysis of the lateral end of the clavicle has been known to occur after trauma to the shoulder, but with the increasing popularity of weightlifting, this condition has become a more common occurrence. Although its etiology is not completely understood, osteolysis is seen more often in male athletes who have a long history of strength training. Treatment consists of activity modification. Eliminating the exacerbating activities and lifting exercises may prevent progression and alleviate symptoms. Failure of conservative treatment is an indication for surgery, consisting of resection of the distal clavicle.

Glenohumeral Instability

Glenohumeral joint instability is a common disorder of the shoulder. There is a spectrum of presentation with shoulder instability. Traumatic anterior dislocation represents one end of this spectrum, while multidirectional instability would represent the other end. Variations in definition, such as voluntary or involuntary instability or traumatic versus atraumatic, make the diagnosis of this entity even more difficult. The treatment options for glenohumeral instability and dislocation include nonoperative and operative approaches. Patients with multidirectional instability generally refer satisfactory outcomes after a comprehensive rehabilitation program that addresses kinetic chain deficits, scapulothoracic

mechanics, shoulder girdle strength, flexibility, and neuromuscular control.

For patients who have a first time anterior dislocation, the decision between nonoperative approach and immediate surgical stabilization is more controversial.

Nonoperative Treatment

Conservative treatment should include pain and swelling control, restoring shoulder girdle ROM, protection of the static glenohumeral joint stabilizers, obtaining full function of the dynamic stabilizers, restoring joint proprioception, and correcting associated kinetic chain deficits. The goal of this program should be the unrestricted return to preinjury activities.

Traditional nonoperative treatment after acute dislocation includes a period of immobilization with the arm in internal or external rotation [19]. Initially, treatment emphasizes controlling pain and inflammation, protecting healing tissues, and decreasing the deleterious effects of immobilization. Modalities may be useful also for promotion of tissue healing. Taping the unstable shoulder can help to improve joint biomechanics and enhance neuromuscular reeducation of the shoulder complex musculature.

The principles of glenohumeral joint protection include avoiding impingement positions, decreasing capsular stress, and preventing tendon overload. Exercises in the plane of the scapula are recommended. If posterior glenohumeral joint capsular tightness is present, mobilization using posterior glide techniques while horizontally adducting the internally rotated arm may be useful. Reestablishing appropriate force couples about the glenohumeral and scapulothoracic joints is crucial during rehabilitation.

When strengthening the rotator cuff for the treatment of specific instability pattern, it is important to remember Dempster's ring concept [20]. It has been found that any stress on the stabilizers of one side of the glenohumeral joint also places stress on the stabilizers of the opposite side of the joint. So it is important to strengthen anterior and posterior cuff and scapular muscles for the treatment of anterior and posterior unidirectional instability (Fig. 6.3). Multidirectional instability requires strengthening of all the rotator cuff muscles, long head of biceps, and deltoid. A comprehensive rehabilitation program needs to address the strength, endurance, and neuromuscular control of the rotator cuff and scapular stabilizers. Initial exercises should include multiangle, submaximal isometric contractions to activate neuromuscular control, develop strength, and improve local blood flow. Then rehabilitative exercises should progress to multiplanar activities in the full ROM, incorporating isotonic and isokinetic resistance at submaximal and maximal levels. The use of closed kinetic chain exercises is important for strengthening the unstable shoulder. Exercises with combined movement patterns (PNF) are important to reestablish function. Plyometric exercises are added in the last phases of rehabilitation.

Patients with shoulder instability frequently experience proprioceptive deficits, so rehabilitative exercise to enhance joint position sense and kinesthesia should include rhythmic

Fig. 6.3 Exercise for rotator cuff with software providing feedback for strength and kinesthesia

stabilization and ball tossing activities in varying degrees of shoulder position and PNF drills using Bodyblade, tubing, and manual resistance.

Postoperative Care

The biological healing response of the repaired and imbricated tissue must be respected. Although the specific postsurgical rehabilitation protocols vary according to the surgeon and type of surgery performed, the goals of rehabilitation are to regain full static and dynamic function of the shoulder and return to sports or activities of daily living in a reasonable amount of time. Many of the exercises used postoperatively are similar to those used for the standard conservative management of shoulder instabilities. However, postoperative ROM considerations are crucial in these patients, and specific rehabilitation protocols must be tailored based on surgical procedures (Bankart repair, Latarjet procedure, capsular shift, etc.) and quality of the tissues found at the time of surgery.

Initially, a period of immobilization in a sling is advocated. This must be worn from 2 to 4 weeks, including during sleep, in respect to the surgical procedure. ROM of the elbow, wrist, and hand is encouraged in the immediate postoperative period, and cryotherapy is recommended for 15 min three or four times a day. Gentle, small arc Codman's pendulum exercises are started in the early phase. Passive and active assisted ROM exercises start in the maximum protection phase, and it is imperative not to start strengthening or repetitive exercises until full ROM has been established. Early proprioception exercises are recommended. Strong resistance exercises with aggressive early postoperative rehabilitation do not appear to offer substantial advantages and could compromise the repair. In the minimum protection phase, exercises progress from isometrics for scapulothoracic and glenohumeral musculature, performed at submaximal intensity with no to minimal pain, to active concentric exercise, using Thera-Band or self-assisted closed kinetic chain patterns [21].

The goal is to achieve normal scapulothoracic and glenohumeral mechanics and good muscle endurance. Eccentric exercises should be performed in the functional phase, with continued progression of therapeutic exercise. Propedeutic to sport is gradually initiated, with plyometrics, isokinetics, and increasing difficult tasks aimed at restoring good balance and proprioception.

Frozen Shoulder

Frozen shoulder, or adhesive capsulitis, describes a common shoulder condition characterized by painful and limited active and passive ROM. A common quandary with this set of complaints exists in determining the cause-and-effect cycle of the symptoms. Terminology and classification, as well as etiology, pathophysiology, epidemiology, natural history, and diagnostic evaluation, are explained elsewhere in this book (Chap. 28).

Treatment Guidelines

Indications, technique, and effectiveness of corticosteroid injections will not be discussed in this chapter as far from the tasks of rehabilitation professionals. Identifying the stage of frozen shoulder in which a patient is presenting is important to determine the appropriate treatment regimen. Even though multiple interventions have been studied [22], the definitive treatment for frozen shoulder remains unclear. The overall goal of treatment is well accepted: relieve pain and restore motion and function.

Establishing treatment effectiveness is also difficult because the majority of patients significantly improve in approximately 1 year. Additionally, frequency and timing of visit and discharge criteria have not been established. Patient education about the natural history is probably an important treatment aspect.

Exercise, Modalities, and Manual Therapy

Exercise is the key to any treatment protocol for frozen shoulder. A typical exercise program is one of active and passive stretching with the goal of maintaining and regaining ROM. The basis of this program is the "four-quadrant stretching": forward flexion, internal rotation, external rotation, and cross body adduction and Codman's pendulum exercises. Stretching a frozen shoulder can be painful. Modalities are suggested to influence pain and muscle relaxation. Application of heat (microwave diathermy or moist heat) in conjunction of stretching has been shown to improve muscle extensibility [23]. This may occur by a reduction of muscle viscosity and neuromuscular-mediated relaxation. Transcutaneous electrical nerve stimulation (TENS), together with a prolonged low-load stretch, resulted in less pain and improved motion in patients with frozen shoulder. The basic strategy in treating structural stiffness is to apply appropriate tissue stress. The primary factors that guide this process are pain and ROM. Applying the correct tensile stress dose is based upon the patient's irritability (high – moderate – low). A pulley or a cane/stick may be used, depending on the patient's ability to tolerate the exercise. Many authors and clinicians advocate joint mobilization and aquatic therapy for pain reduction and improved ROM [24]. Finally, when functional ROM has been obtained and pain has improved, gentle strengthening and proprioception exercises can begin. There is no clear evidence to determine which patients may need formal supervised therapy rather

than a home program. Factors that may favor use of the former may be greater disability, more comorbidities, lower social support, lower education level, or high fear or anxiety. If the symptoms and motion are unresponsive to the different treatments over time (3–6 months) and quality of life is compromised, a manipulation under anesthesia or surgical capsular release should be considered. Postoperative protocols may vary from using a continuous passive motion device and exercise to a daily comprehensive physical therapy program as described before [25].

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Part II

Principles of Shoulder Arthoscopy

Instrumentation in Shoulder Arthroscopy

7

Maristella F. Saccomanno, Silvia Careri, and Giuseppe Milano

Arthroscopy is a reliable and effective minimally invasive technique that has gained popularity in the past 20 years among shoulder surgeons. Arthroscopic surgery requires specific and complex instruments whose evolution has followed that of the surgical procedures. These instruments are expensive, and therefore, proper utilization and maintenance are essential. Furthermore, different surgical procedures, such as rotator cuff repair and capsulorrhaphy, call for different instruments which are specific to the operation being performed. Many surgical instrument companies currently produce tools which are very similar to one another with only minor technical differences; the selection of the most appropriate instrumentation depends on the individual discretion of the surgeon. In fact, each arthroscopic surgeon generally has his own set of instruments.

Systematicity is fundamental to successfully manage the instrumentation. The entire operating room team must be thoroughly trained on the equipment utilized for every type of surgery and informed of the technical preferences of the surgeon. The surgical instruments must always be positioned on the operating table in the same order; proper placement of the surgical equipment is critical and must be established before the operation.

Arthroscopy Tower

The arthroscopy tower consists of a vertical cart with various shelves on which the electronic equipment used for the arthroscopic procedure is placed.

Modern arthroscopy towers have a modular design to conform to any setup need. The power cords of the various units are pre-wired, and cable management is accomplished on either side of the cart. The carts are configured with wheels which allow them to be moved to the optimal position during surgery. The standard equipment to be placed on the tower includes a high-definition (HD) flat-screen monitor with dimensions varying from 25″ to 32″ suspended by a large moveable arm which enables the screen to be appropriately positioned according to the surgeon's needs. The video camera unit is placed on the first shelf of the cart followed by the light source. The cameras are generally equipped with 3 CCD (Interline Transfer Micro-Lens High Sensitivity CCD Image Sensor, 768 × 494 pixels each); these sensors allow for a resolution of 800 horizontal lines and 450 vertical lines to be displayed on the monitor. Adjustment for the brightness is automatic, thanks to an auto shutter with a speed of 1/10,000 s, controlled by the unit or by a control button located on the top of the camera head. With these buttons, it is possible to program the main functions: brightness, white balance, and peripheral illumination correction. The light sources consist of xenon 100–300 W lamps with a color temperature of 5,700–6,000°k and utilization temperature of 5–38°. A fiber-optic cable, approximately 2.5 m long with a diameter of 4 mm, is connected to the unit. It is currently possible to have a single, integrated control unit that combines the HD video camera (1080 p), "xenon bright" LED light source, and image management console with a tablet that is not only able to record videos and/or photos of surgical procedures and memorize the surgeon's preferences regarding setup but that also enables any authorized workstation to follow the surgery in streaming. Next on the arthroscopy cart, there is an irrigation pump, a unit for the motorized instruments that allows two motorized tools to be used at the

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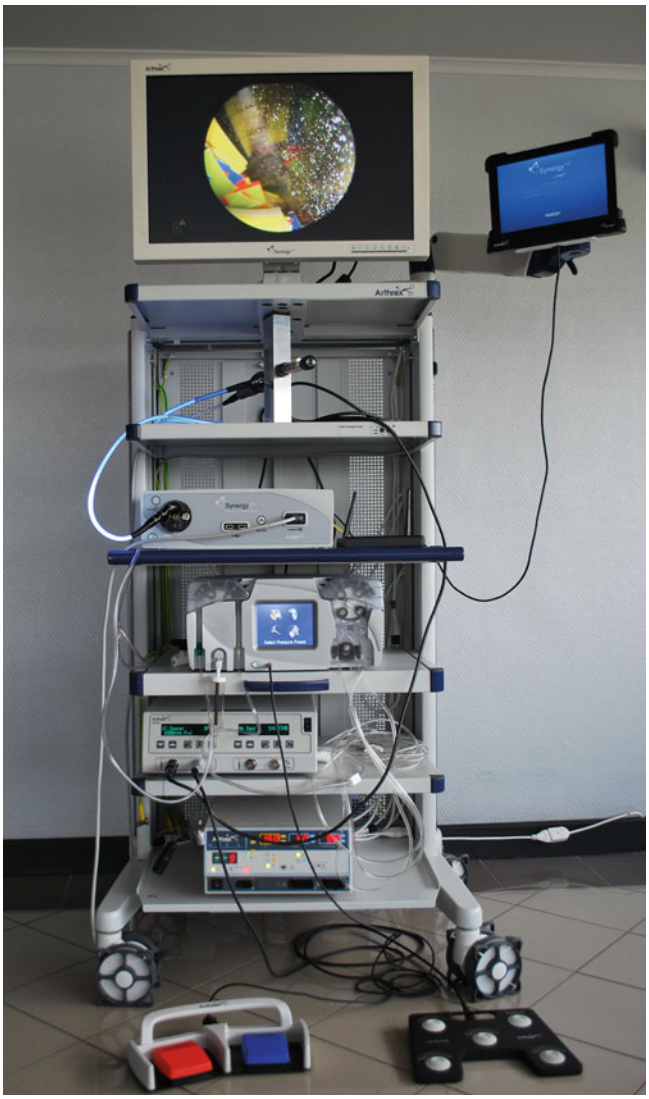

Fig. 7.1 Arthroscopy cart: a high-definition (HD) flat-screen monitor and a tablet suspended by a moveable arm are placed on the top; an integrated control unit that combines the HD video camera, the "xenon bright" LED light source, and the image management console is placed on the second shelf, followed by the irrigation pump, the motorized instruments unit, a radiofrequency generator, and the footswitches

same time, a radiofrequency generator, and a space for the footswitches (Fig. 7.1).

The arthroscopy cart is placed on the operative side of the operating table and in front of the surgeon (see Chap. 8).

Arthroscope

The arthroscope is composed of a rigid external sheath, at the distal end of which there is an optic device (objective end) that is able to reproduce the image of an object placed in front of the instrument. The area being examined is illuminated using a fiber-optic bundle which is placed on the inside of the metal sheath parallel to the axis of the optical system, along with the utilization of a light source. The image is transmitted to the ocular end, located at the proximal end, through an efficient system of lenses strategically positioned on the inside of the sheath.

The ocular end is equipped with wiring which enables it to be attached, using a special adapter, to the video camera; it is also connected to the light source.

The arthroscope is inserted into a metallic sheath, which is characterized by two lateral extensions (one for the inflow and one for the outflow of fluids) and a diameter that is large enough to assure an adequate flow (generally 4.5 mm).

There are different size arthroscopes currently available on the market with diameters ranging from 1.9 to 4.0 mm; those utilized in shoulder surgery generally have a diameter of 4.0 mm.

The arthroscope is characterized by field of view, inclination of view, and movements. There are two fields of view: one apparent and one real.

The apparent field of view is determined by the diameter of the circular image as seen through the ocular end of the arthroscope and displayed on the monitor. This field is influenced by the distance between the object and the arthroscope. The larger the circle, the larger the image will appear. The real field of view is the angle of view produced by the arthroscope and generally varies from 80° to 115°.

The inclination of view is the angle of projection at the objective end of arthroscope. The inclination is calculated by drawing a line along the axis of the arthroscope that intercepts the line drawn from the center of the arthroscopic image on the lens. There are various angles: 30°, 70°, and 90°. These angles allow for a complete inspection of the joint in all its corners because the structures being evaluated are often found on the side of, above, and below the position of the arthroscope. The standard 30° view is the easiest and most widely utilized because rotating the optical system at this angle provides for the best surgical viewing.

Moreover, the arthroscope is designed to perform three movements: pistoning, angulation, and rotation. The forward and backward movement of the arthroscope is called pistoning. The diagnostic arthroscopy begins with a broad overview, and then the arthroscope is moved closer to the specific structures for a better, more in-depth visualization. Angulation is a sweeping motion that allows the inspection of all the structures. Rotation is the most valuable movement in arthroscopy. Once the arthroscope is positioned at an appropriate distance to achieve a broader viewing, rotation of the scope allows the surgeon to inspect the joint without pistoning or angulation (Video 7.1).

The ability to effectively utilize the arthroscope is one of the elements which distinguish a good surgeon from an excellent surgeon because understanding the lesions, choosing the type of repair, and the repair itself depend on good visualization.

Illumination of the operating field is affected by two main factors: the amount of light and the quality of the lens system which transmit the light. Conceptually, the larger the size of the arthroscope, the more space there is for fiber optics which conduct the light beam. The most commonly used lens system is a system of cylindrical lenses (Rod lens system) developed expressly to balance the relationship between the fiber optics and the lenses to ensure a bright and clear viewing of the area being operated.

During the preparatory phase, it is essential to meticulously check all instrumentation relating to the optical system. Potential technical problems that could negatively impact the visualization and that should be investigated prior to surgery include the following:

- Arthroscope: any damage on the surface of the ocular end or the objective end caused by improper use or maintenance of the arthroscopic instruments (i.e., cracks, scratches, burns).
- Light source: any problems stemming from the source or the cable which connects to the arthroscope; both ends of the cable should be inspected for dirt or breakage. Care must be taken not to twist or bend the cable to avoid breakage of the fibers.
- Camera: check the focus and white balance.

Fluid Management

In all surgical procedures, good visualization of the operating field is critical. To obtain good visualization in shoulder arthroscopy, the following conditions must exist: proper functioning of the optical systems, adequate joint distention that allows for thorough inspection of the anatomical structures during the diagnostic phase, and correct utilization of the arthroscopic instruments during surgery. Therefore, the main objectives of fluid management are joint distention and limiting bleeding to ensure a clean operating field and good visibility and increasing the effectiveness of the cutting tools. To achieve these objectives, it is necessary to have constant positive intra-articular pressure in addition to maintaining the correct balance of fluids.

Fluid dynamics are based on four parameters: flow, flow rate, pressure, and resistance. Flow measures the volume of fluids which moves past a cross section of a tube in a given unit of time and is expressed in liters per minute (L/min) or milliliters per minute (mL/min). Flow rate measures the distance that a certain volume of fluids travels in a given unit of time. Pressure (mmHg) refers to the amount of force applied to a specific area or more precisely to the relationship between a mass and the volume in which it is contained. If the volume containing a certain amount of fluids increases because the walls expand, the pressure will decrease. On the other hand, if the walls of the chamber containing the liquid are not able to expand and the amount of fluids is increased, the pressure will increase. Resistance refers to a tubular system's tendency to obstruct the flow of the fluids and is influenced by the diameter of the tube.

Movement of fluids occurs along a pressure gradient; the fluids move from areas characterized by greater pressure to areas of lower pressure. In fact, the flow in a tube is directly proportional to the pressure gradient. This relationship affirms that the greater the pressure gradient, the greater the flow. Furthermore, liquids flow in the direction of least resistance. The flow in a tube is, hence, inversely proportional to the resistance. This inverse relationship confirms that as the resistance increases, the flow will decrease and vice versa. Therefore, flow is determined using the following formula: flow = pressure/resistance. For a liquid which flows in a tube, the resistance is usually impacted by three parameters: the radius of the tube (r), its length (L), and the density of the liquid (η) (eta). The following equation, known as Poiseuille's law, demonstrates the relationship between these factors: $R = 8L\eta/\pi r^4$. If we consider the density of the liquid to be constant, this equation shows that (1) resistance to the flow increases when tube length is increased and (2) resistance to the flow decreases when tube radius is increased.

A complete irrigation system is composed of a pump with a varying number of restrictions which are connected in series (diameter of the inflow tube, arthroscope with sheath, joint, outflow cannula, or suction hose). The pump generates an initial pressure, and flow will vary according to the total number of restrictions in the system. Local pressure will be reduced in every location where there is a restriction, and if there are many restrictions before reaching the joint, the drop in pressure will be substantial, resulting in an intra-articular pressure which is lower than that created by the system. From a practical point of view, the main factor which determines inflow is the resistance encountered at the point of entry (diameter of the entry cannula and/or the sheath of the arthroscope). The inflow tube is generally connected to the sheath of the arthroscope to have a direct flow towards the field of view and to be able to manage modifications in pressure in the event of bleeding. Outflow is typically managed through the cannula in the anterior-superior portal. When inflow is equal to outflow, intra-articular pressure is stable and balanced.

Intra-articular pressure, or subacromial space pressure, is determined by the initial pressure of the system, changes in joint position (abduction and traction reduce the pressure; rotations increase it), and inflow/outflow points controlled by the surgeon.

In general, fluid pressure within the glenohumeral joint is kept close to 30–40 mmHg; it can increase to between 40 and 70 mmHg in the subacromial space to allow for an adequate visualization. Maintaining the mean arterial pressure between 70 and 90 mmHg, or the systolic blood pressure at 100 mmHg, improves the visualization. In a study regarding

the relationship between systolic blood pressure and irrigation pressure in the subacromial space, Morrison et al. [1] demonstrated that a difference of more than 49 mmHg between systolic blood pressure and subacromial space pressure, due to an increase in the patient's arterial pressure or a decrease in irrigation pressure, causes bleeding. An inadequate intra-articular irrigation pressure compromises operating visibility because it produces a collapse of the joint and turbulence secondary to bleeding, increasing the risk of inadvertently damaging articular structures. Nevertheless, excessive irrigation pressure can cause an extravasation in the soft tissue, rupture of the synovial membrane, and even compartment syndrome [2–5]. A constant flow of 5–10 mL/min is sufficient for a proper viewing.

Fluids

The liquids utilized must have osmotic, ionic, and pH biologically compatible properties to not cause tissue damage. Furthermore, they must not conduct electricity to ensure safe utilization of the radiofrequency tools. We use 3 L bags of sterile saline solution with one vial of noradrenaline added to help control any bleeding. These bags are hung at a fixed height from a pole adjacent to the arthroscopic tower and connected to the irrigation pump through a Y-connector.

Irrigation Systems

It is possible to use two different irrigation systems: a gravity system and an automatic pump system.

Gravity System

The gravity system depends on hydrostatic pressure; the pressure gradient and the flow generated by this system are exclusively due to the difference in height between the irrigation solution bags and the joint (30 cm = 22 mmHg) and the diameter of the sheath of the arthroscope (Poiseuille's law). Keeping the bags at a fixed height, the pressure gradient is not influenced by the volume of the bags. Therefore, the flow is modified by altering the height at which the bags are hung and not by altering their volume (Bernoulli's principle).

Intraoperatory vision can be influenced by fluctuations in inflow, for example, when a bag is emptied, and hence, saline solution bags are generally connected to the system in the following ways:

- One open and one closed
- Both open: one higher that will be emptied first and one lower that will subsequently begin to be emptied, generating less of a pressure gradient but ensuring the ability to change the first bag without interrupting the flow [6–8]

The outflow represents another fundamental point to be considered. When motorized instruments are utilized, outflow increases, and the inflow is not able to sustain an adequate intra-articular pressure, resulting in a negative fluid balance, and consequently, the joint will tend to collapse. To avoid this problem, it is recommended that suction be regulated through the manipulation of the motorized instrument or by manually closing the suction hose at intermittent intervals.

The advantages of a gravity system lie in its simplicity, safety, and low cost.

Automatic Pump Systems

In these systems, the pressure gradient is completely controlled by the pump and, therefore, does not depend on the height of the bags, volume, or gravity. These pumps create a constant and predictable flow and are able to produce greater flows and higher pressures than those produced by a gravity system. The higher pressures enable any bleeding to be stopped by plugging the vascular wall, and greater flows can be generated when using motorized tools.

There are two types of pumps: the peristaltic pump and the centrifugal pump.

The peristaltic pump works by pulsing, closing, and opening the inflow tube, releasing a certain amount of fluids. Pressure and flow are regulated by adjusting the revolutions per minute (RPM) on the pump control unit. The disadvantage of this type of pump is that the flow is pulsed; since the pressure is determined by the flow rate, high flow rates can produce pressure surges.

The centrifugal pump utilizes a rotating pump which continually releases a volume of liquid. In this way, there is a uniform control of the pressure, and surges are avoided. The disadvantage is that a continuous flow in an uncontained space (i.e., subacromial area) can cause an excess fluid extravasation if the outflow is not well balanced.

Pumps with independent control of the flow and pressure are available on the market. We use a pump that has an integrated inflow/outflow fluid management system but that can also be used exclusively as an inflow pump. Using piezoelectric sensors, constant control of the pressure is maintained without producing any pulsing effect while, at the same time, adjusting for changes in pressure and intra-articular flow. It is, therefore, able to achieve an adequate intra-articular distention even when the outflow increases as a result of the use of motorized tools. The setting of the pressure and flow values is adjustable using the control unit, either with a touch screen or a remote control (Fig. 7.2).

Hand Instruments

Hand arthroscopic instruments must be the appropriate size for the joint and have magnetic properties which allow them to be recovered in the event of breakage. The tools used for

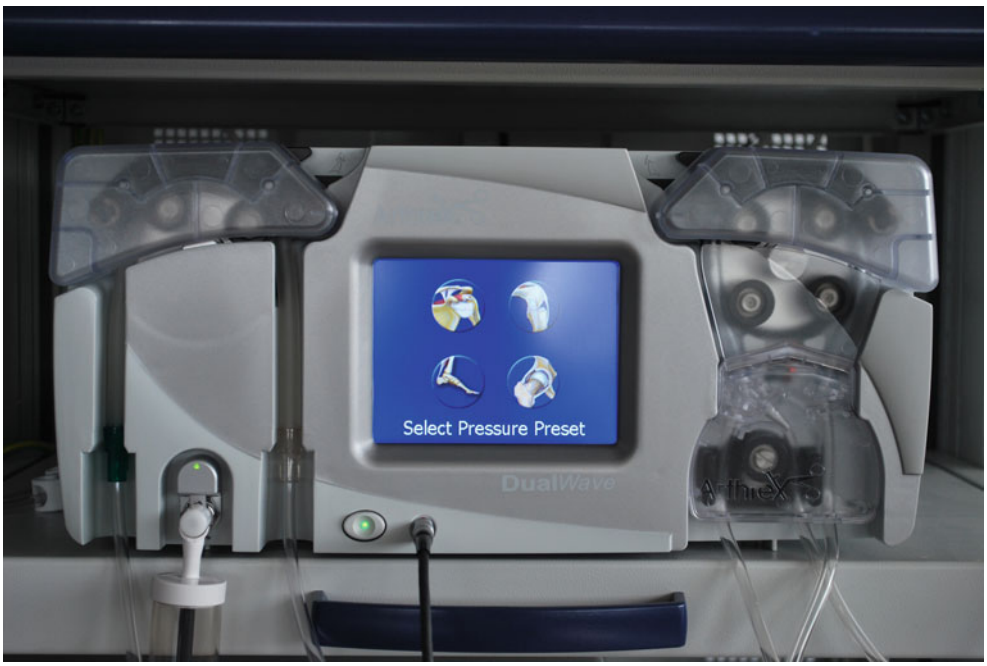

Fig. 7.2 Irrigation pump. Touch screen allows to select preset pressure and flow values according to the procedure (shoulder, knee, hip, or ankle arthroscopy) or to adjust values according surgeon's need

evaluation purposes, or for creating arthroscopic access, usually have blunt ends which reduce the risk of lesions on the joint surfaces and/or peripheral vessel and nerve structures. On the other hand, the instruments used for surgical procedures generally have sharp ends to be able to cut effectively.

Permanent Skin Marker

A dermographic pen is utilized to draw landmarks of the underlying bone structures on the patient's skin. These landmarks help the surgeon to identify arthroscopic access points as well as neurovascular structures which could be at risk during the operation (see Chap. 10).

Needles

An 18 gauge spinal needle is used to correctly identify the arthroscopic entry points on the skin and trajectories leading into the joint (see Chap. 10).

Cannulas

Arthroscopic cannulas can be made of plastic or metal; they are all equipped with a blunt trocar that facilitates the penetration through the soft tissue to reach the joint.

We prefer to use a metal cannula to create the arthroscopic portals. We use plastic cannulas with different calibers throughout the remainder of the surgery: 8.0 mm operative cannulas and 5.5 mm outflow cannulas.

Plastic cannulas have the following characteristics (Fig. 7.3):

- They are available in different colors that indicate the different calibers.
- They can be smooth or threaded; the threaded design helps prevent the cannulas from accidentally slipping out during the operation.
- They can be rigid, semirigid, or flexible,
- They are translucent to facilitate viewing and management of the sutures and knots.
- They are equipped with a lateral spigot which allows outflow to be controlled manually and an anti-reflux valve, in either plastic or silicone, which helps to maintain intra-articular pressure, limiting spontaneous outflow of fluids.

In addition to managing outflow and facilitating the passage of sutures and knots, cannulas are used to ensure the passage of arthroscopic instruments in the joint without damaging soft tissue, avoiding the creation of false routes.

Switching Sticks

There are generally two switching sticks per arthroscopic kit. They are metal rods without a head and with blunt ends that serve as a guide in the creation of portals and to facilitate the exchange between portals and cannulas. If it is necessary to invert the position of the arthroscope and the cannula, the two switching sticks are inserted, one in the cannula and the other in the sheath of the arthroscope; in this way, it will be possible to switch the portals, leaving the sticks inserted so the portals are not lost (Fig. 7.4).

Fig. 7.3 Different types of cannulas are available. They can be smooth or threaded, rigid, semirigid, or flexible. Different colors indicate different calibers. They are translucent to easily manage the sutures. Lateral spigot allows outflow control

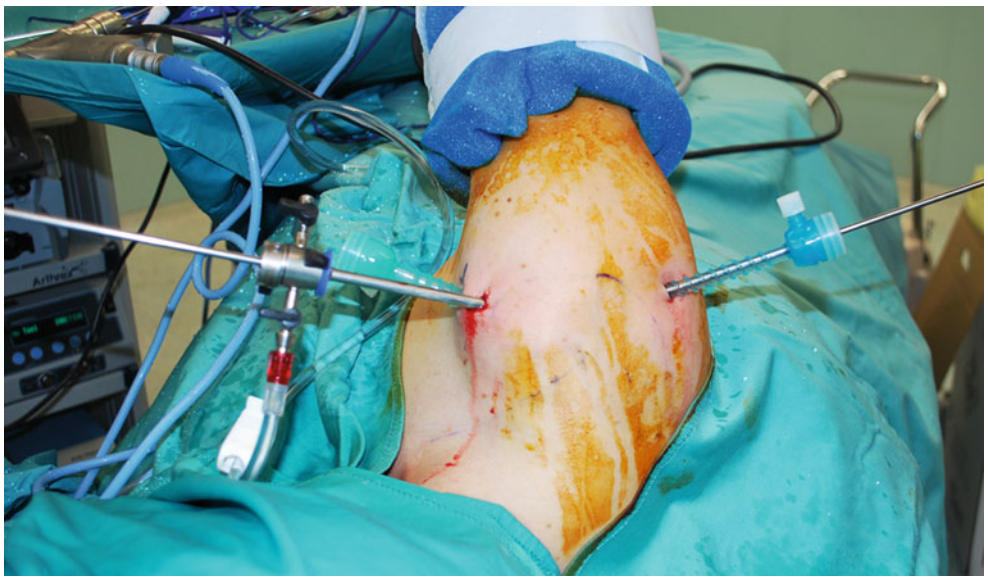

Fig. 7.4 The two switching sticks are used to switch the anterior and the posterior portals

The switching stick can also be used as probe to evaluate texture, thickness, and mobility of an anatomical structure and the tension of a repair after the operation.

Dilators

Dilators are metal instruments that are utilized to dilate the arthroscopic portals in order to facilitate the passage of the cannulas. They are cannulated so they can easily slide over the switching stick, which acts as the guide (Fig. 7.5).

Wissinger Rod

This metal rod has a head and blunt ends; it is used to create an access portal with an inside-out technique which involves the following steps:

- Place the arthroscope with its sheath on the point where the access portal is to be created
- Remove the arthroscope while keeping the sheath in place
- Insert the Wissinger rod into the sheath of the arthroscope until the tip touches the skin

Fig. 7.5 Dilators. Different colors indicate different calibers. They are cannulated, so they can easily slide over the switching stick and facilitate passage of the cannulas, without losing portals

- Create an incision using a scalpel and place a cannula on the tip of the Wissinger rod
- Remove the Wissinger rod after having placed the cannula in the joint and repositioned the arthroscope

Probe

The probe is an instrument with a curved end which represents the "extension of the surgeon's finger." It is inserted into the joint through an arthroscopic portal. In the diagnostic phase, it allows for palpation of the lesion and assessment of its mobility (Fig. 7.6). Some probes are graduated and, hence, are able to estimate the size of the lesion.

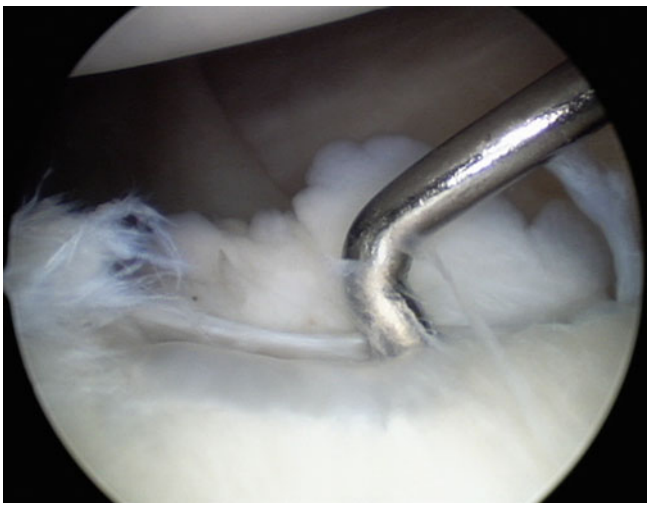

Fig. 7.6 Probe is used for palpation of a lesion of the anterior glenoid labrum

Chisel Dissector

Available in various sizes, the chisel dissector is characterized by a flat and sharp end. It is primarily used in instability surgery and enables the surgeon to loosen scar adhesions and to adequately mobilize the capsulolabral complex from the glenoid neck (Fig. 7.7).

Rasp

An instrument utilized to abrade bone surfaces and/or capsular tissue to create bleeding (Fig. 7.8). This same procedure can be performed using a motorized instrument.

Cutting Instruments

Various cutting instruments are currently available on the market, and they each perform different functions.

Punches

Punches are a particular type of basket scissors; they can be straight, curved, or angled (upturned, right, left) with antegrade or retrograde bite (Fig. 7.9). Because of their shape, they enable the surgeon to reach areas which are typically

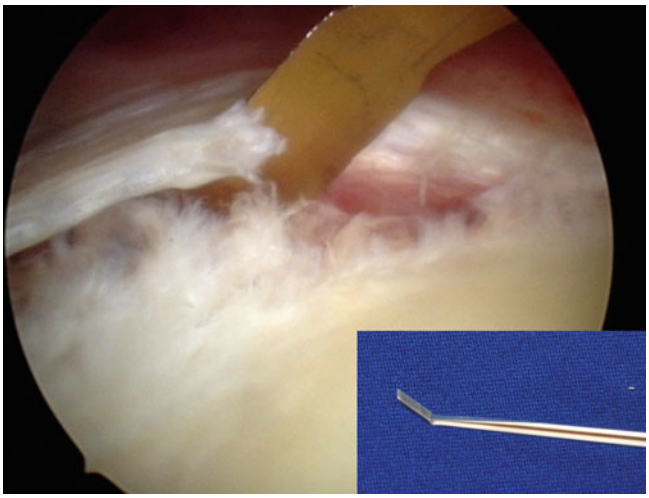

Fig. 7.7 Chisel dissector is characterized by a flat and sharp end. It is used to elevate and mobilize scarred tissue to be repaired

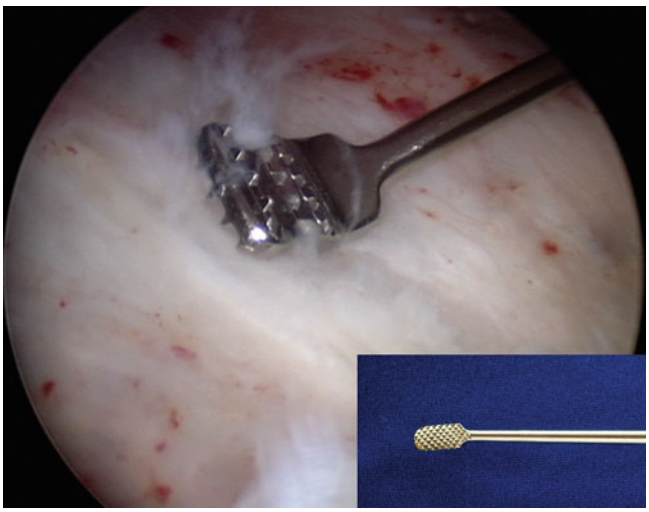

Fig. 7.8 Arthroscopic rasp. It is used to abrade bone surfaces and/or capsular tissue

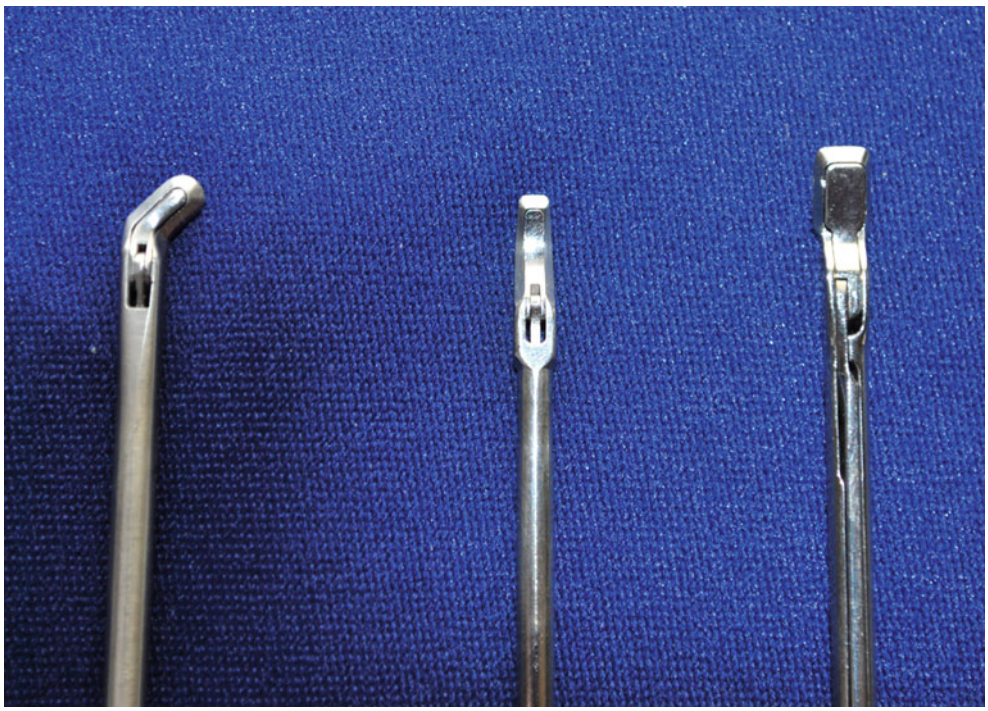

Fig. 7.9 Different types of basket scissors: right angled, straight, and upturned with anterograde bite

difficult to access. The use of basket punches in shoulder surgery is currently limited because of the widespread utilization of motorized or radiofrequency tools. Nevertheless, punches are generally used for the removal of capsular (i.e., arthroscopic capsular release) or tendon (i.e., atrophic edge of a rotator cuff lesion) tissue.

Scissors and Suture Cutters

The scissors can be straight or curved. They are frequently utilized to cut soft tissue, such as the rotator cuff (i.e., during an interval slide procedure), the capsule (i.e., during an arthroscopic capsular release), or the long head of the biceps (tenotomy), as an alternative to radiofrequency instruments. They can also be used to cut the suture strands after a knot has been tied. Suture cutters were designed to facilitate arthroscopic cutting of high-resistance braided sutures, such as FiberWire, and are available in a closed and open end. The suture strands are carried by the instrument to the outside of the joint; the instrument then slides through the cannula to the point where cutting is needed (Fig. 7.10).

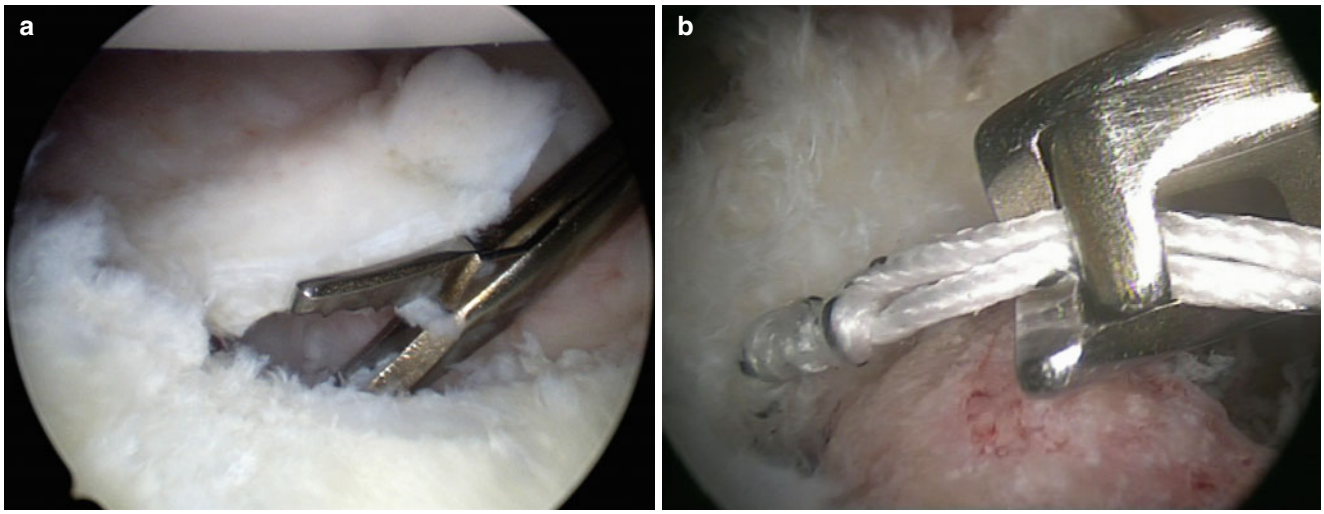

Fig. 7.10 (**a**) The basket scissors are used to perform capsular release. (**b**) Suture cutter is used to cut high-resistance braided suture

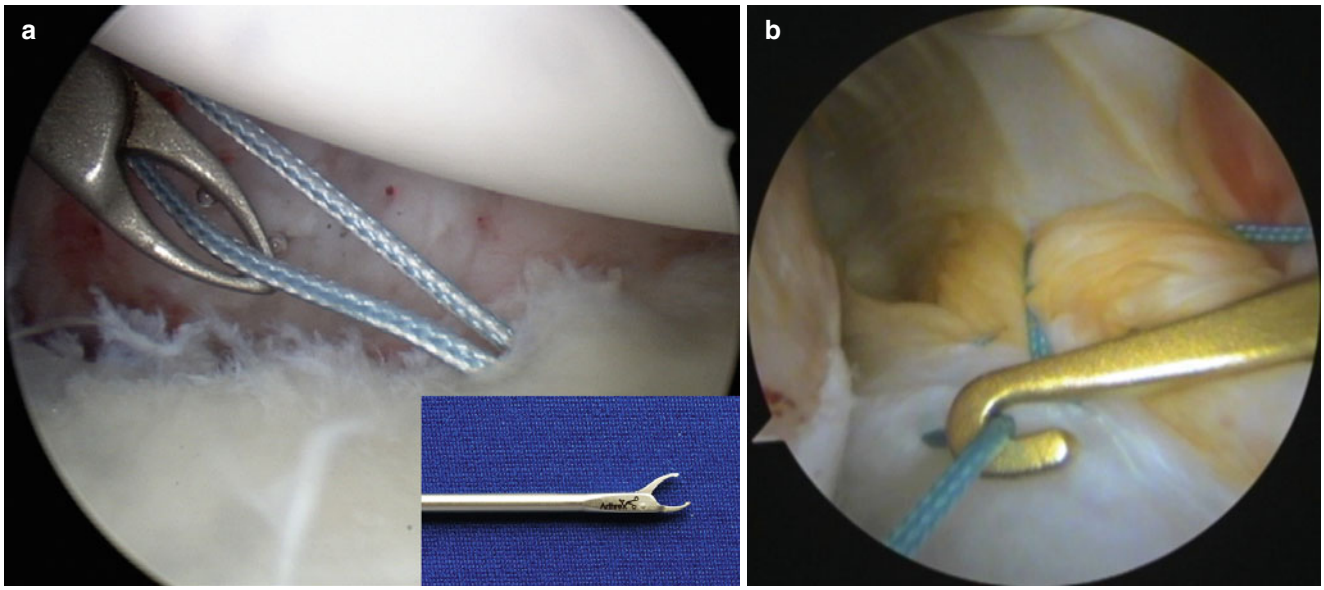

Fig. 7.11 (**a**) Suture retriever. The jaw creates a closed loop (*see* inset). (**b**) The crochet hook is used to recover a suture strand

Grasping Instruments

A wide variety of grasping forceps are available in various sizes and with different bites; they are made out of metal and have either straight or slightly curved tips. They can be locking, non-locking, or with self-releasing locking mechanism.

Suture Retrievers

Suture retrievers are used to recover and manage the suture strands. The jaw creates a closed loop which allows the suture to slide freely during suture extraction.

The crochet hook is another simple tool that performs well in tight spaces to retrieve suture loops during any suturing procedure. The smooth tip prevents abrasion of suture strands, and the ergonomic handle facilitates instrument manipulation in the wet arthroscopic environment (Fig. 7.11).

Graspers

Graspers can be blunt, serrated, or hook shaped. A fundamental requirement of these forceps is the ability to provide an atraumatic grasp that does not compromise the integrity of the structure. They can be used for tissue grasping and/or reduction, foreign and loose body removal, minor arthroscopic biopsies, and suture retrieval and management (Fig. 7.12).

Suture Passers

The role of suture passers is to allow for the passage of suture strands through the soft tissue (tendons or capsulolabral tissue). They are divided into two types: direct and indirect. Direct suture passers enable the passage of the suture directly through the tissues without using suture shuttles. Passers can

be further classified as antegrade or retrograde based on the way in which they are utilized. The type of lesion and quality of the tissue determine which instruments will be used. The technique of passing the sutures through the soft tissue will be discussed in Chap. 13.

Direct Suture Passers

Direct passages are frequently used in rotator cuff repair surgeries. All suture passers are equipped with a safety-lock feature which prevents accidental opening of the forceps during the introduction or extraction of the suture.

For direct antegrade passages, we use suture passers preloaded with a single-use needle, which can be used for all the sutures of a single operation. Before introducing it into the joint, the suture is loaded on the passer's bite. Once in the joint, the passer's bite enables adequate grasping of the free end of the rotator cuff (up to 16 mm), and the preloaded needle pushes (with a direct antegrade approach) the suture through the tissue. Most modern passers have a suture capture trap which allows for suture retrieval during the extraction of the passer; alternatively, a grasper can be used to retrieve the sutures (Fig. 7.13).

For direct retrograde passages, instruments with a sharp end and an open loop, dorsal or ventral, are utilized. They are available with different angles of curvature. The sharp end facilitates the passage of the instrument through the tissue, and the loop aids in the retrieval of the sutures (Fig. 7.14).

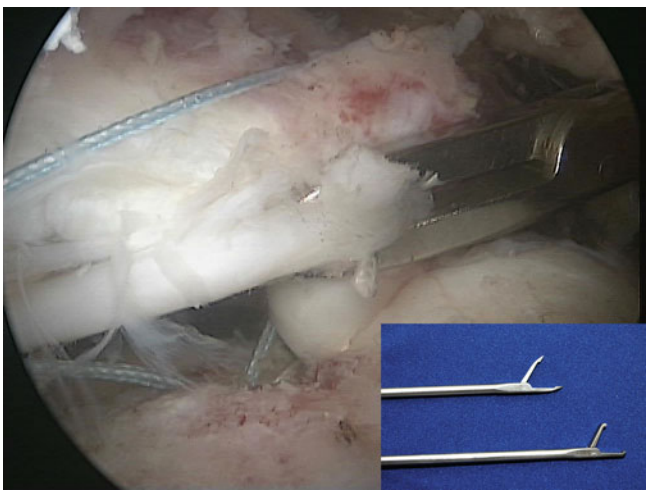

Fig. 7.12 Graspers can be blunt, serrated, or hook shaped (*see* inset). They can be used for tissue grasping, loose body removal, and suture retrieval

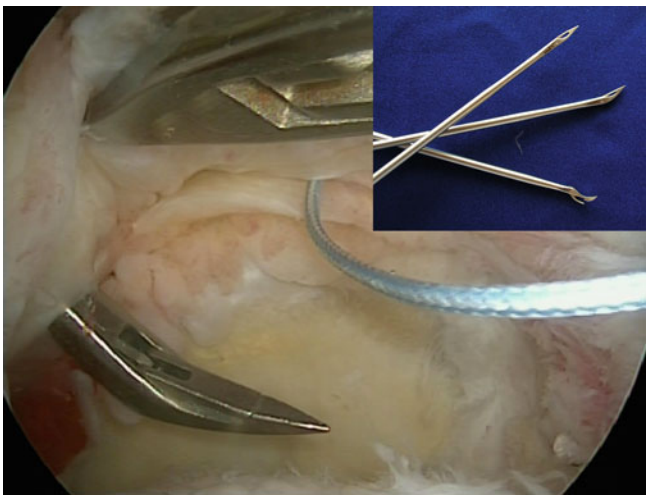

Fig. 7.14 Direct suture passers for retrograde passages. They can be straight or angled and are characterized by a sharp end and an open loop, dorsal or ventral (*see* inset)

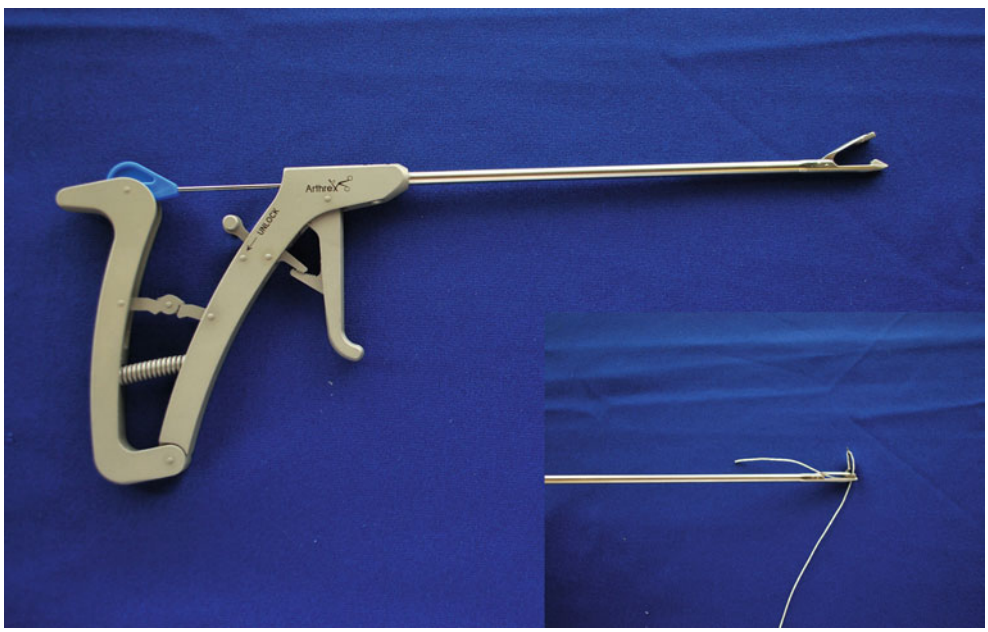

Fig. 7.13 New generation direct antegrade suture passers have a suture capture trap (*see* inset) which allows for suture retrieval during the extraction of the passer

Indirect Suture Passers

Indirect suture passers rely on a suture shuttle to pass the suture through the tissue using a retrograde approach. They are hook-shaped instruments with different inclinations, curvatures, and sizes which allow them to be used effectively into the joint. They are primarily used in the repair of the capsulolabral complex for the treatment of shoulder instability or for certain techniques in rotator cuff repair (i.e., margin convergence). The distal end of the suture passer is cannulated to enable passage of the suture shuttle on which the suture is loaded. This suture shuttle can be manually loaded or preloaded in the instrument and consists of either a monofilament suture or a metal wire coated in a plastic film to make it atraumatic, with an eyelet along its length or at the distal end in which the suture is loaded (Fig. 7.15).

Knot Pusher

The knot pusher allows the knot to be pushed through the cannula into the joint. There are various configurations of knot pushers available on the market: standard single hole, cannulated double-diameter single hole, standard two hole, and modified two hole that, pulling the knot, opens mechanically (Fig. 7.16).

Golden Retriever

The golden retriever is a metal tube (4.2 mm diameter) with a magnet at one end. It is used to recover any metal pieces which have dropped in the joint due to instrument breakage. For this reason, it is fundamental that all the arthroscopic instruments have magnetic properties. The golden retriever functions with

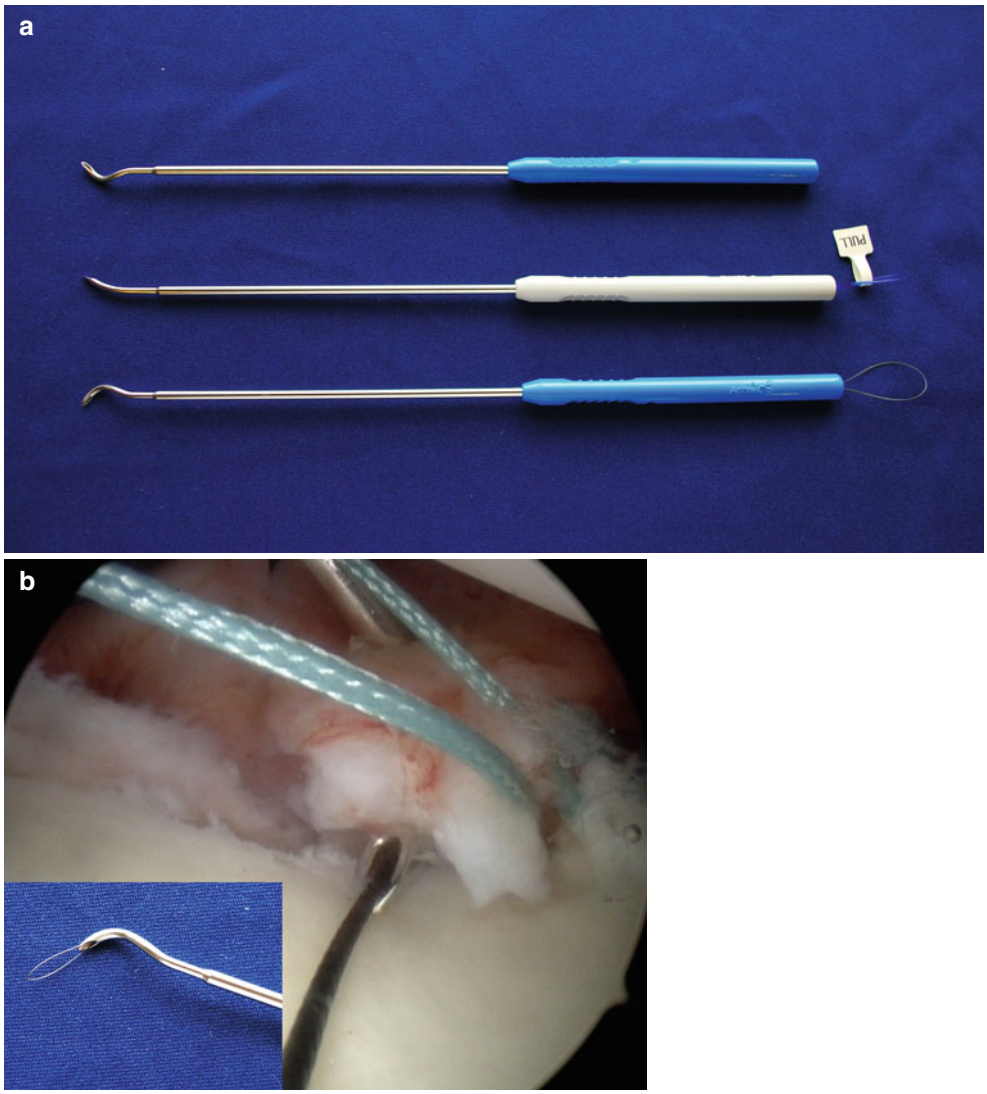

Fig. 7.15 (**a**) Indirect suture passer can be straight or angled (*right*, *left*). (**b**) The distal end of the suture passer (*see* inset) is cannulated to enable passage of the suture shuttle on which the suture will be loaded

both applied suction forces and magnetic power. The suction serves to mobilize the fragment guiding it towards the magnetic field. It was by design that the golden retriever cannot deliver suction as powerful as it would seem to be able to. Therefore, the suction should be removed as soon as the metal piece approaches the magnet, ensuring solid contact between the two surfaces without soft tissue interposition.

The golden retriever cannot be utilized without a cannula. The cannula has the dual function of allowing for the visualization of the retrieved fragment and facilitating its extraction by preventing it from getting lost again in the extra-articular soft tissue. The cannula is removed while it still contains the golden retriever and the recovered fragment.

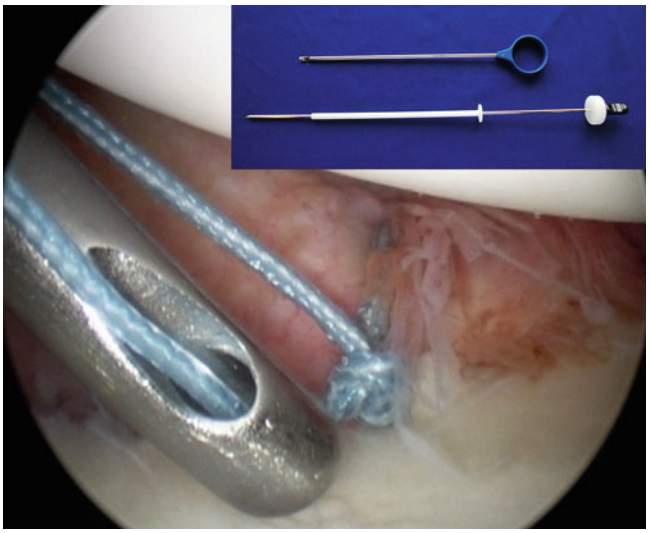

Fig. 7.16 The knot pusher is used to tie the arthroscopic knots. Different types of knot pusher (*see* inset) can be used

Powered Instruments

The shaver is an instrument equipped with a handpiece in which single-use blades, with different shapes and functions, can be inserted. The control unit is inserted in the arthroscopic tower, and it is controlled by footswitch placed near the surgeon's feet or by buttons on the handpiece. The shaver has a suction tube; the suction is managed either directly by the surgeon with a control on the handpiece or by an assistant who adjusts the suction by manually clamping the tube. The suction serves to remove loose tissue or bone fragments in the joint which were generated by the shaver. The rotation speed of the blades is automatically set by the control unit which is able to recognize the type of blade and then adjust the speed from a minimum of 100 to a maximum of 8,000 revolutions per minute. Nevertheless, it is possible to manually modify the setting based on the preferences and needs of the surgeon. The footswitch is used to control the direction of blade rotation: forward, oscillating, or reverse. The blades can be grouped into two major categories: those used for soft tissues and those used for bone. Blades used for soft tissues can have a single or double cutting edge, smooth or toothed. Among the blades used for bone, we can distinguish between those used for cortical abrasion before positioning the anchors (round burr) and those used for

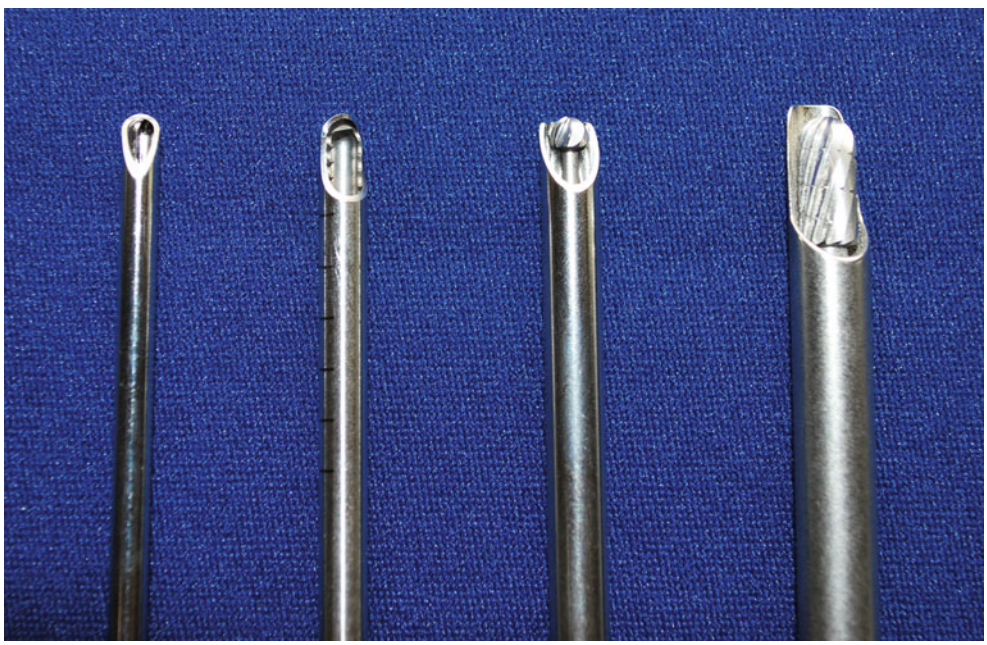

Fig. 7.17 Motorized shaver blades (*from left to right*): smooth for soft tissue, toothed for soft tissue or gentle cortical abrasion, round burr for cortical abrasion, and oval burr for acromioplasty

acromioplasty (oval burr), which are more aggressive (Fig. 7.17). The diameter of the blades can vary according to the model and manufacturer and is selected based on the surgical procedure. The blades most commonly used are medium sized (between 3 and 5 mm). The blade's ability to cut or abrade does not depend exclusively on rotation speed or type of blade employed, but also on the surgeon's ability to manage the instrument. Generally, the harder the tissue, the fewer revolutions per minute are necessary, but greater pressure must be applied on the instrument. The opposite is true for the soft tissues. The hand that guides the instrument is the main determinant of its effectiveness.

Electrosurgery

The use of radiofrequency equipment helps to effectively control bleeding which, together with the irrigation pump, contributes to optimizing the arthroscopic view. The system consists of a radiofrequency generator placed on the arthroscopic tower, a single-use handpiece with integrated electrode, and footswitch control. Reusable handpieces, in which single-use electrodes are inserted, and systems equipped with hand control are also available. The electrodes used vary in terms of shape, size, and angle of curvature. They are selected by the surgeon based on the surgical procedure to be performed. Inside each electrode, there is a code that generates a signal which is transmitted to the generator for the setting of the instrument. There are two basic types of thermal instruments: monopolar and bipolar. Monopolar instruments utilize an "active" electrode placed at the end of the handpiece and a "return" electrode applied to the patient. Bipolar instruments have the active electrode and the return electrode located in the surgical instrument, thereby minimizing the amount of tissue involved in the electrical circuit. Harnessing thermal energy, these instruments allow for effective management not only of coagulation but also of tissue cutting and ablation procedures. The greatest risk associated with the use of these instruments is necrosis induced by high temperatures [9–12].

The thermal effects of radiofrequency waves on the tissue are determined by the following factors: level of energy (power and impedance), duration of the treatment, characteristics of the tissue, type (mono or bipolar), and shape and size of the electrode. Some systems are able to monitor the temperature of the active electrode.

A new method of high-frequency electrosurgery, coblation or "cold ablation" technology, utilizes radiofrequency energy but generates much less heat. This method induces molecular dissociation. Saline solution, typically used in arthroscopy, is introduced in the space between the tissue and the electrode. When the electric current is applied, it creates a layer of charged particles which is referred to as a "plasma" layer. The particles in the plasma layer have enough energy to break the molecular bonds, which results in volumetric removal of target tissue at relatively low temperatures, therefore, minimizing damage to surrounding healthy tissues. The majority of the thermal energy is consumed in the plasma layer due to ionization.

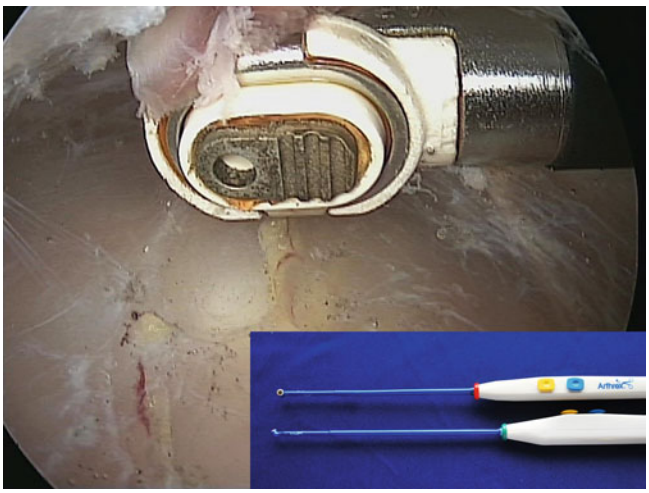

Fig. 7.18 Radiofrequency instrument used to perform subacromial bursectomy. The electrodes vary in shape, size, and angle of curvature (*see* inset)

From a practical point of view, the radiofrequency device helps to achieve pinpoint control of any bleeding; it is indispensable in bursectomy and synovectomy because these procedures involve highly vascularized structures. Radiofrequencies allow for a volumetric reduction of tissues while controlling bleeding and maintaining a clear vision of the operating field (Fig. 7.18).

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Operating Room Setup and Patient Positioning

8

Matteo Salvatore, Carmine Latte, and Andrea Grasso

Arthroscopy has made it possible to define many of the known lesions related to shoulder disorders and to identify other, new ones. The surgeon must be familiar with these different lesions and with their surgical treatment. Although careful planning of an appropriate therapeutic procedure on the basis of clinical and instrumental examinations is crucial, the surgeon, faced with the arthroscopic diagnosis, is often required to use different techniques from those initially planned. In shoulder arthroscopy, the ideal setup is therefore one that is versatile enough to allow the execution of different surgical procedures. Achievement of this objective depends on careful planning and preparation of the instrumentation.

The surgical team, the anesthesiologist, and the operating room staff all play a part in setting up the operating room: only through teamwork is it possible to ensure optimization of complex and versatile surgical procedures.

Operating Room

General Requirements

The success of the operation depends on correct operating room arrangement and setup. A dedicated, well-equipped operating room staffed by a specialist arthroscopy team constitutes the ideal working environment. Having an area specifically set aside for shoulder arthroscopy means that the surgeon and staff are able to enter the theater knowing that everything will be in its proper place: operating table, arthroscopy column, fluids, stands, etc. Any reasonably sized general surgery or orthopedic operating room can be prepared for shoulder arthroscopy, providing this is done by personnel with specific training and expertise in arthroscopy (Fig. 8.1).

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Operating Table

Shoulder arthroscopy can be performed with the patient in the lateral decubitus position or in the beach-chair position. In both cases, a standard Mayo orthopedic table is used. The Mayo table is positioned in the center of the operating room or at least at 45° angle to the long side of the room (Fig. 8.2).

If the beach-chair position is to be used, the table must allow raising of the patient's trunk with flexion of the hip and knee joints and lateral and longitudinal tilting; furthermore, it must be adjustable in height so that the shoulder can be positioned at the correct level in relation to the surgeon. Access to both the anterior and posterior aspects of the shoulder can be guaranteed by using a special head support or modular elements to support the chest, with removable sections in the shoulder area (Fig. 8.3).

Surgeon's Position

The most versatile position for the surgeon is at the proximal end of the operating table; in assuming it he occupies what is traditionally the anesthesiologist's position. In this position, the surgeon has complete access to the anterior, posterior, upper, and lateral aspects of the patient's shoulder. Each of these areas can become crucial during a surgical procedure and must therefore be fully accessible. The "head-of-the-bed" position is also the most indicated when switching to an open surgical phase: the surgeon has a good view of the entire surgical field, and it is easier for the assistants to retract the tissues.

Anesthesiologist's Position

The anesthesiologist stands opposite the surgeon, proximal to and at an angle of around 45° to the patient's head.

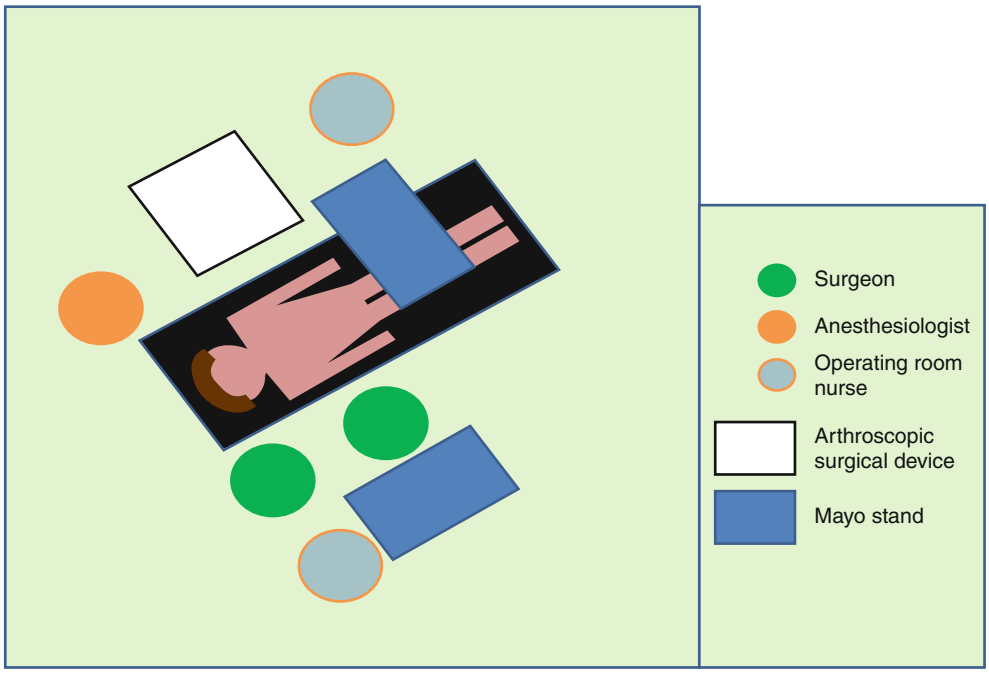

Fig. 8.1 Operating theater arrangement and setup for shoulder arthroscopy

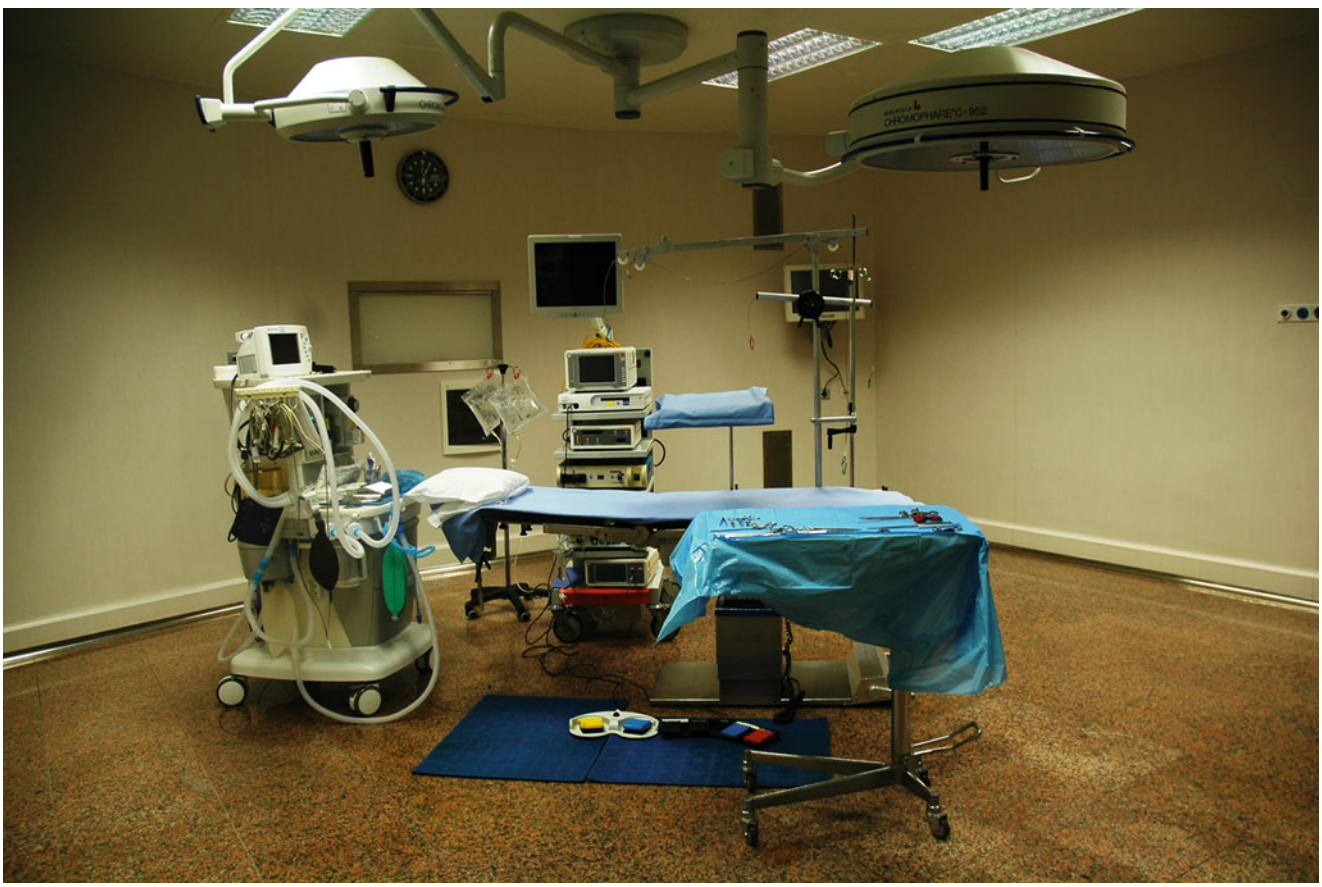

Fig. 8.2 Operative room setup for lateral decubitus position

Fig. 8.3 Operative room setup for beach-chair position

Arthroscopy Unit

The arthroscopy unit is equipped with all the devices that comprise the standard equipment for correct execution of an arthroscopy procedure (see Chap. 7). It is arranged in two columns on two portable trolleys. Arranged on the first trolley, which must be tall enough to allow a good view of the arthroscopic image, are, from top to bottom, the monitor, the video camera, the light source, the motorized system, the radiofrequency system, the video recorder, and the video printer. The second trolley contains the arthroscopy pump together with the holders for the bags of fluid used to distend the joint, which, to guard against the risk of leakage from the system, are kept away from the electrical instruments. If the injection pressure is measured on the fluid delivery line rather than directly on the arthroscope, particular care must be taken over the height at which the arthroscopy pump is positioned, in order to ensure that it can be correctly calibrated in relation to the blood pressure values: it is generally positioned at the level of the patient's chest.

The suction system must be equipped with at least two separate collection bags, so that it is possible to switch to the second as soon as the first is full.

The arthroscopy column is positioned along the opposite side of the table, opposite the surgeon, so that he is easily able to see the images on the monitor and can directly keep all the devices under control. The second trolley (containing the arthroscopy pump and distension fluid bag holders) and the suction system are placed alongside and distally to the arthroscopy column.

Stands

The main stand is situated just behind the surgeon. It contains the surgeon's arthroscopic instruments and the other specific surgical sets (Fig. 8.4).

The first Mayo stand, positioned next to and within easy reach of the surgeon, contains the instruments needed to mark the bone contours, prepare the arthroscopic portals, and introduce the arthroscope: 20 ml syringe of distention fluid, scalpel blade n°11, skin-marker pencil, 19-G spinal needle, cannulas, blunt trocars for the arthroscope sheath and cannulas, switching stick, and Wissinger rod.

The arthroscope, the motorized handpiece, the electrosurgery system electrode, and the infusion and suction tubes are

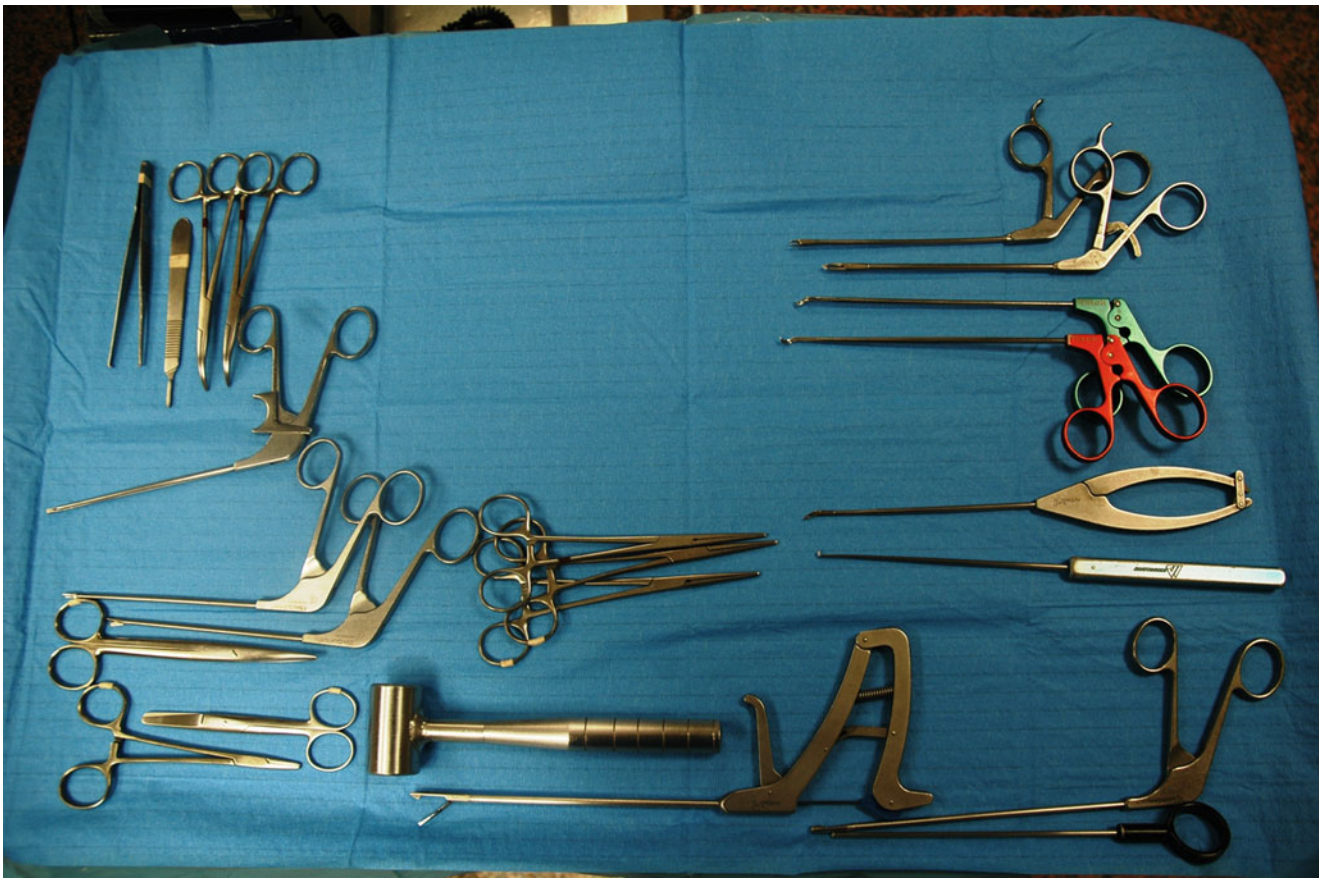

Fig. 8.4 Main stand arranged with surgeon's arthroscopic instruments

arranged on the second Mayo stand, positioned above the patient on the opposite side of the operating table.

Fluid Collection on the Floor

Two rectangular suction mats for collecting waste fluid are placed in an L shape on the floor at the proximal corner of the operating table, under the position of the shoulder to be operated on.

Operating Room Staff

Nothing favors efficient work more than the presence of competent staff who are familiar with the techniques and instruments used. Since surgeons do not always have their own staff available – for most this is a luxury – it is crucially important to establish standardized protocols according to which staff members have their own specific tasks, which they perform in accordance with clearly defined rules and directives. This creates the right feeling, confidence, and spirit among the staff and enables them to work in an environment where everything is optimally set up.

The instrument technician is responsible for preparing the main instrument stand and the two Mayo stands. The scrub nurse arranges and sets up the various components of the room (operating table, arthroscopy column, fluid collection mats), looks after the positioning and preparation of the patient, prepares the instruments and monitors their functionality during the operation, and responds to any requests from the surgical team.

Patient Preparation

In the Department or at Home

The patient comes to the surgical unit from the department, in the case of an inpatient, or from home, if the treatment is being performed as a day-hospital procedure. In any case, the patient is always instructed beforehand not to assume food or liquids after midnight the night before the operation. His body must be thoroughly cleaned with an antiseptic skin

cleansing fluid, paying particular attention to the arm to be operated on (armpit, hand, nails, etc.). The arm in question is marked using a skin-marker pencil; the mark is made on the lateral aspect of the arm.

Trichotomy

Complete trichotomy of the arm, axilla, and ipsilateral hemithorax is performed in the department or in the admission room in the case of a day-hospital procedure. It is necessary to check that this procedure has been correctly performed before the patient enters the operating room.

Checking the Patient's Documentation

Before the patient enters the operating room, the surgeon must check that all the patient's documentation, clinical and instrumental, is present: fully compiled medical records, preoperative examinations, informed consent, and radiological examinations (X-rays, MRI scans, CT scans). A member of the surgical team must mount the most significant radiological images on the negativoscope in the operating room before the start of the operation. Alternatively, electronic material (CDs, DVDs, or images on PACS: picture archiving and communication system) should be uploaded and checked on a computer in the operating room or surgical unit.

Patient Positioning

Positioning the patient is one of the steps in shoulder arthroscopy whose importance is often underestimated, thereby compromising the success of the operation. Correct positioning of the patient on the operating table is important for the patient's safety and also because it guarantees the surgeon optimal surgical access to the glenohumeral joint and subacromial space in order to perform the procedure. Incorrect positioning of the patient can restrict the surgeon's maneuvers and interfere with the handling of the instruments; it can prevent precise placement of the portals and anatomical arrangement of the lines of force for the traction of the limb, and it can facilitate the onset of complications resulting from injuries caused by compression or stretching of nerves.

In addition to guaranteeing excellent exposure of the arthroscopic portals, the position must be such that the patient's respiratory and circulatory function is not compromised, the peripheral neurovascular structures are protected against possible compression injury, and, in the case of regional anesthesia, the anesthesiologist can work in comfort and the patient is comfortable.

Shoulder arthroscopy can be carried out with the patient in the lateral decubitus position or in the beach-chair position. The surgeon indicates the required position beforehand, in the list of surgical specifications. The scrub nurse consults the surgeon personally before the operation to confirm the type of position indicated in the list. It is the surgeon's responsibility to verify the protection of neurovascular structures. The nurse prepares the various supports and accessories necessary for positioning the patient and then coordinates the process.

Lateral Decubitus Position

A U-shaped beanbag surgical positioner (Olympic Vac-Pac; Natus Medical Inc., San Carlos, CA, USA) is placed on the operating table with the base of the U positioned at the level of the scapula, and, on top of it, an anti-decubitus gel pad (Fig. 8.5). The patient is moved onto the table and positioned on his contralateral side. Given that there is a risk of compression injury to the contralateral brachial plexus, an anti-decubitus gel pad must be placed between the operating table and the axilla in an attempt to prevent this complication with under his axilla. If the patient is under general anesthesia, the anesthesiologist monitors the patient's head and coordinates the actions of a nurse and surgeon, one on each side of the table, as they rotate the patient onto his side. The patient's head is positioned on a double pillow. Another pillow is placed between his legs to avoid stress on the hip and knee joints. The bony protuberances of the contralateral elbow, hip, knee, and ankle are protected with a gel pad. Gross and Fitzgibbons [1] modified this position, rotating the patient 30–40° posteriorly, so as to position the glenoid surface on a horizontal plane. This slight but effective modification allows more comfortable maneuverability of the instruments, correct placement of the glenohumeral joint rim in horizontal position, and a more anatomical arrangement of the lines of force during the traction. Because this latter factor allows less force to be applied during the traction, it eliminates the occurrence of traction-induced brachial plexus injuries [2].

Lateral supports for the gluteal region and sternum are positioned. While keeping the Vac-Pac wrap adherent to the patient, a suction device is used to create a vacuum in it, thus stiffening the structure; this is secured in position to the bed using a strap, and the patient is covered with a thermal drape (Fig. 8.6). A 3-Point Shoulder Distraction System (Arthrex, Naples, FL, USA) is then positioned at the distal end of the table on the contralateral side. This allows positioning of the arm in between 0° and 70° of abduction and between 0° and 30° of flexion, making it possible to obtain sufficient distension of the glenohumeral joint and subacromial space. This position was originally described by

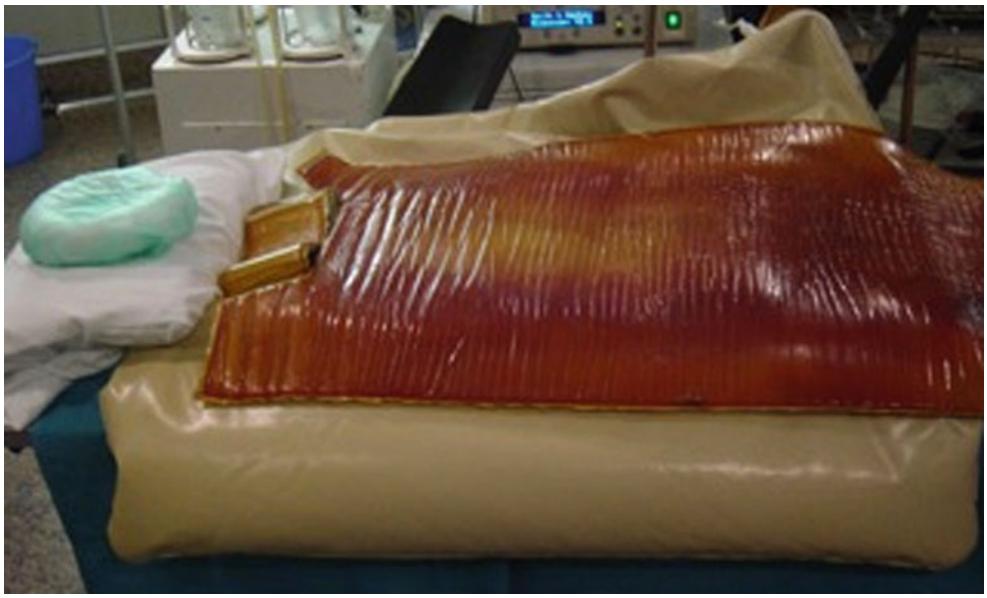

Fig. 8.5 Operating table setup for the lateral decubitus position. A U-shaped beanbag surgical positioner and a protective anti-decubitus gel pad are placed over the table

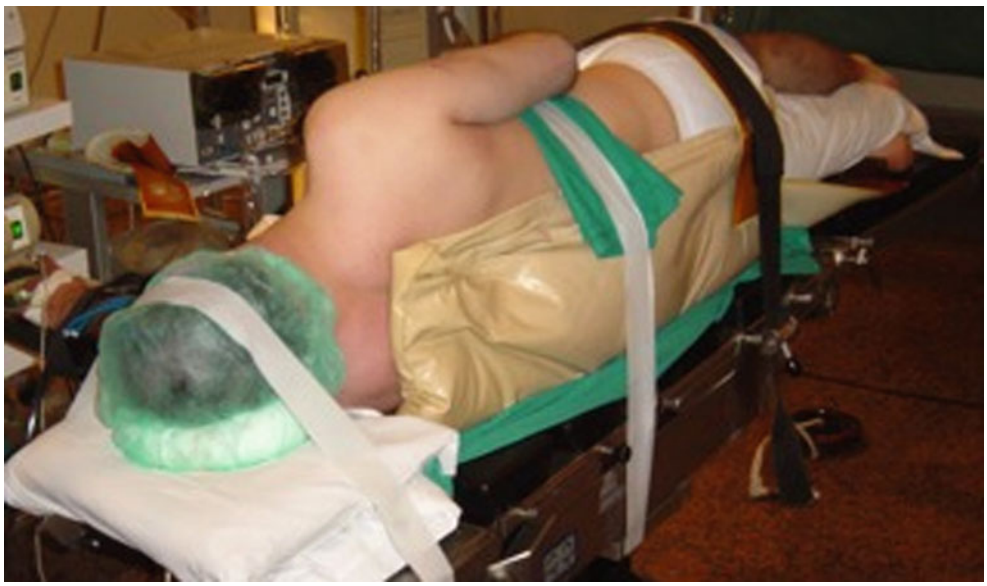

Fig. 8.6 Lateral decubitus position. The beanbag surgical positioner wraps the patient's body. Bony protuberances are protected with pillows and pads

Andrews et al. [3] and involved placing the arm at 70° of abduction and 15° of forward flexion. The 15° forward flexion is mandatory as it reduces the risk of injury due to traction of the brachial plexus [4]. This is a not infrequent complication, and it is a risk that the surgeon should always bear in mind. Several authors have studied its incidence, analyzing it in relation to the position of the limb and the weight applied as traction [5, 6].

A sterile drape is positioned, at a 45° angle, at the level of the patient's head in order to create a barrier between the surgical field and the anesthesiologist's position, and the second Mayo stand is positioned above the patient (Fig. 8.7).

Beach-Chair Position

The need to define an easier, more versatile, and more comfortable position, for both patient and surgeon, has stimulated the imaginations of a number of distinguished specialists. Hence, in 1988 Skyhar et al. [7] coined the term beach-chair position to describe this second position in which the patient is semi-seated with his trunk inclined at an at least 60° angle and his arm free.

Placing the patient in the "beach-chair" position facilitates exposure of the various landmarks. Its versatility is such that it allows a smooth and easy transition from the arthroscopic to the open surgical phase.

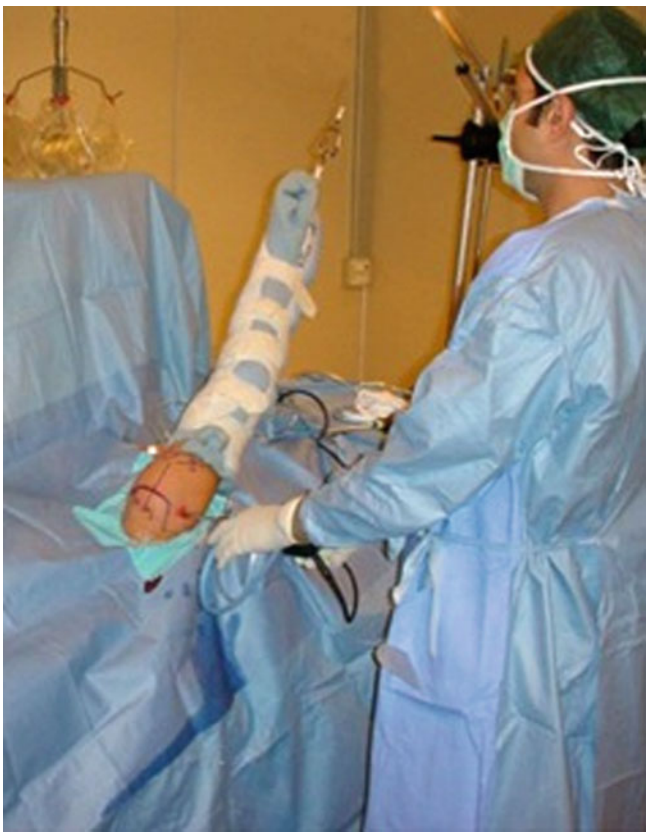

Fig. 8.7 Lateral decubitus position. Operative field is complete. The arm is held in traction and a barrier was placed between the surgical field and the anesthesiologist's position

In this position most of the patient's weight is borne by the gluteal region: the patient is positioned on the operating table in such a way that the gluteal region is directly over the pivot point of the operating table. The table is tilted to the Trendelenburg position. The backrest is raised to obtain a 90° sitting position. A wedge pillow or, alternatively, a flat pillow folded in two is placed under the patient's knees to avoid excessive stress on the myotendinous and neurovascular structures. The end of the operating table is tilted to prevent excessive pressure on the back of the foot. A gel pad is placed under the heels to prevent pressure sores. The patient's legs are secured to the table by means of a strap applied over a gel pad. The arm to be treated can be left free in an arm sling (Fig. 8.8).

The fact that the arm does not have to be put in traction is a huge advantage for the surgeon, as the normal anatomy is respected and no strain is put on the various capsular, ligamentous, and tendinous structures. The absence of traction greatly reduces the risk of brachial plexus injury. Furthermore, with the careful help of an assistant, the arm can easily be positioned as needed.

It should nevertheless be borne in mind that the literature contains several reports of brachial plexus and hypoglossal nerve injury due to hyperextension of the cervical spine [8].

A variation of this position allows traction to be applied with the arm in 0° of abduction and 45–90° of elevation. Traction is applied via a leg support positioned at the end of the table. With this variant, the less experienced surgeon avoids the need to pay attention to the correct arm position also during surgery, repeatedly adjusting it to the different surgical steps. In a detailed cadaver study, Klein and Fu [9] established that this type of traction was the least harmful to the brachial plexus.

The contralateral limb is positioned on a support with an anti-decubitus gel pad. The patient's head, supported by a special headrest which is adjusted in height and extension, is turned slightly away from the surgical field and secured with silk tape applied to the forehead. Two lateral dorsal supports are used to stabilize the trunk (Fig. 8.8). The patient is covered from the chest down with a thermal drape. A sterile drape is positioned, at a 45° angle, at the level of the patient's head in order to create a barrier between the surgical field and the anesthesiologist's position. The arthroscopy instruments are arranged on the second Mayo stand, secured to the table above the patient (Fig. 8.9).

Preparing the Surgical Field

The scrub nurse cleanses the patient's arm, shoulder, and hemithorax with iodopovidone.

The surgeon, wearing adequate protection on his feet (rubber boots), after disinfecting his hands and forearms, puts on a sterile disposable reinforced surgical gown. He starts disinfecting the patient's skin with sterile betadine, initially assisted by the scrub nurse who supports the patient's arm during the disinfection of the hand and forearm. When this is complete, an assistant wearing an impermeable stockinette supports the patient's hand while the disinfection of the arm, shoulder, hemithorax (anterior, posterior, armpit), and neck is completed. The instrument technician covers the patient from the chest to the distal end of the table with a sterile full-length drape. The stockinette is unrolled so that it covers the patient's arm and forearm, and a compression bandage is applied to the arm using cohesive elastic bandage. A U-shaped impermeable drape is made to adhere around the shoulder, taking care to leave enough space on the anterior and posterior hemithorax to allow a large and convenient field for the portals. The two arms of the U drape are rejoined at the base of the neck and made to adhere to each other with their ends on the patient's head. An upper extremity patient isolation drape arranged transversely completes the preparation and, with its two upper ends secured by the scrub nurse to two holders, separates off the anesthesiologist's position. It is necessary to cut the incise film around the hole of the upper extremity drape from which the arm is passed to completely expose the surgical field: an adhesive

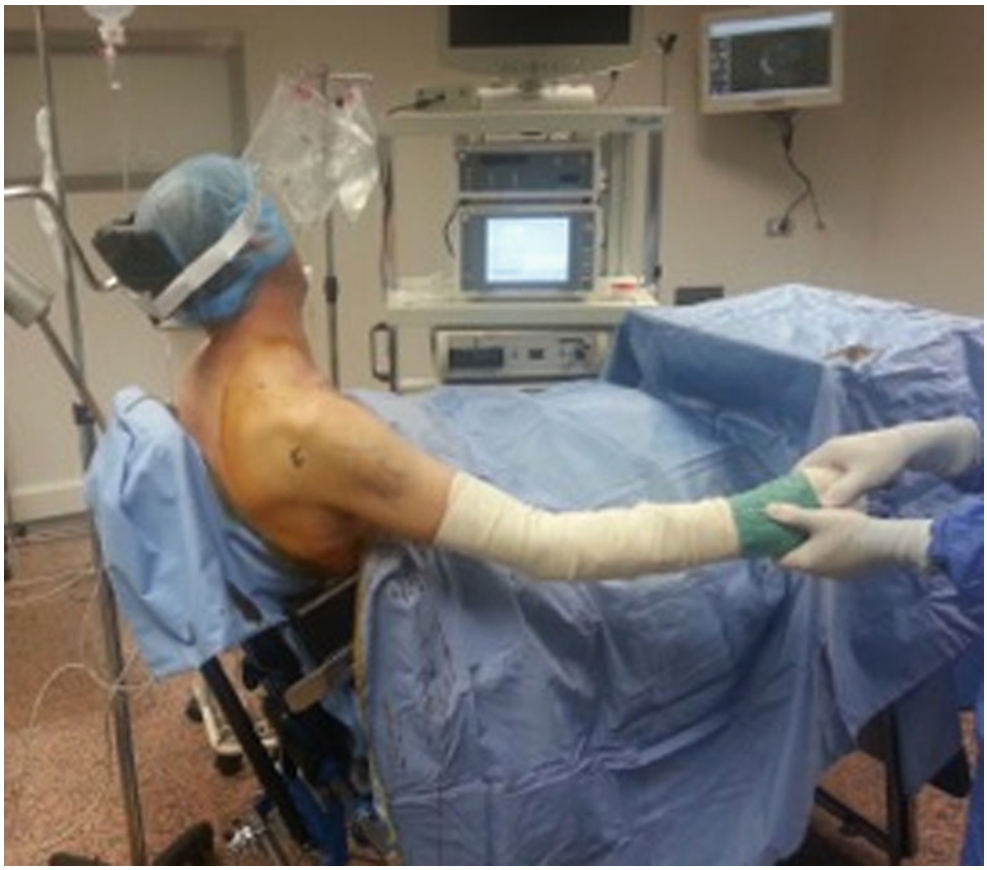

Fig. 8.8 Beach-chair position. The patient's head, supported by a special headrest, is turned slightly away from the surgical field and secured with silk tape applied to the forehead. Lateral dorsal supports are used to stabilize the trunk

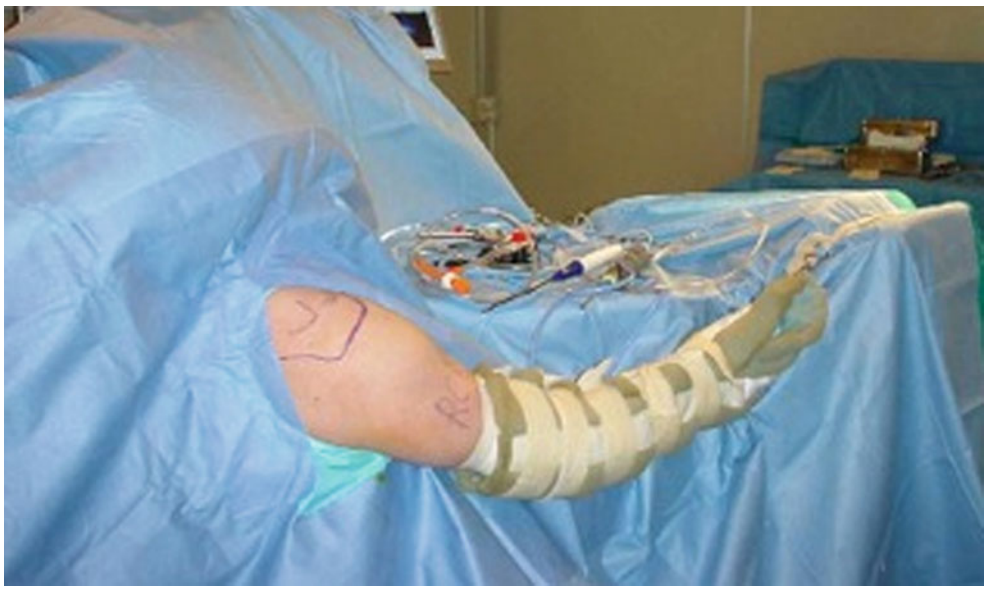

Fig. 8.9 Beach-chair position. Operative field is complete. The arm is held in traction and a barrier was placed between the surgical field and the anesthesiologist's position. The arthroscopy instruments are placed on a Mayo stand above the patient

strip is used to secure the incise film to the underlying skin to prevent fluid strike-through and reduce risk of contamination.

The surgical field is now ready. If the arm has to be placed in traction, care must be taken to avoid compression of bony protuberances and neurovascular structures. The STaR (Shoulder Traction and Rotation) Sleeve (Arthrex) is a useful device; it is a sterile, soft foam traction boot with five Velcro straps: the distal strap is closed first, followed by the others, proceeding proximally. Finally, the distal strap is tightened again. The sleeve is secured to the arm holder by the nurse who then attaches weights to the pulley system.

Once the field is ready and, if necessary, the arm is in traction, the surgeon, using a sterile skin-marker pencil, marks out the bone contours of the shoulder, indispensable landmarks for the correct execution of the arthroscopic portals (see Chap. 10).

Checklists

The *instrument technician* is responsible for preparing the materials and surgical instruments necessary for the procedure.

Necessary Materials for the Surgical Field

- U-shaped impermeable drape
- Upper extremity patient isolation drape
- Impermeable stockinette
- Two full-length drapes (one for the main stand, one for the patient)
- Two Mayo stand covers
- One Fixona bandage
- One 90×150 cm drape to cover the main stand
- Two 75×90 cm drapes to cover the Mayo stands
- One adhesive strip
- One sleeve for traction of the arm
- Reinforced surgical gowns
- Surgical gloves

Surgical Equipment

- One pouch
- Gauze swabs 10×10
- Compresses
- Scalpel blade n° 11
- One 19-G spinal needle
- Two 20 cc syringe
- Cannulas
- Blades for the motorized shaver (full radius blade, acromionizer blade)
- Electrosurgical device (radiofrequency)
- Pump circuit

Instruments

- Basic arthroscopy set
- Surgeon's arthroscopy set
- Other specific sets
- Individually packaged instruments

The scrub nurse is required to:

- Transport the patient to the operating room.
- Consult the surgeon about the type of position required.
- Coordinate positioning of the patient.
  - For operations performed in the beach-chair position:
    - Move the patient onto the operating table.
    - Tilt the table to the Trendelenburg position.
    - Raise the backrest to obtain the sitting position.
    - Place a pillow, folded in two, under the patient's knees and secure them with a strap.
    - Lower the end of the table.
    - Apply the support with anti-decubitus gel pad for the contralateral arm.
    - Block the arm with a band.
    - Apply the support for the arm on the operative side or the leg support for traction at the end of the table.
    - Apply the dorsal supports, the larger on the operative side and the smaller on the contralateral side.
    - Adjust the headrest in height and extension; position the patient's head so that it is turned away slightly from the operative side.
    - Secure the head using silk tape applied to the forehead.
  - For operations performed in the lateral decubitus position:
    - Position the Vac-Pac and anti-decubitus gel pads on the operating table.
    - Move the patient onto the operating table.
    - Position the patient on his contralateral side with an anti-decubitus gel pad under his axilla. If the patient is under general anesthesia, this operation is carried out together with the anesthesiologist and the surgeon.
    - Raise the patient's head on two pillows.
    - Attach the arm sling with gel pad to the operating table.
    - Place a pillow between the patient's legs.
    - Place anti-decubitus gel pads under the patient's knee and malleolus.
    - Wrap the Vac-Pac around the patient.
    - Place supports in the sternal and gluteal regions.
    - Create a vacuum in the Vac-Pac.
    - Fix the traction system to the end of the table, on the other side.

  Once the patient has been positioned:
- Cover the patient with a thermal drape.
- Position the sterile drape.
- Cleanse the limb with iodopovidone.
- Position the two waste fluid suction mats on the floor on the side of the table where the operation is to be carried out.

  Once the preparation of the surgical field is complete:
- Connect up the camera head and fiber-optic cables from the sterile field to the arthroscopic tower.

- Adjust camera color balancing.
- Enter the patient's data in the documentation systems (video printer or imaging system).
- Prepare the 5,000 ml bags of saline, putting them in their holders.
- Receive the sterile pump circuit from the surgical field and connect it with the pump, bags, and suction devices.
- Start the pump.
- Connect up the pedal of the motorized handpiece and place it within the surgeon's reach.
- Connect up the power cable of the sterile motorized handpiece from the surgical field.
- Connect up the sterile electrosurgery system handpiece from the surgical field.

The surgeon must:

- Check the patient's documentation and imaging studies.
- Check that the patient has received prophylactic antibiotics.
- Help to position the patient.
- Check that the positioning of the patient has been performed correctly.
- Check that the peripheral neurovascular structures are properly protected.

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Anesthesia in Shoulder Arthroscopy

9

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and Daniela Maria Micci

Introduction

Surgical procedures in shoulder arthroscopy can be performed under regional blockade, general anesthesia, or a combination of the two techniques. The anesthesiologist's preoperative assessment is crucial to the formulation and execution of the anesthetic plan. The patients must be evaluated for coexisting medical problems, potential airway management difficulties, and considerations related to intraoperative positioning. This evaluation together with an understanding of the surgeon's need is used to formulate the anesthetic plan.

Hypertension is the prevalent medical problem observed in elderly patients undergoing shoulder surgery. Hypertensive patients will experience wider fluctuations in blood pressure intraoperatively than normotensive individuals, especially in the beach-chair position. Noxious stimuli will lead to exaggerated hypertensive responses. Conversely, since hypertensive patients tend to be intravascularly depleted, once general anesthesia is induced, hypotension may occur. In general, hypertensive patients should continue their antihypertensive therapy perioperatively. Some patients undergoing shoulder arthroscopy have rheumatoid arthritis. This disease could involve lungs, heart, and musculoskeletal system. Rheumatoid involvement of the cervical spine may result in limited neck range of motion, which interferes with airway management.

Shoulder arthroscopy can be performed with the patient in either the lateral decubitus or the sitting position. The sitting position offers an excellent intra-articular visualization for all types of arthroscopic shoulder procedures, less intraoperative blood loss, a lower incidence of traction neuropathy, and ease of conversion to an open approach if needed [1]. Position during shoulder arthroscopy may influence the choice of anesthetic plan since regional anesthesia is poorly tolerated in patients in the lateral decubitus position. A combination of regional and general anesthesia is recommended in the lateral decubitus, offering patients the advantages of long-acting local anesthetics (ropivacaine, levobupivacaine) in postoperative pain control and ensuring deep hypnosis for the uncomfortable position with ultrashort-acting modern medications (remifentanil, propofol, desflurane). However, like in the beach-chair position, unconsciousness due to general anesthesia could favor neurological and vascular lesions due to patient's positioning. Especially in the upright position, maintaining a safe position for the head during shoulder surgery under general anesthesia can be challenging. Reported complications attributed to an incorrect head position during surgery in the sitting asset have ranged in severity from cutaneous neurapraxias to complete midcervical quadriplegia [2–4]. In the lateral position cerebral hypoperfusion events are uncommon, and hypotension due to general anesthesia is less worrisome than in the upright one [5]. Shoulder surgery in the beach-chair position under general anesthesia is associated with significant reductions in cerebral oxygenation and subsequently with higher risk of neurological damage like visual loss and ischemic brain and/or spinal cord injury [6, 7].

Actually, regional anesthesia has several advantages for patients undergoing shoulder surgery: excellent anesthesia, reduction in both intraoperative and postoperative doses of opiates, delay of the onset of postoperative pain, a shortened postanesthesia stay, rapid discharge from the hospital, improved outcome, and increased patient satisfaction [8]. Furthermore, brachial plexus blockade is a cost-effective method for arthroscopic shoulder surgery [9].

During the last few years, ultrasonographic guidance has become a widely used technique for regional anesthesia, with safer procedures and faster onset time [10, 11]. Direct view of needle and anatomic structures reduces approximately to zero the incidence of intravascular injection, systemic

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local anesthetic toxicity, block failure, pneumothorax, and incidence of permanent nerve injury. Postoperative neurological symptoms are uncommon. Furthermore, ultrasound guidance allows shorter procedure time, fewer needle punctures, and reduced local anesthetic volume and postoperative pain when compared to neurostimulation technique for interscalene block [12–14].

Hypotensive Bradycardic Events During Shoulder Arthroscopy

When operating in the sitting position, one of the major concerns for anesthesiologists is cardiovascular instability during the shoulder procedure [15]. Hypotensive and bradycardic events (HBEs) have been reported in 13–28 % of patients. A HBE was defined, according to Liguori et al. [16], as a decrease in heart rate of >30 bpm in <5 min or any decrease <50 bpm and/or a systolic blood pressure decrease of >30 mmHg in <5 min or any decrease <90 mmHg. This event must have been accompanied by intraoperative treatment by the attending anesthesiologist. Light-headedness, nausea, and sweating were recorded but were not necessary in defining a HBE. Onset time of these transient but considerable events is 40–80 min after the plexus block or 25–45 min from the sitting position [17–19]. Most HBEs appear to be transient occurrences without complications such as brain hypoperfusion injury, but few severe cases of HBE have been reported, including asystolic cardiac arrest [20]. Underlying mechanisms responsible for the cardiovascular adverse effects are not completely understood. Several causes have been suggested: vasovagal syncope, carotid sinus hypersensitivity, orthostatic syncope, stellate ganglion block, drugs administration, and Bezold-Jarisch reflex [21]. The last one is a cardioinhibitory reflex. It is triggered by an empty hypercontractile ventricle, which causes activation of intramyocardial mechanoreceptors (C fibers) and results in a sudden reduction of sympathetic outflow, increasing vagal tone and thus causing bradycardia and hypotension. Some authors thought that the mechanism of the reflex was due to a reduced venous blood return induced by the beach-chair position and to a hyperexcitable heart caused by the βadrenergic effects of endogenous and exogenous epinephrine. These mechanisms result in an arterial vasodilation induced by activation of the parasympathetic nervous system and a subsequent vagally mediated bradycardia [17, 18]. Nevertheless, Seo et al. [22] suggested that the exogenous epinephrine does not augment the incidence of hypotensive-bradycardic events and that increased contractility due to epinephrine used in local anesthetic mixtures for nerve blocks is not certainly documented [23]. More studies are necessary to demonstrate the exact role of epinephrine. At the present time, there is no certain data in the literature to support the role of central volume depletion and hypercontractile empty ventricle for the activation of the Bezold-Jarisch reflex in the shoulder arthroscopy in the sitting position [24].

HBEs are only observed in awake conditions under isolated interscalene brachial plexus blockade. In awake settings, several stimuli may trigger the vasovagal reflex: fear, pain, prolonged sitting position, heat exposure, exertion, and coughing may lead to inhibition of the sympathetic system and to activation of the parasympathetic system, causing respectively hypotension and bradycardia. The afferent neural signals are probably derived from organ receptors, like cardiac mechanoreceptors, which respond to mechanical or chemical stimuli. Vagal triggering and sympathetic inhibition should be activated also by stimulation of carotid sinus. In the shoulder arthroscopy associated to brachial plexus block, sinus stimulation should be activated by denervation of sternocleidomastoid muscle due to block of fibers of C2–C4 spinal nerves. In fact, sternocleidomastoid proprioceptive information seems to have an important role in the regulation of baroreflex arc of the carotid sinus [25].

Stellate ganglion block occurs in 75 % of patients undergoing interscalene block with the Winnie's approach [26]. The symptoms after stellate ganglion block may be caused by baroreflex mechanism failure because of impairment of both cardiac sympathetic nerves and vagal afferents, including aortic depressor nerves [27].

Considering all these concerns, anesthesiologists should perform the brachial plexus block reducing the risk of local anesthetic diffusion toward medial structures, trying to avoid the block of the sympathetic chain and stellate ganglion, phrenic and laryngeal recurrent nerves, vagal nerve, and C2–C4 spinal nerves. Laterally directed needle [28] and lower local anesthetic volume could help obtain a safer technique. The patient's position should be very well executed avoiding abdominal and thoracic compression and maintaining a neutral and comfortable head position. Any neck stretching or stress could induce HBE directly stimulating carotid sinus, and uncomfortable sitting could augment patient's anxiety and start a vasovagal reflex. Furthermore, the anesthesiologist should consider the patient's medical history. Patient with a syncopal history and/or with a major anxious-depressive syndrome may not be candidate to isolated brachial plexus block. Although good sedation is necessary to perform both regional anesthesia and surgical procedure, the anesthesiologist should avoid opioids administration to patients, preferring the use of benzodiazepine. Opioids induce bradycardia and reduction in blood pressure. Fentanyl inhibits GABAergic transmission to cardiac vagal neurons in the nucleus ambiguous [29], inducing bradycardia. Song et al. demonstrated that the incidence of HBE is increased in patients receiving an intravenous bolus administration with 100 mcg of fentanyl (27.5 %) compared with the saline group (10 %) [30].

Therapy of HBE should be obtained with fluid and ephedrine administration. When a profound and dangerous reflex occurs, the first, and often the only, therapy is to lay down the patient.

Ultrasound-Guided Brachial Plexus Block

The brachial plexus is composed of the ventral roots of spinal nerves C5–T1, and sometimes it contains small fibers from C4 to T2. The roots exit the lateral foraminal spaces and pass between the anterior and the middle scalene muscles to innervate the upper limb. In the interscalene space, the roots coalesce to form the superior (C5–C6), middle (C7), and inferior (C8–T1) trunks that proceed laterally and inferiorly toward the space between the clavicle and first rib and then into the axilla. Several important branches are released from the brachial plexus at this level, including the suprascapular nerve, the dorsal scapular nerve, and the long thoracic nerve. When the trunks meet the first rib, they lie dorsolateral to the subclavian artery and superior to the rib. The brachial plexus and the artery are enclosed in a connective vagina. At the root level, the fascicles are surrounded by dura/perineurium. Within the perineurium there is little or no stroma, and care must be taken not to position the needle within the nerve root itself.

The anesthesiologists need a variety of supplies to perform ultrasound-guided brachial plexus block. Usually blocks are conducted in the preoperative holding area or in the operating room itself. Patients should have an intravenous catheter initiated, monitors placed, and supplement oxygen delivered prior to the block. The induction of regional anesthesia has risks that are similar to those of general anesthesia. For this reason, the practitioners should arrange a block cart containing both equipment for regional anesthesia and equipment and drugs for resuscitation [31]. Patients should be prepared with sedation and analgesia to remove anxiety and stress but still able to cooperate and without precluding feedback to the anesthesiologist.

The skin is prepared in sterile manner, and subcutaneous local anesthetic solution is injected at the site in which the block needle is to be inserted. The ultrasound probe is covered with a sterile, transparent membrane for single-shot blocks, while the anesthesiologist wears sterile gloves. It is controversial which is the best block needle to use. Most authors recommend the use of short-beveled needles, as it appears to be more difficult to penetrate the perineurium with this type of needle [32], although clinical outcome data is lacking. The choice of local anesthetic solution depends on the anesthesiologist's intent. Blocks intended for rapid onset and short duration may be conducted with mepivacaine or lidocaine, whereas longer-acting blocks will require ropivacaine or levobupivacaine. Mixture probably adds little to speed onset while significantly reducing the duration of long-acting agents. The ultrasound system should be portable with high definition and the probe linear and high frequency (10–13 MHz) to allow imaging of superficial nerves.

On performing the interscalene brachial plexus block under ultrasound guidance, the anesthesiologist should start identifying a reliable landmark at the base of the neck that consists of the subclavian artery and the brachial plexus, which lies dorsolateral to the artery and superior to the first rib. This requires placement of the ultrasound transducer in the supraclavicular fossa with a sagittal oblique orientation. At this level, the plexus appears as a cluster of grapes (Fig. 9.1). Keeping in the middle of the monitor the nerves, focusing on them, moving the probe slowly cranially, and titling the transducer more horizontally, the practitioner will appreciate the appearance of the anterior and middle scalene muscles with the plexus between them. Approximately at C6 level the anesthesiologist may display roots or trunks or a combination of the two aligning vertically like a traffic light. In 13 % of plexuses, variation from the typical relationship of the scalene muscles and nerve roots were present, the most common being the C5 nerve root running anterior to or directly through the anterior scalene muscle [33]. These anomalies may be responsible for occasional incomplete blocks. The sternocleidomastoid muscle at this level is visible like a triangular blanket lying superficial to the plexus and the scalene muscles.

The roots and trunks appear as hypoechoic nodules [34]. In fact, in most cases, the closer the nerve lies to the spine, the more likely it is to be hypoechoic. The hypoechoic aspect is due to cerebrospinal fluid that intersperses axons. At this level, nerves are surrounded by perineurium, which appears hyperechoic, but little or no stroma and fat is present. The peripheral nerves, instead, have hyperechoic stroma and fat outside the perineurium and assume the typical honeycomb aspect. In most patients, C5, C6, and C7 are easily visible in the same image. In some patients, also C7 and C8 are visible with this approach. The nerves lie at a mean depth of 5.5 mm from the skin surface [35]. Vascular structures of interest in this region are carotid artery and internal jugular vein, separated from the plexus by the anterior scalene muscle, the transverse cervical artery and associated vein, crossing transversely the interscalene space, and the external jugular vein, just beneath the skin. Fortunately, these vessels are seldom in the path of the blocking needle.

The brachial plexus can be approached in several ways. The most common is the interscalene block, although the posterior, the parascalene, and the supraclavicular techniques are well described. Performing the interscalene block, the patient should be positioned in the supine position with the head turned contralateral to the surgical site. The ultrasound transducer orientation should be transverse over the sternocleidomastoid muscle at the level of C6,

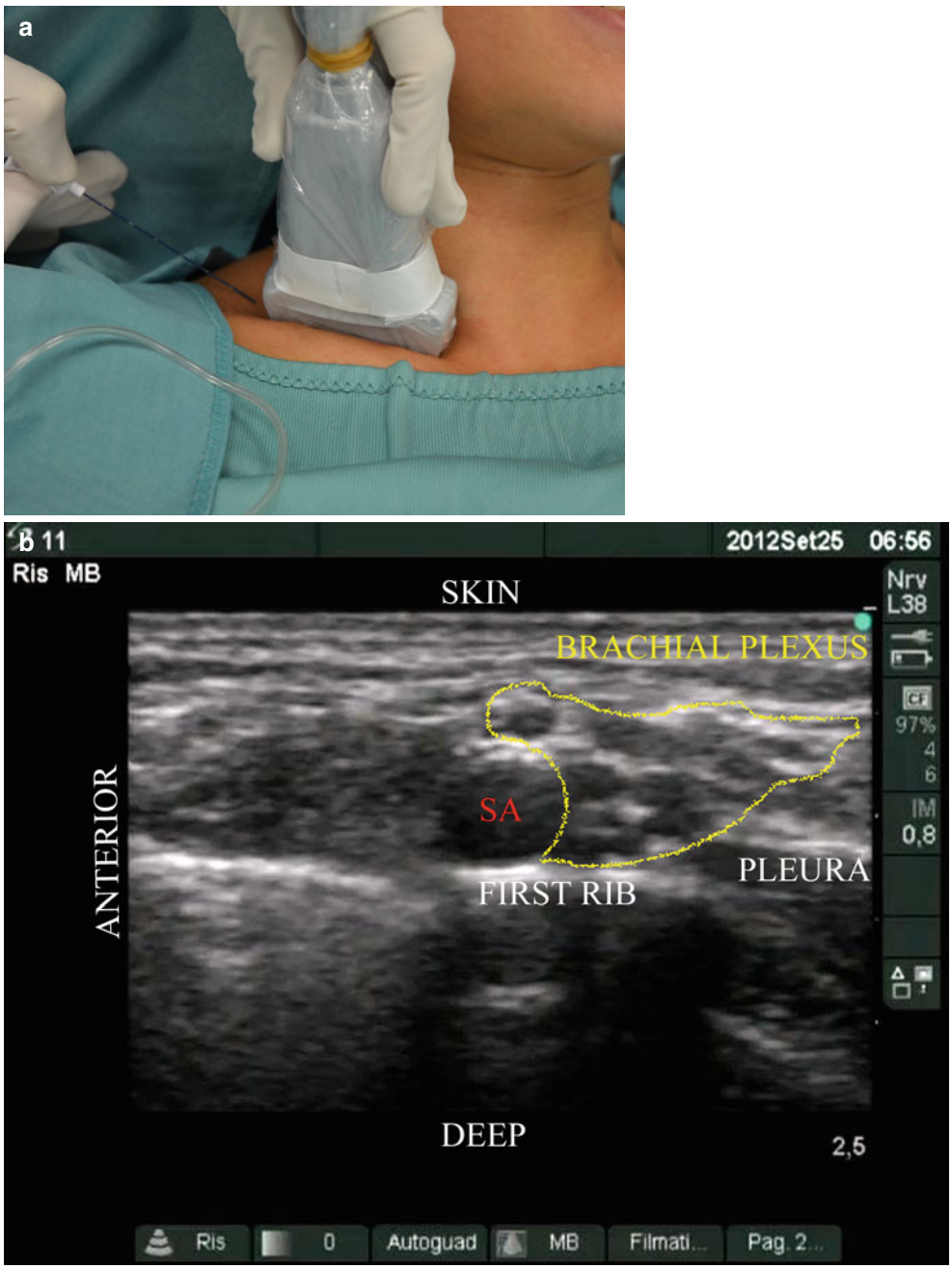

Fig. 9.1 (**a**) Placement of the ultrasound transducer in the supraclavicular fossa. (**b**) The brachial plexus at the supraclavicular level. *SA* subclavian artery

moving the probe until C5, C6, and C7 spine roots are well visible (Fig. 9.2). The ultrasound beam may be refracted as it passes through tissues. Fat globules below the skin, in the muscles, and around nerves serve as scattering and diffraction sites for the incident and reflected ultrasound beam and cause a spotted appearance in the image (this phenomenon is called "speculation"). For these reasons, obese patients can be very difficult to image. The image formed by ultrasounds is very sensitive to the angle of insonation, which is the angle of incidence of the beam relative to the nerve. Sometimes, changing the angle of insonation by only a few degrees can bring the nerve into focus. In obese patients, a light pressure of the tissue may improve the quality of the image. Once the optimal ultrasound image is obtained, it should be centered on the screen by sliding the probe on the patient's skin.

The needle may be inserted either posterior or anterior to the transducer, although a medial to lateral direction is preferable, for reasons exposed in the HBE paragraph. The in-plane approach offers the advantage of a perfect visualization of the entire needle and especially of the tip, and that is of fundamental importance to perform a successful and safe block (Fig. 9.3). After establishing the appropriate approach and the right image of the nerve root, the skin is anesthetized

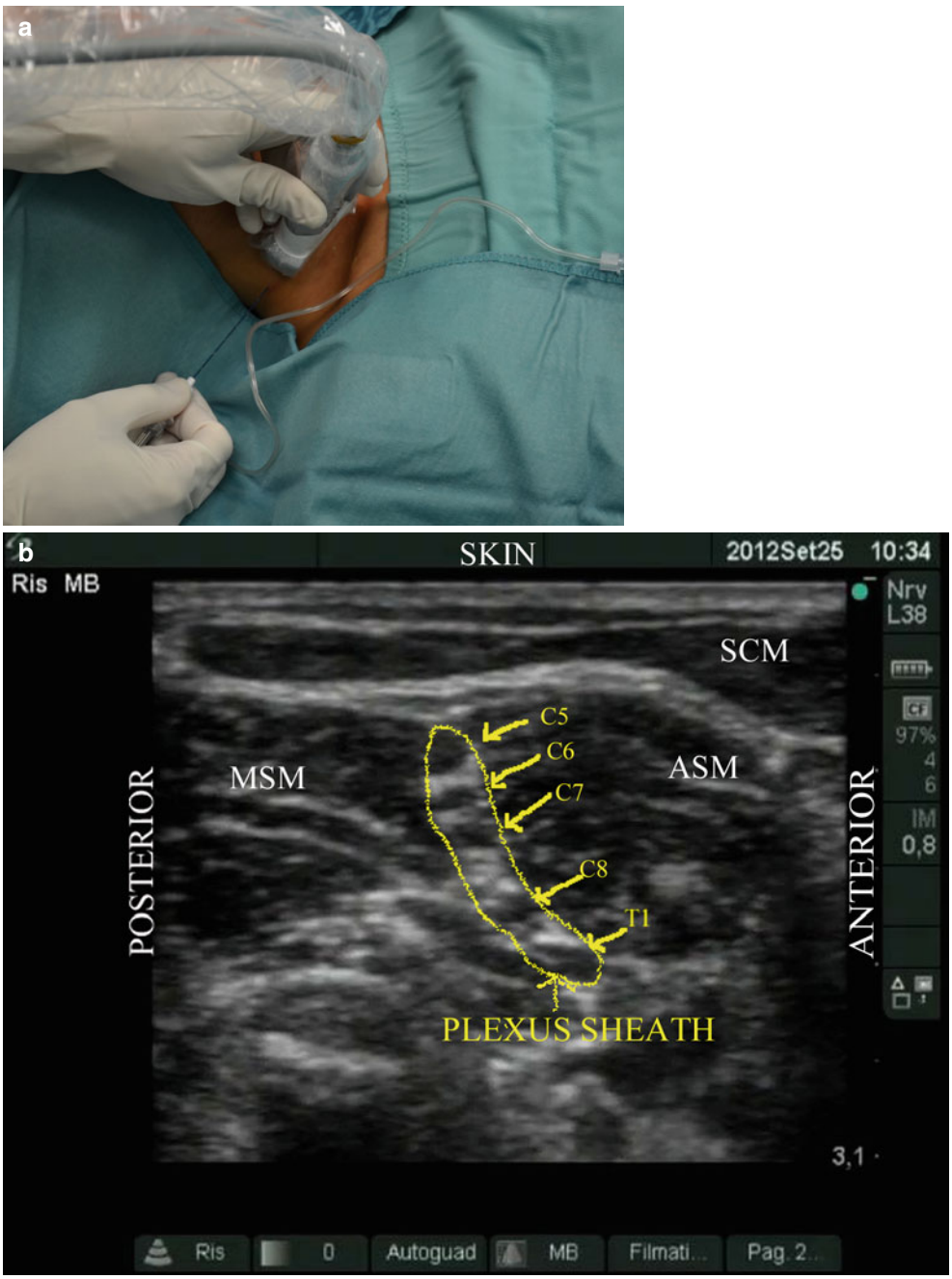

Fig. 9.2 (**a**) Placement of the ultrasound transducer for interscalene block. (**b**) The interscalene brachial plexus. *ASM* anterior scalene muscle, *MSM* middle scalene muscle, *SCM* sternocleidomastoid muscle. In this patient C8 and T1 are also visible

and the block needle introduced toward the goal. Little probe adjustments may be necessary to maintain a good visualization of both nerves and needle. Once the nerves are reached, the local anesthetic solution, usually 10–20 mL, is injected to surround the nerve elements. Local anesthetic should be injected between scalene muscles and brachial plexus sheath (peri-plexus), or within the brachial plexus sheath (intra-plexus) (Fig. 9.4). Intra-plexus block has longer duration then peri-plexus [36].

Complications in performing interscalene brachial plexus block under ultrasound guidance are uncommon. However, practitioners must be aware of the potential difficulties to prevent, recognize, and treat both light adverse effects and catastrophic complication. Vascular puncture, which may lead to systemic local anesthetics toxicity, can be easily avoided with a clear imaging and well needle-tip positioning. Aspiration before injection is always a necessary practice. Intravascular administration of local anesthetics may be responsible of neurological signs and symptoms, from mild tongue dysesthesias and speech difficulties to seizures and coma, depending on the plasmatic concentration of the anesthetic, and cardiovascular complications, including arrhythmias and cardiac arrest. The therapy consists in fluid and oxygen administration, supporting vital function, and

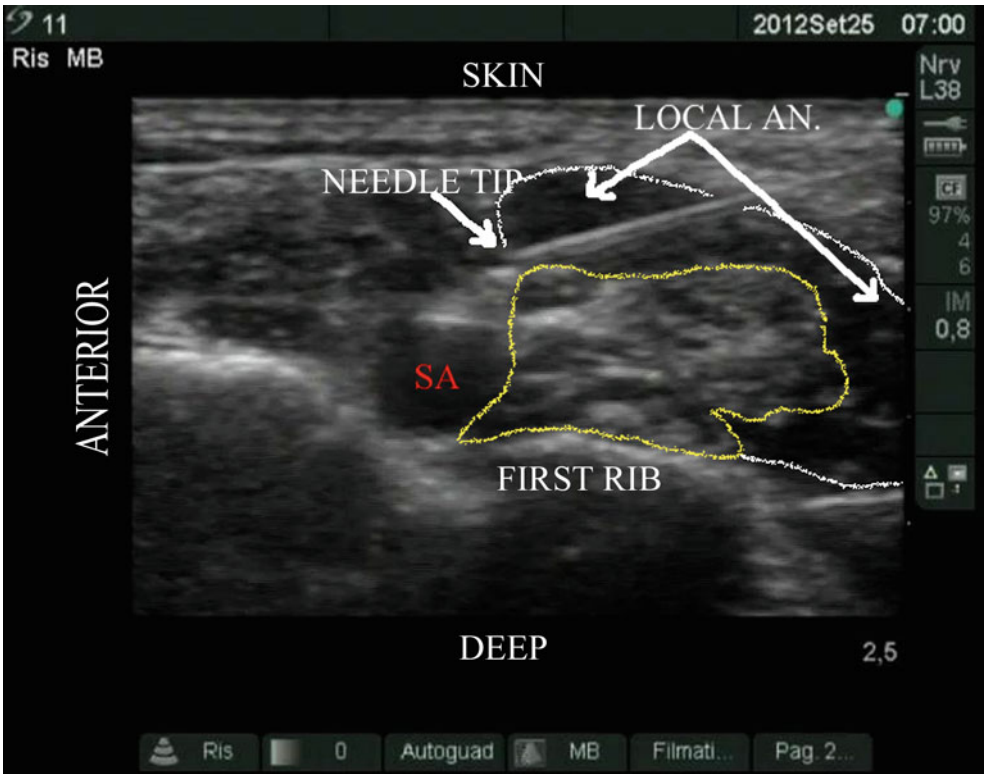

Fig. 9.3 Local anesthetic surrounding supraclavicular brachial plexus. With the in-plane technique, the needle and its point are well visible. *SA* subclavian artery

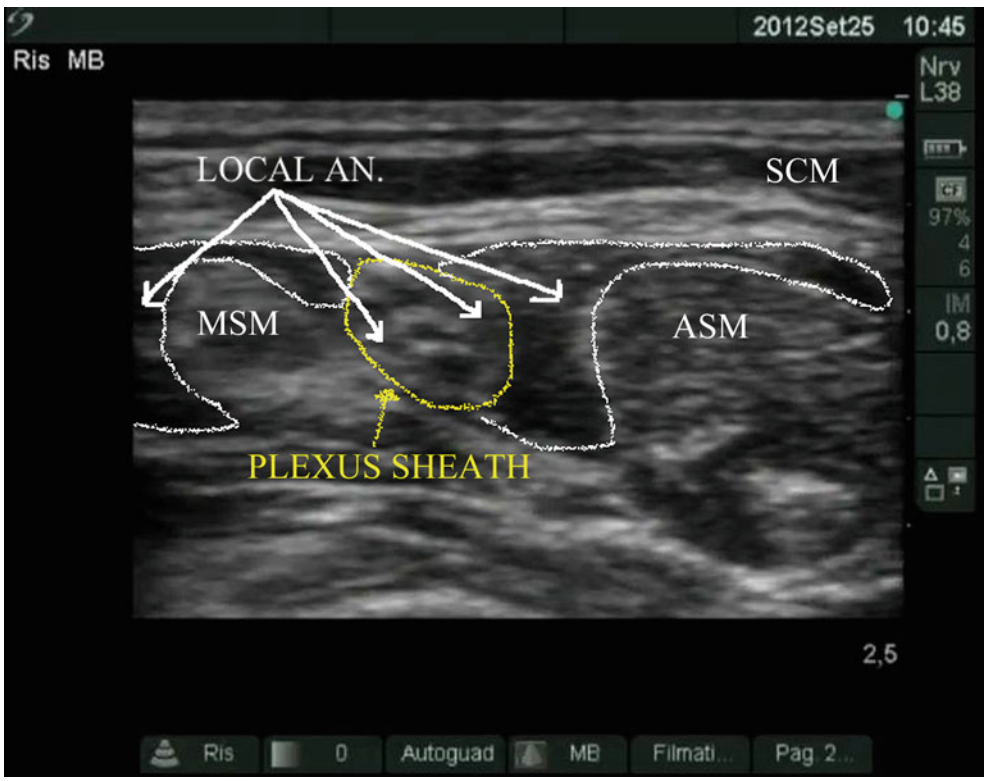

Fig. 9.4 Local anesthetic surrounding brachial plexus (peri-plexus) and intra-plexus (plexus swelling). *MSM* middle scalene muscle, *ASM* anterior scalene muscle, *SCM* sternocleidomastoid muscle

administration of antiepileptic drugs and lipid emulsion [37]. Lipid rescue has led to a reduction in fatalities associated with severe systemic toxicity. The underlying mechanisms of the lipid resuscitation may be a combination of a lipid catching and metabolic effect, but continued research is necessary for a better mechanistic understanding.

Phrenic nerve and laryngeal nerve block, generally, do not need of any intervention. However, being them common

side effects of the interscalene nerve block, the block is strictly contraindicated in patients affected by paralysis of contralateral phrenic nerve or laryngeal nerve, and, also, the anesthesiologist should carefully evaluate patients with respiratory diseases.

Like intravascular injection, pneumothorax is uncommon under a well-executed ultrasound-guided block. Extreme care must be adopted when the supraclavicular approach is chosen.

Catastrophic events were described in association with interscalene plexus block, like permanent loss of spinal cord function or total spinal anesthesia [38, 39]. Although these complications happened without ultrasound guidance and under general anesthesia, anesthesiologist must reflect on the opportunity to avoid intra-roots or intrafascicular injection as well as keep the needlepoint under strict vision.

After shoulder arthroscopy, patients may complain of neurological symptoms. It is improbable that they depend directly on a well-executed ultrasound-guided nerve block. In case of persistent paresthesia, dysesthesia, or pain not related to surgery after interscalene block, sulcus ulnaris syndrome, carpal tunnel syndrome, or complex regional pain syndrome should be excluded since specific treatment may be required [40].

Bleeding and Clear Surgical Field

The use of hypotensive anesthesia during orthopedic procedures performed on patients in the supine or decubitus position has been shown to be a safe and effective technique for reducing operative blood losses and helping maintain a clear surgical field [41, 42]. This said, in the beach-chair position, major complications have been reported under the use of hypotensive anesthesia. Pohl and Cullen reported four cases of ischemic brain and spinal cord injury after both open and arthroscopic procedures performed on patients in the sitting position under a hypotensive controlled anesthesia. Although the ideal perfusion pressure varies among patients, there exists a critical cerebral perfusion pressure below which cerebral blood flow will be compromised. Cerebral perfusion pressure is defined as mean arterial pressure less intracranial pressure (5–10 mmHg). When the patient is in the sitting position, there is a significant hydrostatic gradient between the brain and the site of blood pressure measurement, usually the contralateral arm to the surgical site. The difference is approximately 25–30 mmHg. When we measure a mean arterial systemic pressure of 80 mmHg, it could be not enough to guarantee an adequate cerebral perfusion pressure. Deliberate hypotension and errors in blood pressure reference point can be catastrophic [43]. Blood pressure should be maintained stable and near baseline parameters. Only hypertension should be mildly treated obtaining no more than 20 % decrement of preoperative resting values.

Since the 90s, surgeons investigated characteristics of fluid irrigation to augment visualization during shoulder arthroscopy and to prevent intraoperative and postoperative complications. They evaluated the temperature of the irrigation fluid and its influence on core body temperature, the way to administer it, and the possibility of diluting it with some vasoactive agents. The use of warm irrigation fluid during arthroscopic shoulder surgery decreases perioperative hypothermia, especially in elderly patients [44]. Surely, the thermic homeostasis is important to preserve patient's comfort and stability and favor hemostatic mechanisms. Anesthesiologists could contribute to this aim warming saline solutions and using forced air warming. In 2001 Jensen et al. [45] randomized 44 patients that underwent routine arthroscopic shoulder surgery into a prospective, double-blinded, placebo-controlled study to determine whether diluted epinephrine saline irrigation (0.33 mg/L) significantly reduces intraoperative bleeding. A pressure-controlled pump delivered the irrigation fluid. The study also evaluates potential adverse cardiovascular reactions of adding epinephrine to the irrigation fluid. Intraoperatively, intra-articular bleeding was estimated by multiplying the total volume of the irrigation fluid used by the hemoglobin concentration of the irrigation fluid. The clarity of visualization during the procedure was assessed asking the surgeon to quantify it by a visual analogy scale. In the treatment group the intraoperative bleeding was significantly reduced ($P=0.008$), and the clarity of the visual field was significantly better ($P=0.0007$) compared to the control group. No cardiovascular adverse events were observed administrating the intra-articular epinephrine.

Postoperative Pain Control

Arthroscopic shoulder surgery performed under general anesthesia is associated with severe postoperative pain requiring large doses of opioids. Nausea, vomiting, sedation, and lack of complete pain control are often associated with intravenous opioids analgesia [46, 47]. The interscalene brachial plexus block offers a valid postoperative analgesia, reducing both the request of rescue medications and consequently side effects. In the single-shot setting, the duration of analgesia is up to 13 h [48]. Addiction of adjuvants to the local anesthetic, like buprenorphine or tramadol, may prolong analgesic time up to more hours [49, 50]. Continuous peripheral nerve blocks provide optimal analgesia, prolong brachial plexus local anesthetic delivery in the outpatient setting, have minimal side effects, and avoid premature regression of an analgesic block. Furthermore, an improvement in patients' health-related quality of life and outcome has been demonstrated [51]. Continuous intra-articular infusion of local anesthetics should be avoided because of the risks of chondrotoxicity. Continuous

intra-articular infusion of bupivacaine with or without epinephrine led to significant histopathologic and metabolic changes in articular cartilage [52].

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Portal Placement and Related Anatomy

10

Andrea Grasso, Domenico A. Santagada, and Matteo Salvatore

Shoulder arthroscopy is a minimally invasive technique that allows optimal visualization of the glenohumeral joint, subacromial, and coracoid space. A long learning curve is required to achieve a safe and effective surgery that passes through a deep knowledge of the anatomy and the pathology of the shoulder.

The only way to have a correct approach to the joint, as in every arthroscopic procedure, is the adequate knowledge and the correct placement of the portals. This will improve intra-articular and subacromial visualization and also allow the right angle of approach and the range of motion required by the surgical procedure.

The proximity of vascular and nervous structures confers a high risk level to the establishment of arthroscopic portals. The literature reports a low prevalence of direct arterial or nervous injuries, such as transient neurapraxias involving the musculocutaneous, ulnar, radial, axillary, and median nerves [1], but a high number of venous injury, especially to the cephalic vein [2].

This chapter is focused on this preliminary step of the shoulder arthroscopy by describing fundamental portals used to perform diagnostic and operative shoulder arthroscopy. Additional portals used for specific procedures are described in detail in the dedicated chapters of this book.

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Landmarks

Lining the bony landmarks is an essential first step before starting shoulder arthroscopy. It helps the surgeon to establish additional portals easily even if soft tissues are swollen by the irrigation.

First, the scapular spine, the posterolateral, and the anterolateral corners are identified and palpated with the first three fingers and then marked with a dermographic pen. Then, the anterior and posterior profiles of the distal clavicle are identified, and the acromioclavicular joint is drawn. Finally, the coracoid process is identified, and the coracoacromial ligament is drawn between the coracoid process and the anterolateral corner of the acromion (Fig. 10.1).

An intra-articular injection of 20 ml of a diluted solution of epinephrine/norepinephrine (1:80,000) performed with a spinal needle before surgery is a useful tool to facilitate joint access and reduce bleeding during the procedure The needle is inserted through the soft spot of the shoulder, which is located 2 cm inferiorly and 1–2 cm medially to the posterolateral edge of the acromion (Fig. 10.2).

Glenohumeral Joint Portals

Posterior Portal

The posterior portal (or "soft-spot" portal) is commonly used as viewing portal at the beginning of almost all the shoulder arthroscopic procedures. It is performed blindly through a point located 1–2 cm medial and 2 cm inferior to the posterolateral corner of the acromion, by directing the blunt trocar of the arthroscopic sheath towards the coracoid process as described by Andrews et al. [3]. This portal is useful also to introduce the spinal needle for the initial intra-articular distension of the joint. Wolf [4] described a central posterior portal whose only difference with the "soft-spot" portal is the skin incision located 2–3 cm distal to the posterolateral

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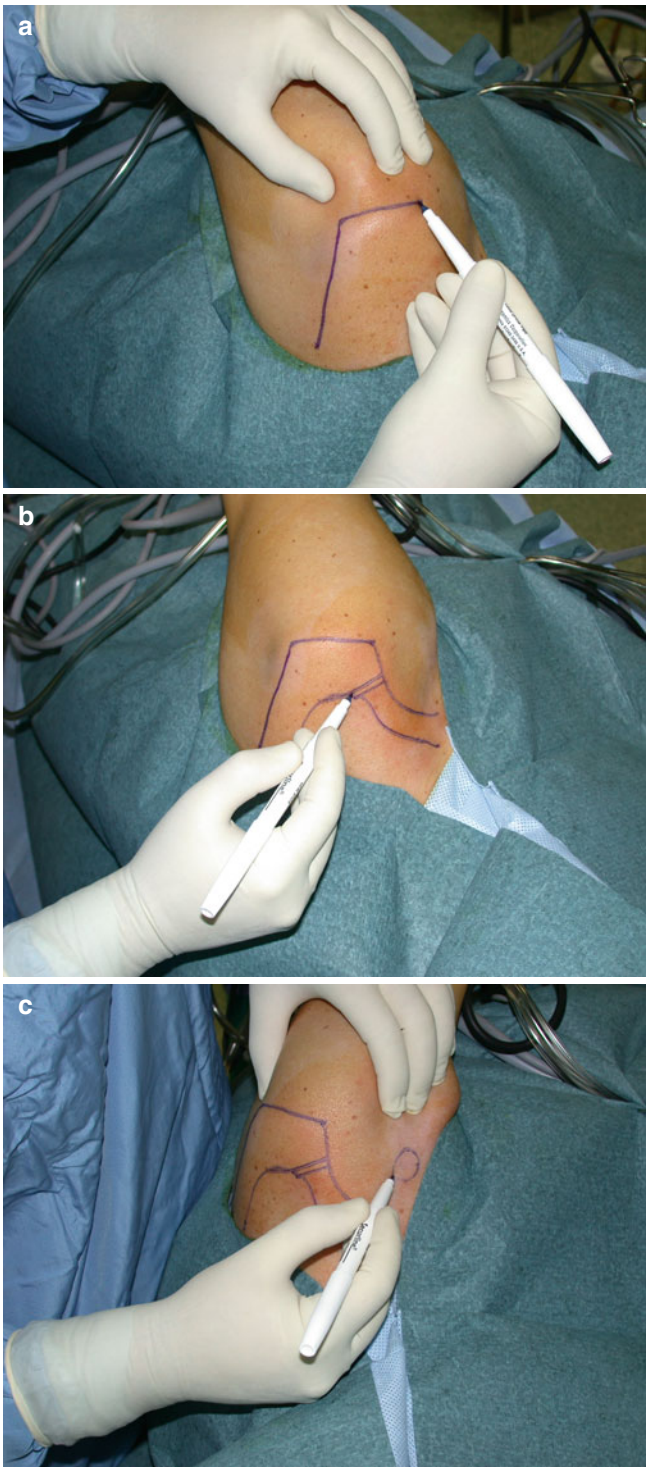

Fig. 10.1 Bony landmarks in shoulder arthroscopy. (**a**) The scapular spine, the posterolateral, and anterolateral angle and the lateral border of the acromion are drawn. (**b**) The acromioclavicular joint is identified between the acromion and the distal end of the clavicle. (**c**) The coracoid process is identified just below the distal end of the clavicle

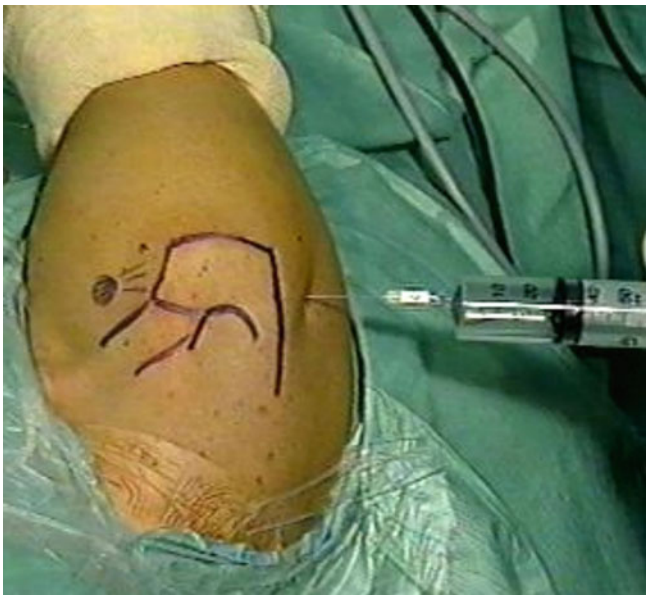

Fig. 10.2 Intra-articular distension from the "soft spot"

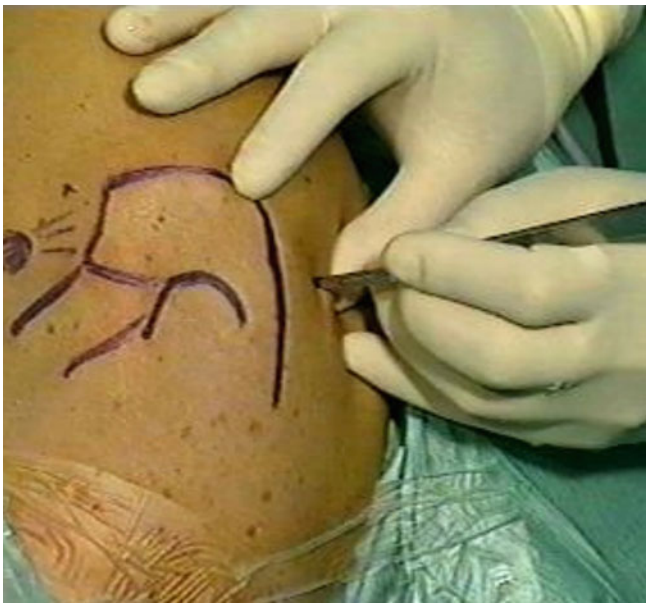

Fig. 10.3 Posterior central portal is established 2 cm medial and 2–3 cm distal to the posterolateral corner of the acromion

corner of the acromion. It corresponds to the level of the posterior joint line so it can pass the joint parallel to the glenoid surface (Fig. 10.3). If the patient is in beach-chair position, slight abduction and lateral traction can ease the establishment of this portal by tensioning of the posterior capsule.

The axillary nerve inferiorly and the suprascapular artery and nerve medially are potentially at risk when this portal is established. They are situated at a mean distance of 49.3 and 29 mm from the portal, respectively [5].

After the introduction of the scope into the joint, it is possible to perform an air-distended diagnostic arthroscopy. Keeping inflow closed the joint is distended with 30 cc of air through the trocar. Once this phase is completed, the joint is

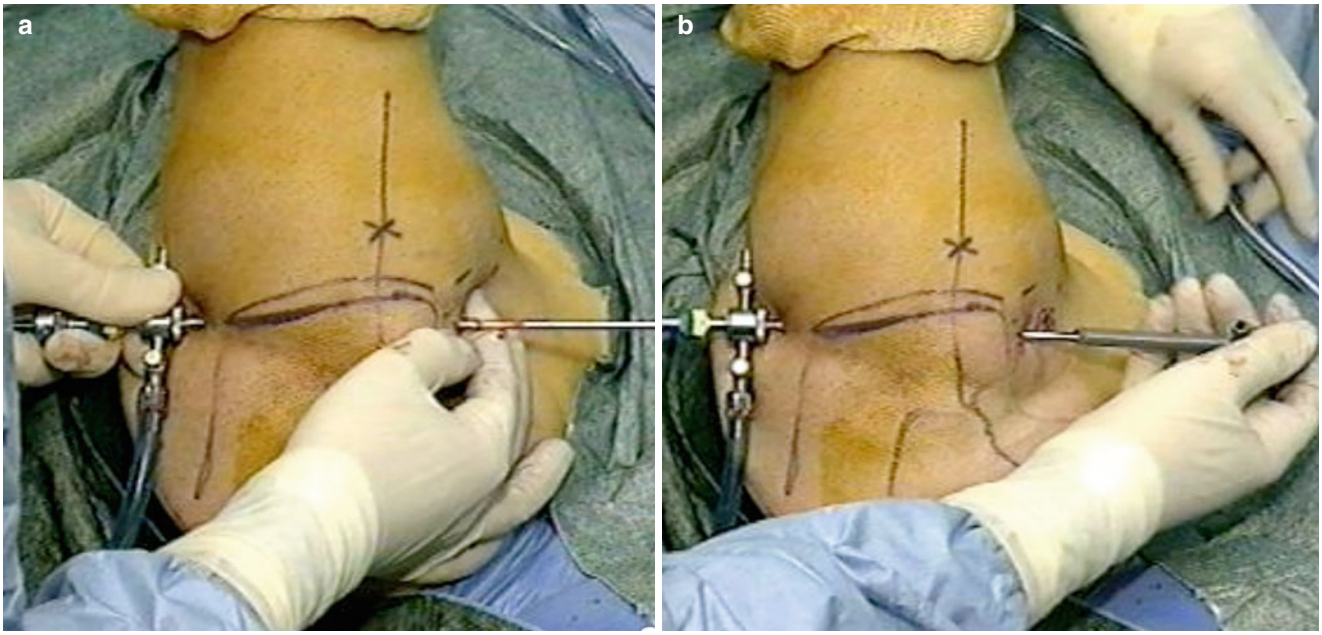

Fig. 10.4 Anterior central portal. (**a**) The portal is located with the inside-out-technique using a Wissinger rod. (**b**) A plastic cannula is introduced over the Wissinger rod

irrigated through the arthroscopic sheath and the anterior portals can be established.

Anterior Portals

Anterior Central Portal

The anterior central portal, or Matthews' portal [6], can be established using an inside-out or an outside-in technique.

Using the inside-out technique, the portal is established using a Wissinger rod inserted through the arthroscopic sheath; it passes through the "safe triangle" delimited by the medial margin of the long head of the biceps tendon, the superior margin of the subscapularis tendon, and the humeral head, pointing towards the coracoid process. Once the safe triangle is exceeded, the Wissinger rod is directed anteriorly pushing upward the skin, just laterally to the coracoid. A skin incision is performed over the tip of the Wissinger rod, which is used as guide to introduce a plastic cannula into the joint (Fig. 10.4).

In the outside-in technique, the portal is marked by using a spinal needle (20G) introduced from a skin point laterally to the coracoid process into the joint, going through a triangle limited by the humeral head laterally, the glenoid rim medially, and the long head of the biceps tendon superiorly, while remaining above the subscapularis tendon (Fig. 10.5).

This portal is particularly useful during procedures that involve the anterosuperior part of the glenohumeral joint (long head of the biceps tenotomy/tenodesis, subscapularis tendon repair, anterosuperior capsulolabral repair).

The axillary and the musculocutaneous nerve, the axillary artery, and the cephalic vein are potentially at risk when this portal is established. They are situated at a mean distance of 31, 21.3, 33 and 17 mm from the portal, respectively [5].

Anteroinferior Portal

The anteroinferior portal, or Wolf's portal [4], can be established using an inside-out technique or an outside-in technique. For a correct execution of this portal, it is advisable to use the outside-in technique, which allows a direct visualization of the superior margin of the subscapularis tendon, thus avoiding its violation.

The cutaneous incision is made just laterally and inferiorly to the coracoid process. A spinal needle (20G) is inserted as mentioned above, passing through the safe triangle just upon the superior margin of the subscapularis tendon (Fig. 10.6).

This portal is normally used for suture anchor placement and suture hook insertion during anteroinferior capsulolabral repair.

The axillary and the musculocutaneous nerve, the axillary artery, and the cephalic vein are potentially at risk when this portal is established. They are situated at a mean distance of 14, 19, 42 and 14 mm from the portal, respectively [5].

Anterosuperior Portal

Wolf also described an anterosuperior portal [4] created following an outside-in technique and located between the coracoid and the acromion. It enters the joint just anterior to the long head of the biceps tendon (Fig. 10.7). This portal is used

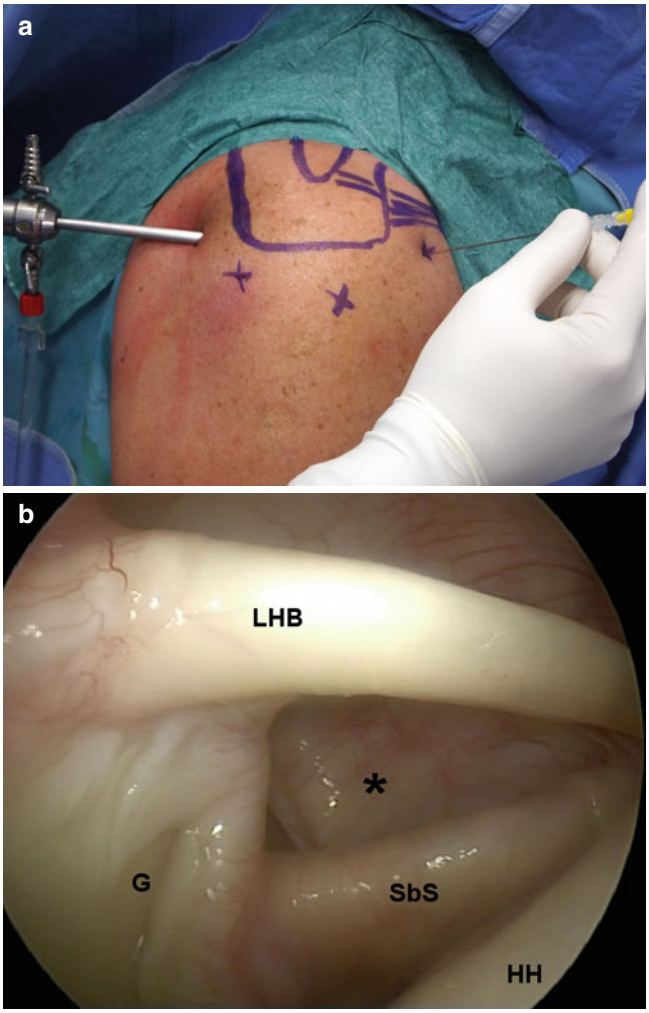

Fig. 10.5 Anterior central portal. (**a**) The portal is located with the outside-in technique. (**b**) The entry point of the spinal needle (*asterisk*) should be included in the triangle limited by the humeral head (*HH*) laterally, the glenoid rim (*G*) medially, and the long head of the biceps (*LHB*) tendon superiorly while remaining above the subscapularis tendon (*SbS*)

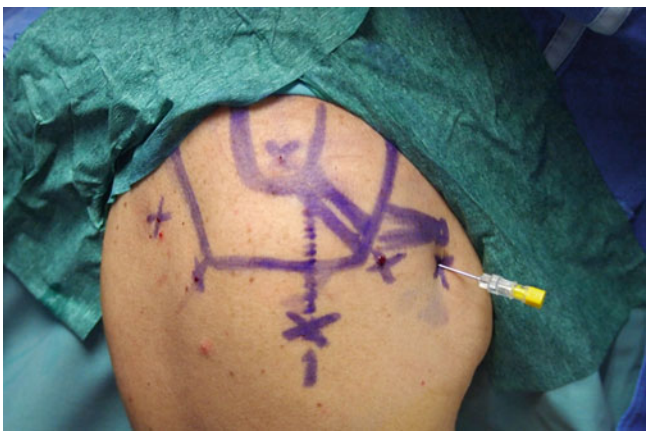

Fig. 10.6 The spinal needle is used to locate Wolf's anteroinferior portal with the outside-in technique

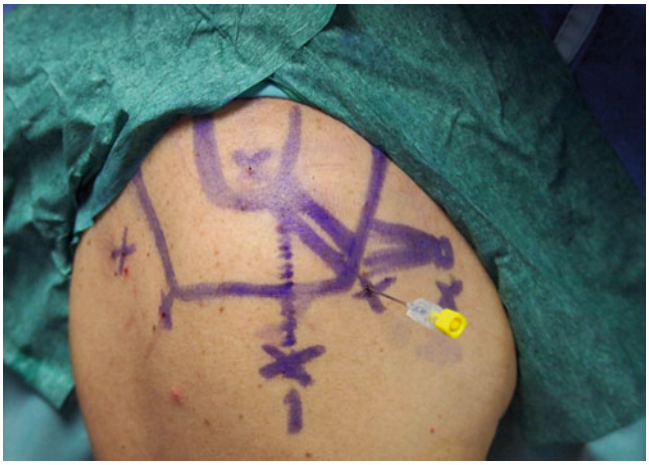

Fig. 10.7 The spinal needle is used to locate Wolf's anterosuperior portal with the outside-in technique

as outflow and/or retrieval portal during Bankart repair, as well as viewing portal during anterior or posterior capsular repair.

Five O'Clock Portal

Davidson and Tibone [7] described the "5 o'clock portal" to allow the approach of the inferior glenoid rim and facilitate the insertion of the suture anchors at a right angle during Bankart repair. The portal is established using an inside-out technique: the scope is inserted through the posterior portal and pointed at 5 o'clock position (for a right shoulder) of the glenoid rim; it is then replaced by a Wissinger rod passing through the anterior capsule. The humerus is maximally adducted while establishing the portal to medialize neurovascular structures.

The 5 o'clock portal is considered the most unsafe approach because of its close position with the anterior neurovascular structures; in fact the axillary nerve and artery are situated approximately at 13 and 15 mm from the portal, while the cephalic vein at 17 mm [5].

Superior Portal

The superior portal (or Neviaser's portal), also called the "supraclavicular fossa portal," is located in the supraspinatus fossa, between the clavicle (anteriorly), the scapular spine (posteriorly), and the medial border of the acromion (laterally) [8] (Fig. 10.8).

This portal was initially described as an additional inflow portal but can also be used during suture anchor placement for SLAP repair [9]. At present, this portal is no more commonly used.

The mean distance to the scapular nerve and suprascapular artery is 24 and 26 mm, respectively [4].

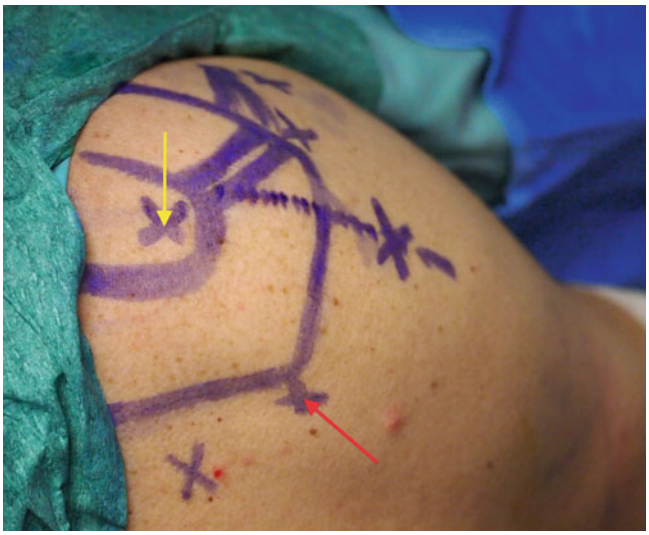

Fig. 10.8 Location of Neviaser's superior portal (*yellow arrow*) and the port of Wilmington (*red arrow*)

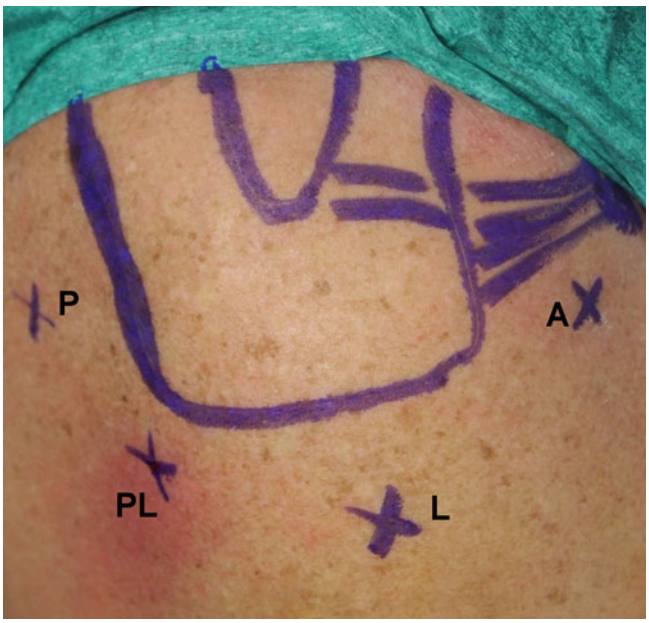

Fig. 10.9 Cutaneous landmarks of the subacromial space portals. *P* posterior portal, *PL* posterolateral portal, *A* anterior portal, *L* lateral portal

Lateral Portals

Port of Wilmington

This portal is made under direct visualization of the intra-articular side of the joint, with the scope normally positioned anteriorly. A spinal needle is inserted 1 cm laterally and 1 cm inferiorly the posterolateral corner of the acromion, passing through the posterolateral part of the cuff (Fig. 10.8).

The main advantage of this portal consists in its direct approach to the posterosuperior labrum; therefore, it is considered useful to repair posterior SLAP lesions [10]. The main disadvantage is represented by the violation of the cuff; to avoid tendon rupture, its incision should be performed parallel to the tendon fibers, for no more than 5-mm length.

The axillary nerve is located at a mean distance of 55 mm from this portal [4].

Superolateral Portal

The superolateral portal, described by Laurencin et al. [11], is located just lateral to the anterolateral margin of the acromion, and it is helpful in the anterior stabilization procedures.

The axillary nerve is located at a mean distance of 70 mm from this portal [4].

Subacromial Space Portals

Posterior Portals

Posterior Central Portal

This portal is located in the "soft spot" as well as the posterior portal for the glenohumeral joint access (Fig. 10.9). After inserting the arthroscopic sheath with the blunt trocar, to proceed to the subacromial space it is important to move cranially just deep to the subcutaneous tissue. The instrument is used to palpate the posterior margin of the acromion and is progressively introduced under the acromion into the subacromial space; the orientation and the shape of the acromion are identified during insertion of the instrument by manual palpation of its posterolateral and anterolateral margin.

The mean distance between this portal and the axillary nerve is 49 mm; the suprascapular nerve and the suprascapular artery pass at a mean distance of 29 mm inferiorly and 27 mm medially, respectively [4].

Posterolateral Portal

As well as for the posterior portal, this one is used to introduce the scope during rotator cuff repair. According to Ellman [12], this portal is situated about 2 cm below the lateral margin of the acromion in the prolongation of its posterior edge (Fig. 10.9). The experience can show that through this portal, it is possible to have a superior mobility and a better view of the acromioclavicular joint and rotator cuff respect the one given by the direct posterior central portal. It is normally used during the passage from the intra-articular to the subacromial space during shoulder arthroscopy and is often performed together with the anterior portal (see below for the description of the inside-out technique).

This portal is located in a relatively safe zone. In fact, the nearest neurovascular structure is the axillary nerve, which passes at a mean distance of 56 mm away from it [4].

Anterior Portal

The anterior portal is positioned between the coracoid and the anterolateral border of the acromion. It must be placed just laterally to the coracoacromial ligament to avoid its violation that results in loss of mobility and potential risk of bleeding (Fig. 10.9). Usually, it is performed with an inside-out technique, using the arthroscopic sheath with its blunt trocar from the posterolateral portal as a guide to introduce an outflow 6 mm cannula directly into the subacromial space (Fig. 10.10).

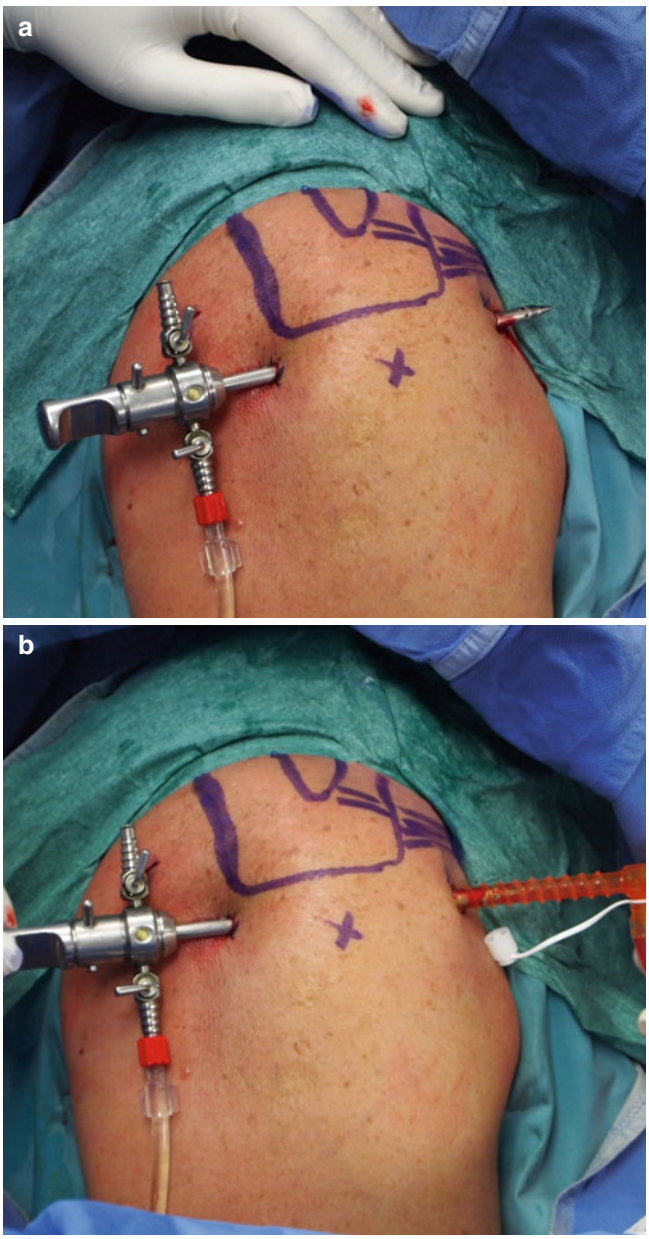

Fig. 10.10 Anterior portal. (**a**) The portal is located with the inside-out-technique using the blunt trocar of the arthroscopic sheath introduced through the posterolateral portal. (**b**) A plastic cannula is introduced over the arthroscopic sheath through the anterior portal

The mean distance with the axillary nerve and artery is 54 and 53 mm, respectively; the cephalic vein lies within 39-mm mean distance [4].

Lateral Portals

Lateral Portal

This portal is located 2–3 cm inferiorly to the lateral border of the acromion, in the prolongation of the posterior margin of the acromioclavicular (AC) joint (Fig. 10.9). It is better to perform this operative/viewing portal under direct visualization with the scope in a posterior portal, and a spinal needle inserted laterally and inferiorly to the lateral margin of the acromion. If the portal is correctly centered on the middle part of the rotator cuff tear or on the central part of the acromion, it will be easier to perform the next surgical step, such as rotator cuff repair or acromioplasty (Fig. 10.11).

The axillary nerve passes more than 5 cm inferiorly to the lateral border of the acromion.

Anterolateral Portal

This second Ellman's portal is localized 2–3 cm inferiorly to the lateral border of the acromion, in the prolongation of its anterior edge [12]. This portal allows direct access to the undersurface of the acromion and is commonly used to perform arthroscopic acromioplasty. It is established by using an outside-in technique with a spinal needle as guide under direct visualization from a posterior portal.

The anterolateral portal is particularly safe as the mean distance with the axillary nerve is 70 mm [4].

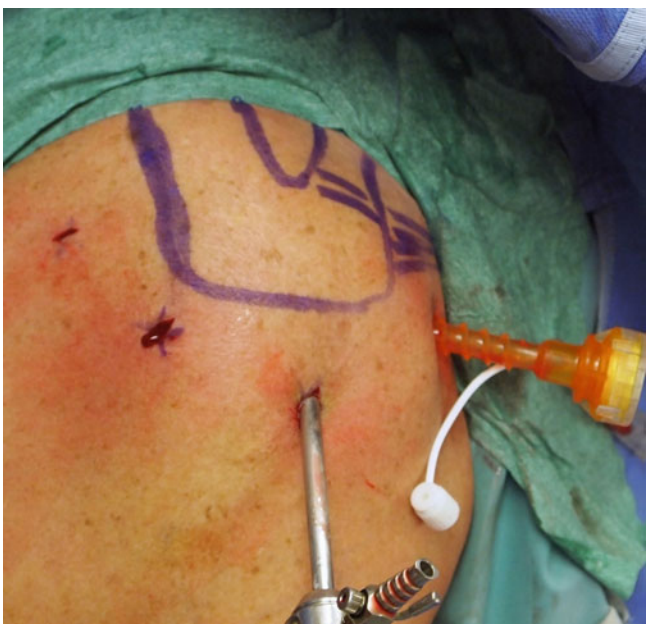

Fig. 10.11 The lateral portal is used as viewing portal during subacromial space procedures

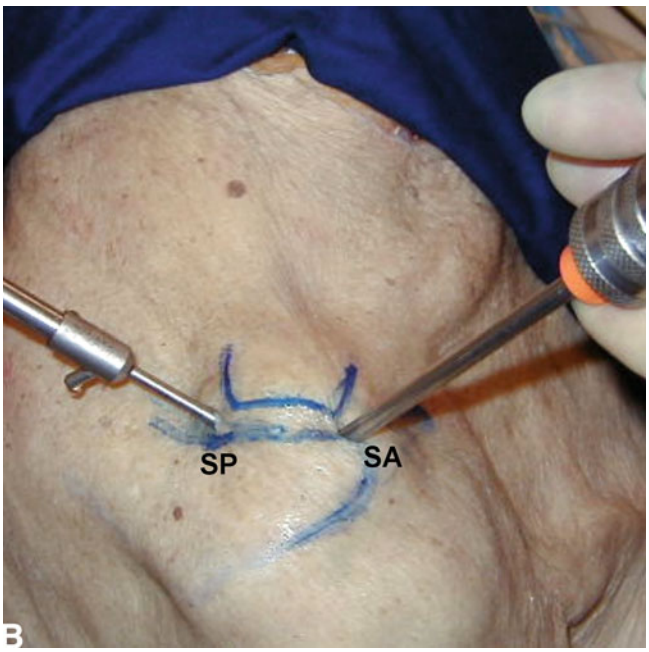

Fig. 10.12 Direct portals to the AC joint (*SA* superoanterior, *SP* superoposterior)

Acromioclavicular Joint Portals

The AC joint can be managed with a direct or indirect approach.

Direct Approach

This technique requires portals with which directly approach the AC joint. Due to the small size of the joint and to its anatomical conformation, confortable instruments are required: a 2.7-mm scope and a 2.0-mm burr. It is often used to treat AC joint disease, which leads to an enlargement of the articular space, such as in the advanced osteolysis of the distal end of the clavicle.

The direct approach needs two portals: the superoanterior and the superoposterior (Fig. 10.12).

Superoanterior Portal

After having identified the joint space line between the distal end of the clavicle and the medial margin of the acromion, the approach is performed 7.5 mm anteriorly to the joint line of the AC joint.

Superoposterior Portal

After having identified the joint space line between the distal end of the clavicle and the medial margin of the acromion, the approach is performed 7.5 mm posteriorly to the joint line of the AC joint.

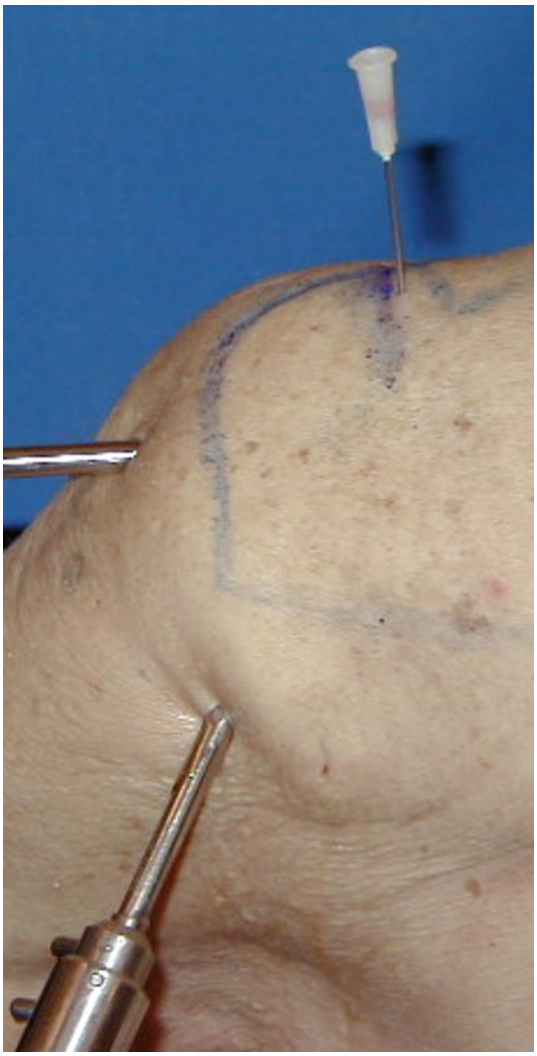

Fig. 10.13 A spinal needle is used to locate the superior direct portal to the AC joint

Indirect Approach

This approach needs the same portals seen for the subacromial space. Normally, it is used not only to treat diseases that involve the AC joint but also to perform the other subacromial procedures (i.e., rotator cuff repair, acromioplasty). Subacromial impingement due to spurring of the distal clavicle is easily addressed by a direct approach.

Sometimes, a superior direct portal can be used as additional portal of the indirect approach, to directly evaluate the articular space (Fig. 10.13). This portal is performed only when really required because it violates the superior AC joint capsular ligament, which represents the main superior stabilizer of the AC joint together with the trapezius fibers.

Author's Preferred Technique

Instability, subacromial impingement with or without rotator cuff pathology, and AC joint disease often require different approaches. We want to give an indication on how normally we approach each group of these shoulder conditions:

- Glenohumeral Instability
  - Posterior portal
  - Anterior central portal
  - Anterosuperior portal
- Subacromial Diseases
  - Posterolateral portal
  - Anterior portal
  - Lateral portal
- AC Joint Diseases
  - Indirect technique (arthrosis, soft osteolysis)
  - Direct technique (severe osteolysis)

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Diagnostic Shoulder Arthroscopy

11

Antonio E.G.C. Cartucho

Introduction

Shoulder arthroscopy turned, in the last 20 years, in a fundamental diagnostic and treatment tool. Since the first description of subacromial decompression by Ellman [1], there has been a constant evolution driven by basic science, biomechanics, and engineering that turned possible that nowadays most of the instability, rotator cuff, and part of the articular degenerative problems of the shoulder girdle are treated using arthroscopy.

Arthroscopy should be looked as a tool to deal with certain problems. This means that it should be used in the right indication, with the correct technique and taking into account the "state of the art" for the treatment of the pathology. Behind the technical gesture lies the most important part of arthroscopy. The ability of the surgeon to recognize what is not normal, what is a normal variance, if the structural damage observed is the primary cause of the patient complaints, and finally the decisions of the correct way to deal with the problem, allied with a good technique, are altogether the keys of success.

In this chapter, we will explain the technical aspects together with the pathologic and clinical relevant issues.

Operating Room and Patient Positioning

The rational use of the space available at the operating room with a correct positioning of the operating table is vital. This position and the distribution of other equipment like the camera, the pump, the radiofrequency device, and the screen are dependent of the patient positioning.

The "beach chair position" (BCP) and "lateral decubitus position" (LDP) are equally used with no definitive advantage of one over the other. Nevertheless, it is consensual that BCP permits an easier turn to open surgery and that LDP permits a better access and visualization on instability procedures.

In BCP the patient is seated with a 45–80º back inclination. An operating table with a segmented back, permitting to uncover the back of the shoulder to be operated, is an important asset. If not available, the patient's arm is pulled to the extremity of the table, and a small pillow can be used under the shoulder blade in order to slightly rotate the shoulder; otherwise, the manipulation of the arthroscope will be disturbed by the operating table specially when the surgeon tries to look with the arthroscope to the lateral side of the shoulder.

When using LDP, a standard operating table can be used. The patient should lay on the side with the support of a vacuum cushion or pubic and sacrum supports. A 3 kg traction device is used in order to maintain the arm at 70º of abduction and 20º of forward flexion. This position may be changed in order to inspect the subacromial bursa.

In both operating positions, the necessary equipment is placed opposite to the surgical team with the ventilator and the anesthesiologist at the head of the patient.

Arthroscopic Instruments

For shoulder arthroscopy, the necessary basic instruments are similar to the ones used for the knee:

- A 4.5 mm arthroscopic sheath with at least one fluid entrance and ideally with another for aspiration control
- A 30º optical device
- A cold light system
- A camera
- A probe (preferably long)

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- A monitor
- A fluid pump
- A digital recording system
- A mechanic cutting device (shaver)
- A radio frequency device
- An operating cannula

The recording system although not essential is important to review cases with a bad clinical outcome, to use the images for teaching purposes, and last but not least as proof in case of litigation.

Although considered unnecessary by a few surgeons, the fluid pump is the only way to know the exact pressure of fluid inside the joint. This should be kept between 40 and 50 mmHg. The pump also permits to perform short periods of hyperpressure for further distension or hemorrhagic control.

General Principles and Portals

With the patient in the chosen position, drawing of the osseous landmarks should be done. From the spine of the scapula, the external border of the acromion with its posterolateral and anterolateral corners should be carefully marked. The anterior and posterior margins of the clavicle and acromioclavicular joint must be outlined. Finally, the coracoid process is identified (Fig. 11.1). These landmarks will help the surgeon in triangulation techniques and to insert the portals in the proper position. Nevertheless, it should be kept in mind that during the procedure, the skin marks can change as far as 2 cm from the initial mark. This fact is due to soft tissue distension which is particularly evident in extra-articular procedures. A less correct surgical technique with multiple "failed" accesses to the joint also contributes to fluid extravasation turning the surgical procedure more and more difficult.

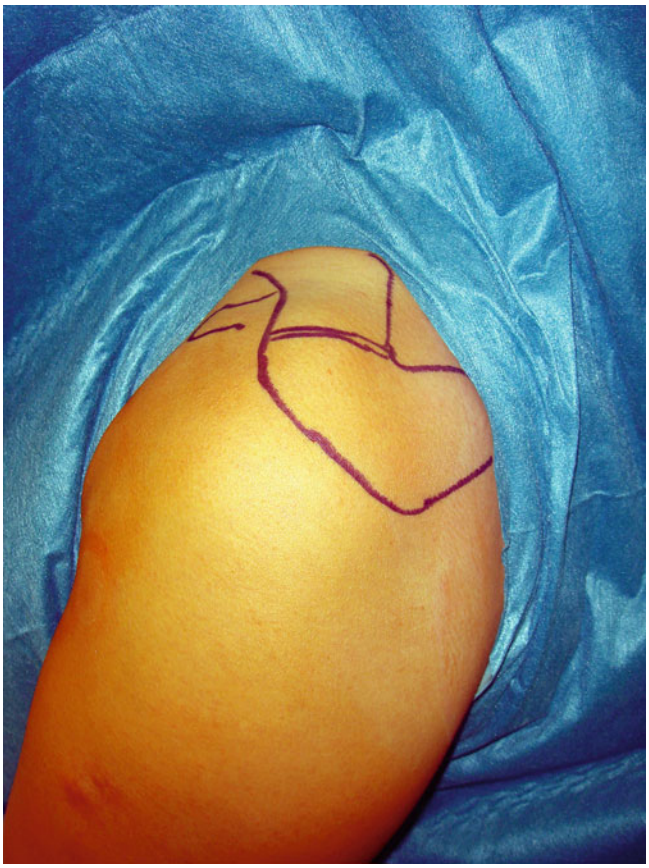

Fig. 11.1 Drawing of the osseous landmarks

Triangulation is the technique that permits the surgeon to know where he/she is, what to do to see a certain structure, and to take the instruments to the visual field. This is accomplished combining exterior visualization of the instruments and their directions combined with the marked bony references. If the surgeon cannot see the probe or surgical instrument he/she is using than with the arthroscope standing still, the instrument should touch it and follow it to the tip of the arthroscope in order to bring the instrument to the visual field.

The initial viewing portal for shoulder arthroscopy is the posterior portal. A stab incision, just enough to permit the introduction of the arthroscopic sheath with a blunt trocar, should be performed, 2 cm inferior and 1 cm medial to the posterolateral corner of the acromion (Fig. 11.2). The arthroscope passes the skin, the posterior deltoid, and the interval between the infraspinatus (IS) and the teres minor (TM). At that point the arthroscopic sheath should be directed towards the coracoid process, penetrating the posterior capsule between the humeral head and the posterior rim of the glenoid, entering the shoulder joint. This way the surgeon will avoid the neurovascular structures of the triangular interval (radial nerve and deep brachial artery), the triangular space (circumflex scapular vessels), and the quadrangular space (posterior humeral circumflex vessels and axillary nerve).

This same posterior portal is used to access the subacromial space after the complete glenohumeral arthroscopy. In order to enter the subacromial space, the arthroscopic sheath with the blunt trocar is withdrawn through the interval between the IS and TM and is redirected more laterally and superiorly in order to pass immediately under the acromion and reach its anterior tip. At this stage the tip of the trocar should be palpated under the skin just beneath the anterior border of the acromion and lateral to the coracohumeral ligament. The common errors are to place the arthroscope either to medial or to stay too posterior not entering the bursa. In that case the surgeon will not have a distended bursa and the vision will be disturbed by soft tissues around the lens. Several attempts should be made to reach the correct position by keeping in mind that this may lead to less fluid extravasation and soft tissue distension.

The posterior portal should be inferior enough to permit a smooth passage of the arthroscopic sheath. If a resection of

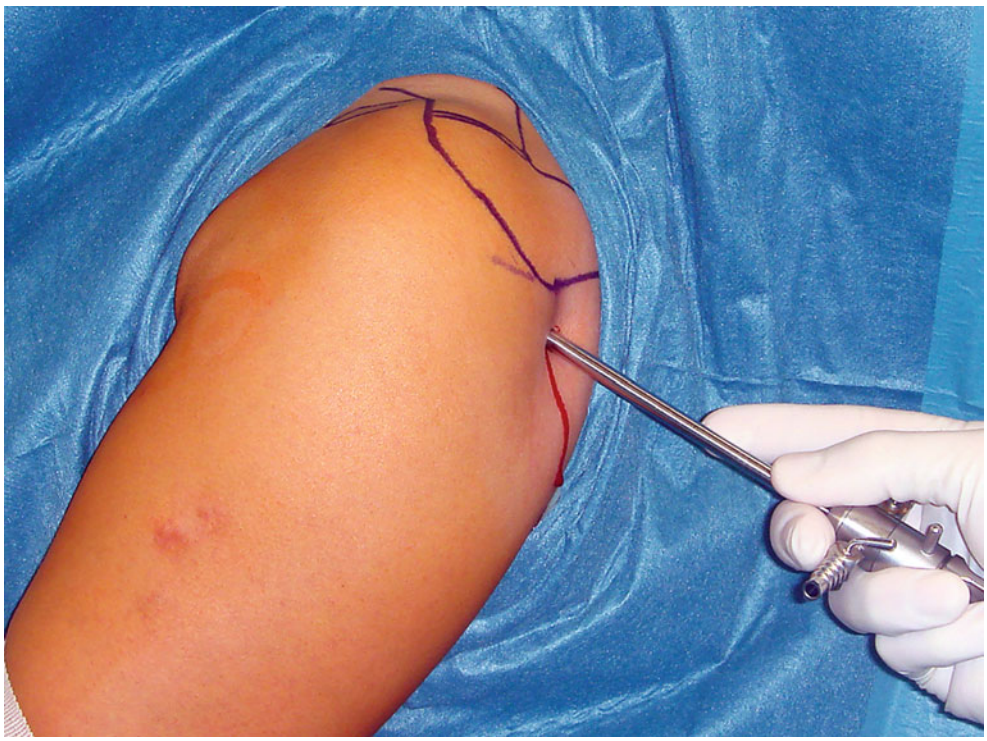

Fig. 11.2 Posterior portal

the distal clavicle is part of the procedure, the posterior portal can be made more medial, and on the contrary if a rotator cuff reconstruction is planned, a more lateral position of the portal is desirable. Nevertheless, it is important to search for balance. If a more lateral posterior portal will facilitate the view of the rotator cuff from the bursal side, a too lateral portal will make it very difficult to access the articular side of the same lesion.

In order to establish accessory portals, two methods are available. "Inside-out" method consists in placing the scope sheath in a chosen space inside the joint under direct view and to make protruding to the skin a switching stick trough the arthroscopic sheath. A skin incision is made, large enough to put a working cannula in place. This method is technically less demanding but limits the possible positions for portal placement and working field of the instruments. For instance, when dealing with an instability case, the instruments should be able to reach the anteroinferior capsule, and the placement of anchors in the anterior glenoid rim requires a 45º angulation.

The "outside-in" method consists in placing a needle inside the joint under direct visualization on a chosen place with the correct angle in order to turn possible or facilitate the procedure (Fig. 11.3). After selecting and confirming this way the correct position, a skin incision is performed and again a working cannula can be used. This is usually the method preferred by experienced shoulder surgeons but the "inside-out" method is very useful at the beginning of the learning curve.

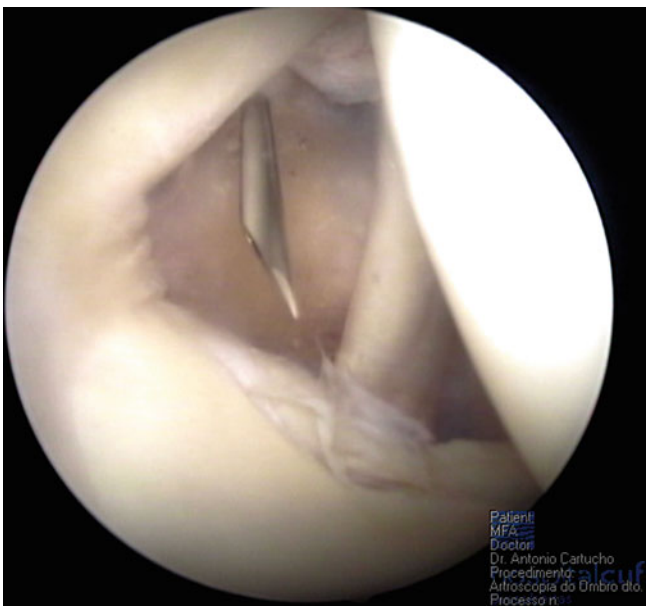

Fig. 11.3 "Outside-in" method

The anterior portal in the glenohumeral joint is created through the rotator interval. This space is limited superiorly by the long head of the biceps (LHB) and inferiorly by the superior border of the subscapularis (SbS) tendon. Again if the "outside-in" technique is used, the angulation on the superior to inferior and medial to lateral axis should be chosen according to the procedure.

The use or not of operating cannulas in this accessory portal is optional. With a cannula the inflow can be changed from the arthroscope to the cannula. These devices also permit a better fluid control and facilitate the repetitive entrance of the instruments. Nevertheless, cannulas may need longer incisions and limit the freedom of movement of the surgeon. For those reasons experienced shoulder surgeons tend not to use them specially when working outside the glenohumeral joint, namely, at the subacromial space.

In subacromial space, a lateral portal can be created in direct line with the posterior border of the clavicle and approximately 2–3 cm distal to the lateral border of the acromion. Again using the "outside-in" technique the surgeon must find the correct position in order to easily reach the undersurface of the acromion or the rotator cuff according to the planned procedure.

Glenohumeral Diagnostic Arthroscopy (Video 11.1)

After entering the joint trough the posterior portal, the surgeon should look for the LHB and the rotator interval limited superiorly by this structure and inferiorly by the SbS tendon (Fig. 11.4).

Diagnostic arthroscopy must be systematic in order to visualize all the structures. Initially the intra-articular visualization can be performed without distending the joint with fluid. This way the inflammatory signs are better quantified as the fluid pressure will alter the superficial vascularization. For didactic purposes four regions will be detailed.

Superior Region

The LHB is attached to the superior labrum. This structure with a triangular shape at its basis may have a meniscal-type insertion and may present several degrees of fraying or detachment as described by Snyder [2]. In order to evaluate the attachment site of this structure, a probe should be introduced from an anterior portal. Fraying and the presence of bare bone are not normal. Nevertheless, a careful inspection, the clinical history, examination, and the presence or not of biceps instability will determine the need for repair or tenodesis.

The coracohumeral ligament (CHL) encircles the biceps, sending fibers to the supraspinatus (SS) and SbS, contributing to form the bicipital groove, whose floor is formed by the superior glenohumeral ligament (SGHL) that runs from the anterosuperior part of the glenoid towards the lesser tuberosity.

The stability of the LHB can be assessed by moving the arm in flexion and abduction with internal/external rotation. At this point a probe from the anterior portal can pull inside the joint the extra-articular part of biceps to search for fraying, partial ruptures, or inflammatory signs.

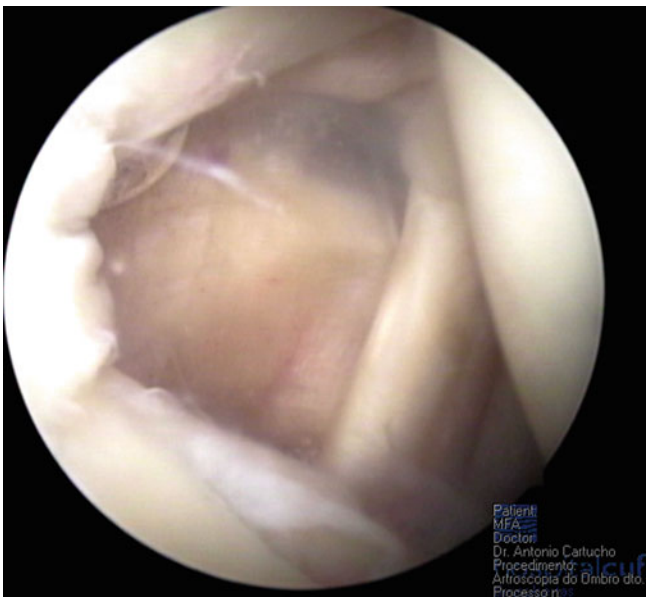

Fig. 11.4 Rotator interval

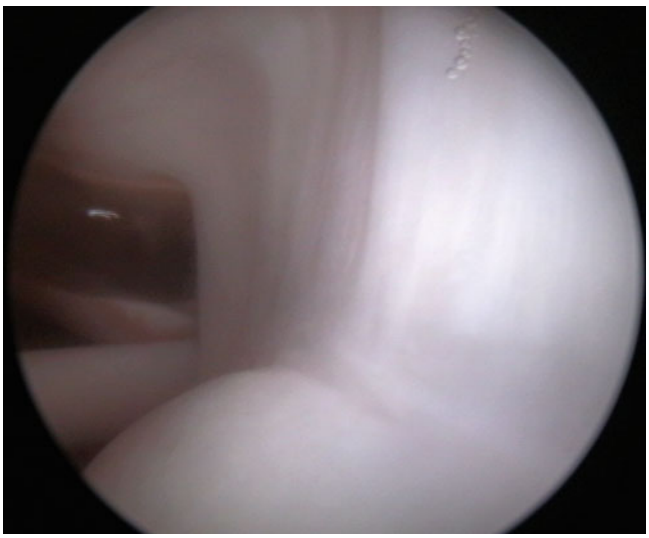

Fig. 11.5 Supraspinatus insertion

The arthroscope should be slightly withdrawn, and lowering the hand with a simultaneous rotation of the optical system, the SS insertion is inspected (Fig. 11.5). Partial degenerative articular-side tears present with a fraying, and usually traumatic partial ruptures have a flap of tissue protruding inside the joint. Any fraying of the SS should be debrided, and a marking suture should be put in place through a spinal needle in order to access the same segment of cuff from the subacromial space during bursoscopy. It is important, for treatment choice, to quantify the depth of the tear. This can be done using an instrument of known size between the cuff and the articular margin.

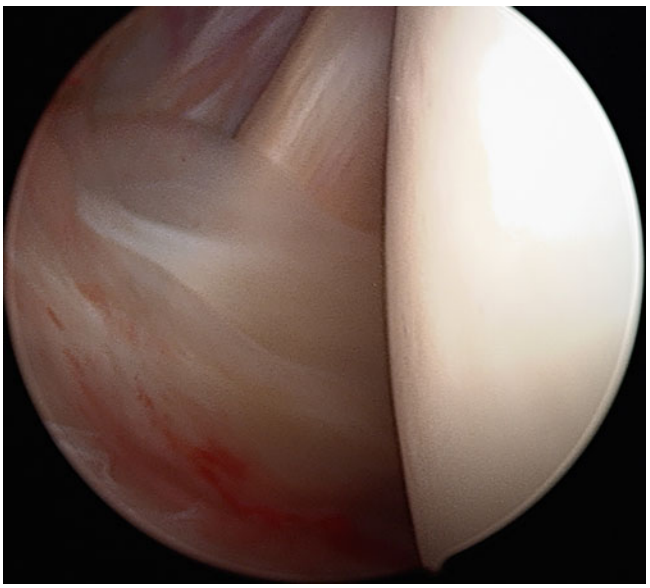

Fig. 11.6 Middle glenohumeral ligament

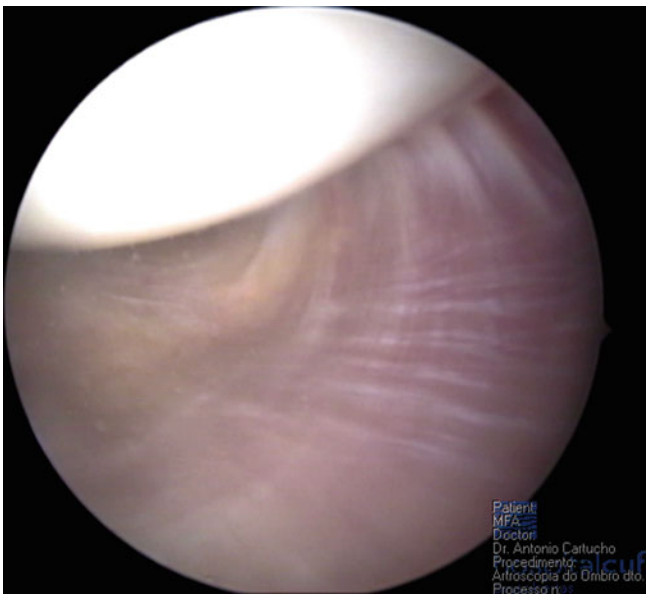

Fig. 11.7 Insertion of the capsule on the humerus

Continuing to go posterior after the bare area on the humeral head (HH) (no cartilage and nutritive holes with a pink aspect), the insertion of the IS is observed for the presence of tears or fraying. In this case the diagnosis of posterosuperior impingement should be kept in mind and confirmed by the clinical evaluation and by the contact of the IS with the posterosuperior labrum with the arm in abduction and external rotation [3].

Anterior Region

The articular cartilage of the HH and of the glenoid should be carefully inspected. An anterior entail of the glenoid rim at the "3 o'clock" position is normal constituting the glenoid notch. Inferiorly to it any detachment of the labrum is considered pathologic. On the other hand, superiorly to the glenoid notch the labrum can present a labral hole or be absent like in the Buford-type insertion of the medial glenohumeral ligament (MGHL) [4]. These variants are normal. Also a central depression of the cartilage is normal.

The SbS tendon should be inspected for intra-articular tears. The MGHL crosses the SbS at a 60° angle and varies from a firm structure to a transparent veil (Fig. 11.6). It runs from the humeral neck near the lesser tuberosity to the anterosuperior glenoid rim. At this stage the SbS recess should be inspect for loose bodies that would be missed otherwise.

Inferior Region

With gentle traction at 45º of abduction, the arthroscope is turned anteroinferiorly. The anterior band of the inferior glenohumeral ligament (IGHL) runs from the humerus to the anteroinferior glenoid rim. Continuing to look down the axillary pouch should be inspected and the presence of loose bodies noted. It should be remembered that the axillary nerve lies just beneath the capsule and can be damaged during surgical procedures for instability (capsular plication) or stiffness (capsulotomy) in this area. Slightly withdrawing the arthroscope, the posterior band of the IGHL can be accessed. In order to see the insertion of the capsule on the humerus with the arthroscope turned to the axillary pouch, the view should be turned up (Fig. 11.7). This way humeral avulsions of the glenohumeral ligament (HAGL) can be diagnosed.

Posterior Region

With the arthroscope nearly out of the joint, the posterior part of the HH should be inspected. Osteochondral lesions (Hill-Sachs lesions) are often seen in instability cases (Fig. 11.8), and engaging of the lesion over the anterior glenoid rim should be tested with the arm in abduction and external rotation. The posterior labrum must be inspected for detachments and fraying. Particularly, fraying of posterosuperior labrum associated with articular-side tear of the IS raises the suspicion of posterosuperior impingement [3].

This region should be inspected also looking from the anterior portal. This allows a better visualization of the posterior labrum and capsule and also an "over-the-top" view (Fig. 11.9) of the anterior structures. This is crucial in instability cases.

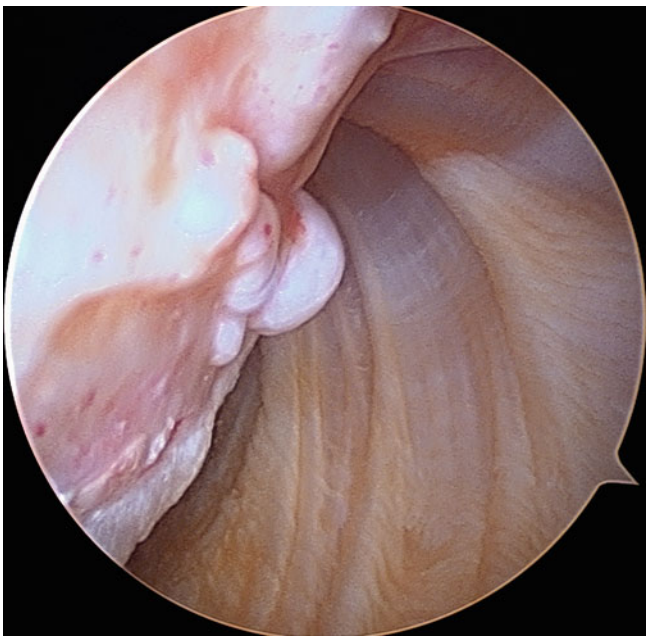

Fig. 11.8 Posterior part of the humeral head

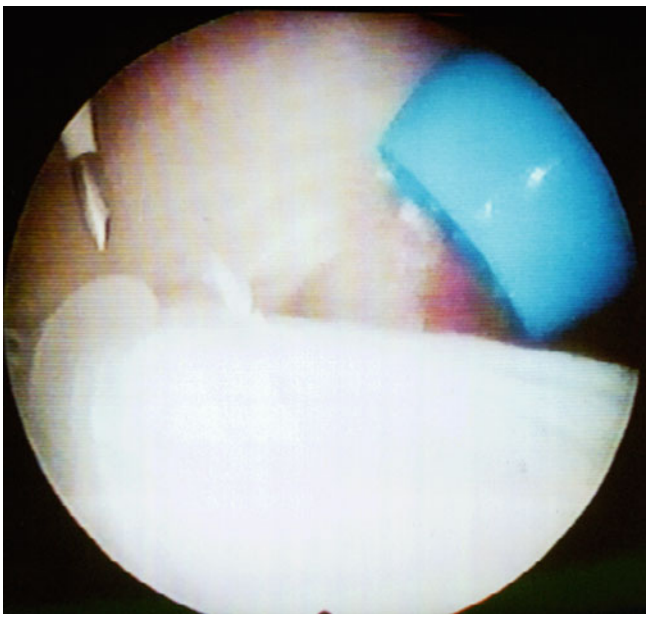

Fig. 11.10 Subacromial bursa

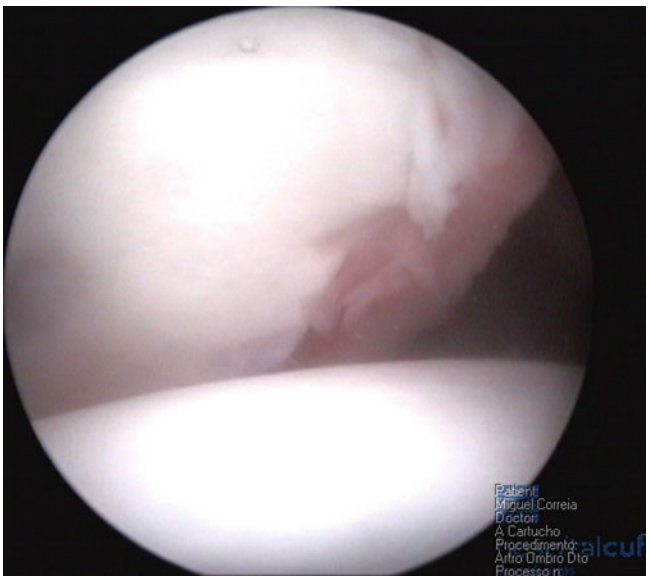

Fig. 11.9 View of posterior labrum and capsule from the anterior portal

Bursal Diagnostic Arthroscopy (Video 11.1)

The scope is introduced from the posterior portal and the lateral portal is placed according to the previously described technique. The surgeon must be aware that distending the bursa makes a view of the tendons possible from the beginning and that the need for extensive soft tissue debridement to have visualization is not frequent (Fig. 11.10). When bursectomy is necessary to improve visualization, after confirmation that the arthroscope is in the correct position, we prefer to use a shaver device that is kept close to the camera and facing up in order to not damage the rotator cuff. It is important to know that a functional rotator cuff tendon is not easy to damage even with a mechanic cutting device. Nevertheless, the surgeon should take all the necessary measures to avoid any iatrogenic damage.

In this compartment, outflow control is crucial. If excessive, the distended bursa will collapse. This causes bleeding, turning visualization very difficult. The acromial branch of the coracoacromial artery that lies near the coracoacromial ligament (CAL) is responsible for important bleeding when this ligament is resected. In this case aspiration should be stopped; if possible, the fluid pressure controlled by the pump is augmented to 70 mmHg, and with the tip off the arthroscope, the bleeding vessel is searched and identified. This way the incoming fluid from the arthroscope will wash away the blood, making it possible to coagulate the vessel with the help of a radiofrequency device. If in spite of these measures outflow control is not obtained, the surgeon must limit all losses of fluid with coverage of all portals with the finger and eventually check with the anesthesiologist the blood pressure of the patient. According to Morrison et al. there should be a difference of around 50 mmHg between the systolic pressure of the patient and the pressure at the subacromial space [5].

With the arthroscope facing down and rotating the arm, the quality of the tendons must be evaluated, and a tear must be characterized in location, shape, retraction, and mobility. At this stage changing the viewing portal to the lateral portal to have a frontal view of the tear can be useful (Fig. 11.11). This also permits to easily debride the posterior bursa with a shaver coming from the posterior portal and to determine the status of the muscle-tendon junction.

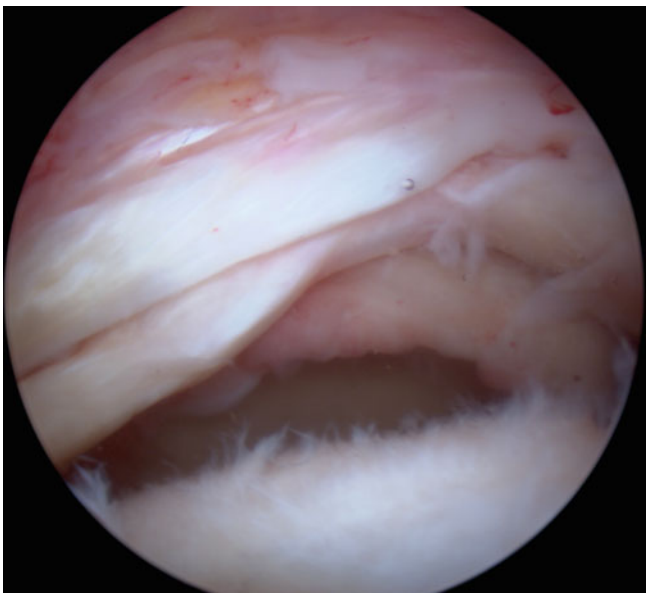

Fig. 11.11 Full-thickness rotator cuff tear (frontal view from the lateral portal)

With the arthroscope facing up, fraying of the undersurface of the acromion and CAL might be associated with subacromial impingement.

Summary

Arthroscopy is a precious diagnostic and surgical tool. Its practice has a long learning curve. Supported by a sound theoretical knowledge, one must practice first technical skills on plastic models and then move to practice in cadaver lab. A fellowship in shoulder surgery and visits to experienced surgeons are of great value. The first procedures should ideally be performed with the cooperation of an experienced shoulder arthroscopic surgeon.

Complications are rare in spite of anecdotic neurological, vascular, infectious, and pulmonary edema reports.

Arthroscopy permits the evaluation of shoulder structures with great detail, and the risk of overestimating the structural findings is a concern. There should be a clear relationship between the arthroscopic findings and the clinical history and examination before considering the proper treatment.

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Anchors and Sutures

12

Sean G. Haslam and F. Alan Barber

Introduction

Arthroscopic shoulder techniques have been significantly advanced as a result of two major events: the development of suture anchors and the introduction of ultrahigh molecular weight polyethylene (UHMWPE)-containing suture. These anchors and their sutures play key roles in current arthroscopic shoulder surgery. In general, shoulder anchors are used in two very different areas: the glenohumeral joint for labral and ligamentous attachment to the dense glenoid bone and the bursa for rotator cuff and biceps tendon attachment to the greater or lesser tuberosity.

Suture anchor designs tend to be procedure specific with different anchor characteristics and techniques applying to each site. Some anchor designs contain multiple sutures and hold well in osteoporotic bone (rotator cuff repairs), while others contain fewer sutures and work better in denser cortical bone (glenoid repairs). Most allow sutures to slide through an anchor eyelet for independent suture tensioning and to facilitate the creation of sliding locking knots. Knotless designs accept sutures from other anchors or the adjacent tissue and eliminate the knot-tying step. Occasionally some overlap does exist between these two areas. Shoulder instability reconstruction may require glenoid anchors for an anterior capsulolabral repair combined with cuff anchors for a remplissage attachment of the infraspinatus into the Hill-Sachs lesion.

This review will cover several different features common to both types of anchor environments as well as features which differentiate the suitability of one anchor type from another. These features include the material properties for anchors and sutures, the various knots and their uses, the currently used suture anchors, the principles of anchor placement and common failure mechanisms, and the current debate between single and double row cuff repair.

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Material Properties

Anchors

Anchors are currently available in metal (usually titanium), plastic (PEEK), biodegradable (PLLA, PDLLA, or PLA-PGA), and biocomposite (containing beta-tricalcium phosphate or hydroxyapatite) materials. While metal anchors were widely used in the past, biodegradable anchors demonstrate comparable pullout strength, eventually degrade completely, and avoid problems during revision surgery or postoperative imaging [1]. Consequently biodegradable anchors have attractive features for a shoulder suture anchor. The recent introduction of biocomposite materials offers the prospect of osteoconductive behavior leading to anchor replacement by bone at the end of the degradation process.

The selection of biodegradable anchors over metallic anchors is increasing as our knowledge of these newer materials expands. Bioabsorbable suture anchors have proven to be just as functional as metallic anchors. In addition to possessing adequate initial strength, these materials degrade over time after the tissue repair has healed and are replaced with fibrous tissue. Degradable materials commonly used in suture anchors are PGA (polyglycolic acid), PLLA (poly-L-lactic acid), stereoisomers of lactide such as PDLLA (poly-D-L-lactic acid), and combinations (copolymers) of lactide and glycolide. Slowly degrading biodegradable implants seldom cause lytic reactions as previously reported with the rapidly degrading PGA implants. However, in an effort to reduce the time needed for an implant to degrade, various stereoisomer combinations of PLLA [PD(96 %) L(4 %)LA or PD(70 %)L(30 %)LA] have been introduced as

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well as copolymers (PLLA co-PGA) which have not been associated with a lytic response.

Nonabsorbable, biologically inert polymers such as polyetheretherketone (PEEK) are also being used. PEEK is a very chemically resistant organic crystalline thermoplastic polymer which is adaptable to a wide pH range from 60 % sulfuric acid to 40 % sodium hydroxide and can resist deformation even at high temperatures. It can be combined with carbon fiber for reinforcement and has many current applications in orthopedic surgery beyond suture anchors.

These nonmetallic anchors offer several advantages including clearer postoperative imaging, easier revision, and fewer concerns about anchor migration, and they are not as likely to cause suture abrasion. While PEEK anchors are also radiolucent and can be drilled through during a revision procedure, since they do not absorb over time and are permanent, they present the same concerns as a metal anchor. Also, while a PEEK anchor can be drilled through in a revision procedure, it is difficult to remove all the small plastic shavings created during this process should they be thrown into the joint. Such PEEK shavings will never disappear and offer the potential to create abrasive injury to the articular cartilage. Finally, because of the age of the patient typically undergoing shoulder instability surgery, having an anchor which degrades over time is attractive because of the patient's anticipated longevity.

Biocomposite anchors reflect another significant advance in materials technology. Biocomposite materials are combinations of a degradable polymer with a bioceramic. Combinations of biodegradable polymers and beta-tricalcium phosphate (β-TCP) blend these two substances and result in a material possessing the properties of the two separate materials. For instance, the compressive strength and stiffness of β-TCP is very high and when blended imparts these characteristics to the biocomposite. As a result, a biocomposite will not only degrade over time but offer the chance of osteoconductive ingrowth of bone into the space previously occupied by the anchor. Biocomposites with β-TCP have demonstrated osteoconductivity which may result in bone ingrowth into the prior anchor site or may enhance material incorporation into host bone [2, 3].

Biocomposite technology promises to be a significant advance in arthroscopic implants. Examples of biocomposite suture anchors on the market today include the Arthrex (Naples, FL), BioComposite Corkscrew FT (85 % PLLA/15 % β-TCP), 3.5 mm BioComposite PushLock (85 % PLLA/15 % β-TCP), and the BioComposite SutureTak which uses a different biocomposite composed of 15 % β-TCP and 85 % PLDLA. The DePuy-Mitek (Raynham, MA) Lupine BR, Healix BR, Healix Advance BR, Transtend BR, and Gryphon BR anchors use a different biocomposite composed of 30 % β-TCP and 70 % PLGA. The PLGA copolymer portion is made of 15 % PGA and 85 % PLLA. Preliminary studies show that reabsorption of β-TCP-containing composite materials occurs within 18–24 months followed by significant bone ingrowth by 36 months.

Finally, anchors composed totally of suture material have been introduced. The first of these was the JuggerKnot 1.4 (Biomet Sports Medicine, Warsaw, IN) which is made from a single strand of No. 1 braided UHMWPE suture. The "anchor" portion is created with a short sleeve of braided polyester suture in the middle of the suture and is inserted into the bone. Traction on the suture bunches up the "V"-shaped suture sleeve creating the anchor within the bone. Additional JuggerKnot sizes have been introduced including a 1.5 and 2.9 mm version. Larger anchors are designed for cuff repair applications. Linvatec (Largo, FL) recently introduced its own suture-based anchor (the Y-Knot) which uses the same principle. As yet, no clinical studies are available on the effectiveness of these suture-based anchors.

Sutures

An arthroscopic suture should possess good handling characteristics, good strength, and good loop and knot security and be biocompatible. If degradation should occur, the suture should not create a significant inflammatory response. Furthermore, a superior arthroscopic suture offers greater strength for its size while maintaining a low friction surface conducive to tying in the wet, arthroscopic environment.

Current arthroscopic sutures can be monofilament, braided, or blended and either absorbable, nonabsorbable, or partially absorbable. Polydioxanone (PDS) is perhaps the most common biodegradable monofilament suture used. It is readily adaptable to arthroscopic surgery and frequently used for instability surgery. PDS can also be used to shuttle braided sutures through tissue. It is sometimes used as a marker stitch for the bursal identification of rotator cuff tears. It is used as a rotator interval closure stitch because it can be inserted directly with suture hook devices (Spectrum system, Linvatec, Largo, FL; Ideal suture hook, DePuy-Mitek, Raynham, MA). Polydioxanone suture degrades quickly. Two weeks after implantation, PDS sutures retain 60 % of the original strength and by 6 weeks 40 %. The suture is almost completely reabsorbed by 9 weeks. While easy to use and possessing reasonable strength, it is stiff and has a "memory" with the tendency for knots to unravel if an insufficient number of reinforcing half hitches are not placed.

Traditionally, nonabsorbable braided polyester suture, such as Ethibond, has been used arthroscopically for soft tissue repair and in suture anchors. In the past decade, this braided polyester has been replaced in arthroscopic applications and in all current suture anchors by ultrahigh molecular weight polyethylene (UHMWPE)-containing suture. The first of this type was FiberWire (Arthrex) which has a braided

polyester coat around a central core of multiple small strands of UHMWPE. FiberWire redefined suture performance because it was much stronger and consequently less likely to break than the braided polyester. Competitors struggling to catch up with this watershed event turned to pure braided UHMWPE suture. The single manufacturer of this UHMWPE fiber made it available to other companies in a braided design, and now braided UHMWPE suture is marketed under several different brand names. The pure braided UHMWPE suture has almost twice the ultimate strength of FiberWire (which is partially braided polyester) and a 500-fold increase in resistance to fraying compared to pure braided polyester suture [4]. The most recent addition to the family of high-strength sutures is OrthoCord (DePuy-Mitek) which is used in the DePuy-Mitek suture anchors. OrthoCord combines both UHMWPE suture with a degradable suture. The size No. 2 combines 32 % UHMWPE with 68 % polydioxanone (PDS) and is coated with polyglactin 910. The OrthoCord design has a PDS core with a UHMWPE sleeve [5] and leaves a lower profile after the PDS reabsorbs while retaining the outer sleeve strength.

While these UHMWPE-containing sutures have distinct advantages in arthroscopic shoulder instability surgery, concerns about mechanical irritation, articular cartilage erosion, tissue abrasion while running the suture through tissue, and impingement persist. As yet, no completely absorbable ultrahigh-strength suture exists although it is a desirable goal.

Additionally, concerns exist that knots tied with these UHMWPE high-strength sutures are susceptible to slippage at loads below the expected failure load [6]. Perhaps this has something to do with the physical properties of the UHMWPE suture and the personality of the knot being tied. In other words are some knots tied with the high-strength suture more susceptible to slippage than other knots? The answer is clearly yes. Certain knots slip more often by the nature of their configuration. The worst of these was the Duncan knot and Weston knot which slipped 97 and 86 % of the time, respectively [7]. The best-performing knots were the SMC and Revo knots which slipped only 1 and 3 %, respectively. The San Diego and Tennessee slider slipped less than 10 % of the time [7]. Using the higher-strength suture may mean a greater risk of knot slippage, but this risk can be mitigated by choosing the right knot.

Knot Types and Uses

Every arthroscopic surgeon needs to know at least two knots: a sliding knot and a non-sliding knot. The surgeon must also become acquainted with the principles of knot security and loop security. Knot security is the ability of the knot to resist slipping when a load is applied. Three factors can effect this: friction, internal interference, and slack between throws [8]. Loop security is the ability of maintaining the size and tension on the loop *during* knot tying [9]. So it is possible to have a nice secure knot on a loose loop (poor loop security), and hence the construct will be ineffective in tissue repair. Both knot security and loop security can be influenced by surgeon technique and the material property of the suture.

All arthroscopically tied knots (whether sliding or non-sliding) have an initial foundational knot that removes slack at the tissue and is then locked by several additional half hitches. Sliding knots start with a specific locking hitch created outside the joint, while non-sliding knots create the locking hitch from a series of half hitches placed at the repair site. In the non-arthroscopic setting, a surgeon is able to create square throws. In the arthroscopic setting only asymmetrical tension can be applied to the two suture strands. This feature can create a less secure knot. More complex sliding locking knots have been developed to counter this issue. These knots develop internal resistance and lock resulting in better knot and loop security.

Sliding Knots

Sliding knots can be used when the suture is able to glide freely either through the anchor eyelet or through the tissue when each arm of the suture is pulled. Sliding knots can be categorized into locking and non-locking knots. A locking knot (such as the SMC, Tennessee slider, San Diego, and Weston) has an internal locking mechanism such that when the non-post limb of suture is tensioned, the knot changes its configuration and locks in place. The surgeon will sense that the knot has locked by feeling a snapping or clunking sensation through the sutures. Once the knot has locked, it cannot be moved, so it is important to visualize the knot and make sure that the knot is in correct position before locking it. A non-locking knot (such as the Duncan knot) is held in place by the friction of the suture as the knot is tightened. There is no locking mechanism. One downside to the non-locking knot is that if the construct is kept under tension, the loop can be easily loosened as the knot backs off. Every sliding knot should be accompanied by 3 or 4 reversing half hitches. Failure to do so will decrease both knot security and loop security, resulting in a weak and loose suture construct [10].

As mentioned UHMWPE-containing sutures have less friction than the previous generation of braided polyester sutures. This results in non-locking knots being much more likely to slip prior to ultimate failure strength. Consequently, tying this type of knot using UHMWPE suture is not recommended.

Duncan Knot

The steps required to create a Duncan loop (Fig. 12.1) start with grasping the sutures between the thumb and index finger and creating a loop by passing the loop strand over the

Fig. 12.1 The Duncan knot is a sliding knot without a locking mechanism. It requires four reversed half hitches to secure the knot (© F. Alan Barber MD reproduced with permission)

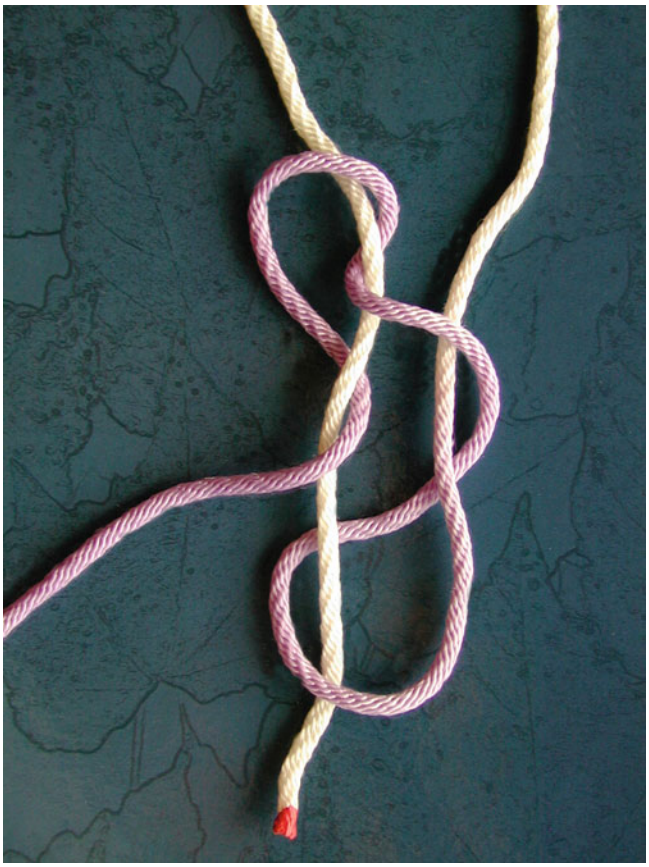

Fig. 12.2 SMC knot is a consistent sliding, locking knot with excellent properties (© F. Alan Barber MD reproduced with permission)

post. The loop strand is wrapped in the same direction placing four subsequent throws around the post limb. The free end of the loop limb is then passed back through the original loop and the knot is tightened removing any slack. Once the knot is created, the post strand is pulled, advancing the knot down the post and to the tissue interface. Three or four reversed half hitches are then tied to secure the knot.

SMC Knot

The SMC (Samsung Medical Center) knot (Fig. 12.2) is a common sliding knot which can provide good loop and knot security. Kim and Ha described the knot in these words: "While grasping the post strand, make an underhand throw with the loop strand under both the loop and the post strands. Make a second underhand throw with the loop strand under the post strand. Bring the loop strand behind the second throw and make an underhand throw with the loop strand under the post strand. After this third throw, do not tighten the knot. By pulling the post strand, the knot is introduced into the joint without difficulty. The post strand is tightened until the snug knot is established. At this stage, a small locking loop is created in the knot. While maintaining the tension of the post strand using the knot pusher, the loop strand is pulled until the locking loop is incorporated into the knot" [11]. While Kim and Ha describe leaving the "locking loop" wide open within in the knot, we prefer to "police" the knot, so that the knot is tidy and ready to slide through the cannula. The knot is then reinforced with four reversing half hitches.

Tennessee Slider

The Tennessee slider (Fig. 12.3) is a clove hitch on the post limb followed by a series of reversed half hitches with alternating posts. To begin, the loop limb is thrown over and around the post limb and then comes back under the loop limb circumnavigating both limbs. It continues around and back over the post limb again in the same counterclockwise direction but is brought back through the interval created between the first pass under the post limb and the second pass over the post limb (creating the clove hitch). At this point, the slack is removed from the knot, and it is advanced into position by pulling on the post limb and pushing with a single lumen knot pusher. Once in position, tensioning the free limb locks the knot. It is then secured with four reversing half hitches.

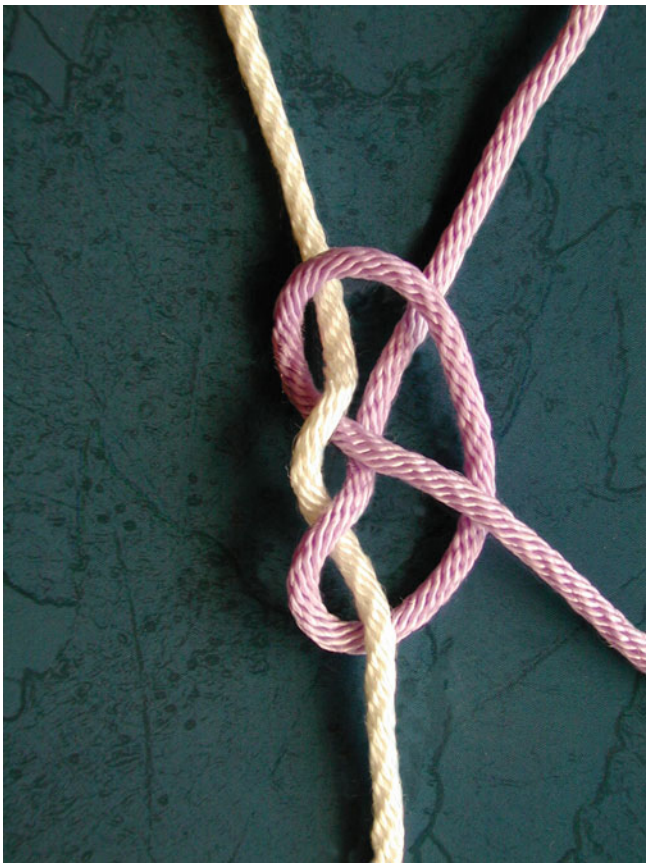

Fig. 12.3 The Tennessee slider is a clove hitch which functions as a sliding, locking knot (© F. Alan Barber MD reproduced with permission)

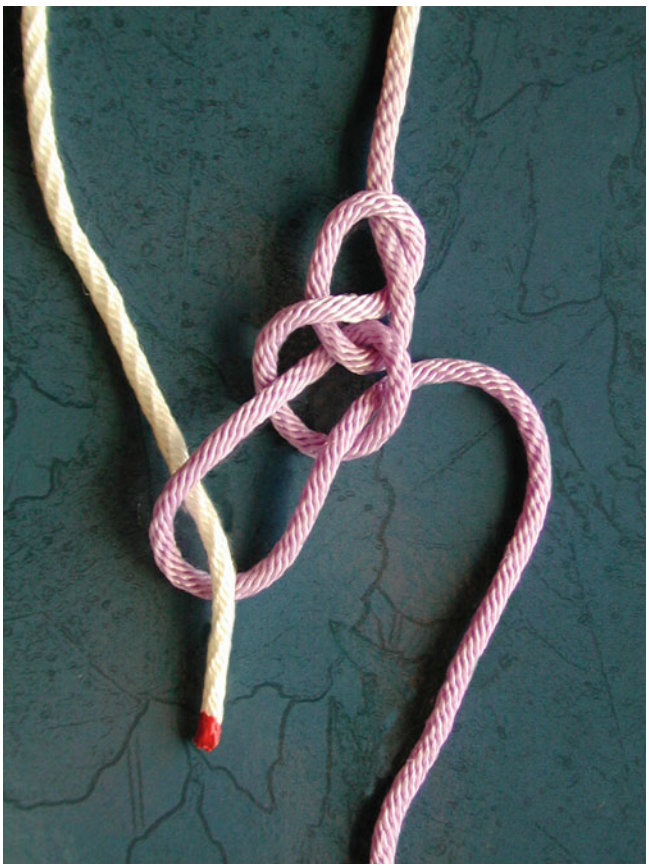

Fig. 12.4 The San Diego knot is a sliding, locking knot (© F. Alan Barber MD reproduced with permission)

San Diego Knot

The San Diego knot, described by Abbi et al. [6] (Fig. 12.4), starts with the post limb, which is shortened as much as possible. The loop limb is used to create a slip loop that is tightened securely to remove any slack. A second slip loop is then created using the loop of the initial slip loop and the loop limb, which is passed into the first slip loop to create the two linked slip loops. The first slip loop is tightened securely as the second slip loop is created. However, the second slip loop is left open. The post strand is then passed through the second (and open) slip loop, and the knot is "policed" by taking the slack from the knot. The knot is delivered down the cannula by pulling the post strand and pushing with a single lumen knot pusher. Once the knot is in proper position, the knot is locked by pulling firmly on the loop limb to lock the loop. It is then secured with four reversing half hitches.

The Weston Knot

The Weston knot (Fig. 12.5) is tied in similar fashion to other sliding knots. The post strand is shortened maximally. The free limb is brought over the post and held between the finger and the thumb. This creates a loop that will be used in the last step of tying the knot. The free limb is brought around the post passing underneath the post, then in between the two suture strands and over the loop strand. The free limb then continues around and underneath the loop strand, then over the post strand coursing between the two suture strands. The free limb is then brought around and underneath both strands circumnavigating the strands until it is brought above then through the loop which was created in the first step of the knot tying. The knot is "policed" and is ready to slide through the cannula and into the joint. Tensioning the free limb locks the knot. It is then secured with four reversing half hitches.

Non-sliding Knots

Non-sliding knots are usually reserved for those times when the suture is not able to move freely through the anchor or tissue. Sometimes the suture is stuck in the tissue or anchor; sometimes concerns exist about suture breaking when a frayed section is observed. The Revo knot (Fig. 12.6) is a commonly used non-sliding knot which has shown comparable performance in terms of knot strength compared to sliding knots [6, 7]. The static surgeon's knot is another

Fig. 12.5 The Weston knot is a sliding, locking knot (© F. Alan Barber MD reproduced with permission)

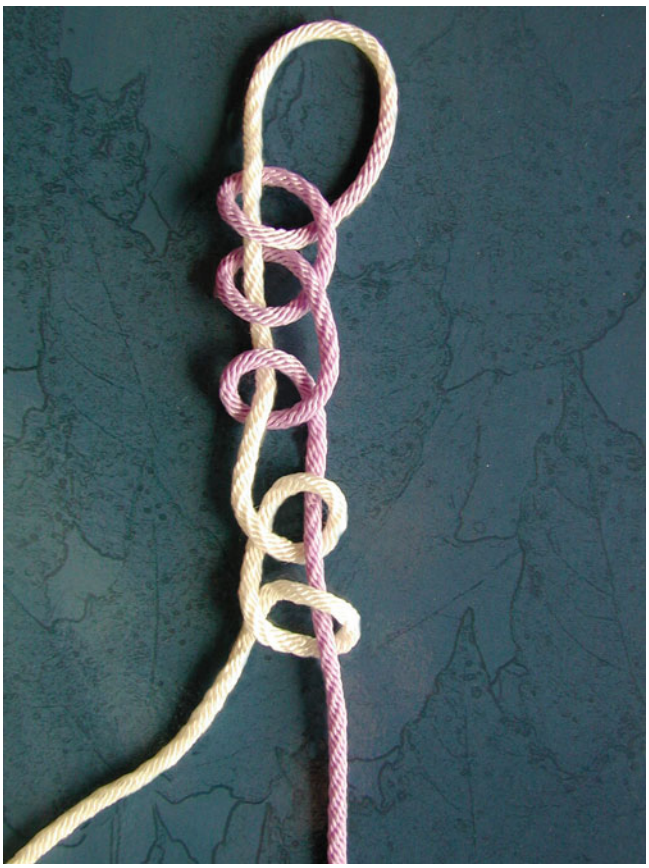

Fig. 12.6 The Revo knot is a non-sliding knot (© F. Alan Barber MD reproduced with permission)

commonly used non-sliding knot. It is tied by throwing three half hitches on the same post, followed by three reversing half hitches on alternating posts. One study found the surgeon's knot to hold the best balance of knot security and loop security when compared to various sliding knots [10].

Revo Knot

The first two half hitches of the Revo knot are thrown in the same direction on the same post. The third half hitch reverses the direction and uses the same post. These three half hitches are tensioned at this point using a "past point" technique with a single lumen knot pusher. Two additional half hitches are then thrown using the other suture limb as the post while alternating the direction of the throw (Fig. 12.6).

Suture Anchors

Anchors used in the shoulder come in three predominant varieties based upon function: medial row rotator cuff anchors, lateral row rotator cuff anchors, and glenoid instability anchors. Medial row anchors tend to be more robust and larger than the other two types. This allows the anchor to withstand greater biomechanical stresses at the medial footprint of the rotator cuff which can measure over 900 N in some instances in the infraspinatus [12]. These medial row anchors are usually inserted into the bone in a screw-type fashion. Lateral row anchors are usually knotless in design. The knotless lateral row anchor allows the use of a suture bridge [13–16]. The sutures from medial row anchors are passed through the tendon to create a mattress stitch. After tying knots in these sutures, a suture bridge is created using the suture tails from the medial row repair. These sutures are left long instead of being cut short. These long tails are threaded through a knotless anchor which is then inserted into a predrilled site lateral to the medial row anchor. One suture from each knot of the medial row is passed through the anterior lateral row anchor, and one suture from each medial row knot is passed through the posterior lateral row anchor before the anchors are inserted. The sutures are tensioned to stretch over the adjacent cuff tendon and to create a bridge over the footprint (Fig. 12.7). This applies pressure to the cuff footprint and compresses the tendon against the greater tuberosity bone bed during healing. Some advocate placing the lateral row anchors "over the top" on the lateral side of the greater tuberosity parallel to the cuff tendon. This

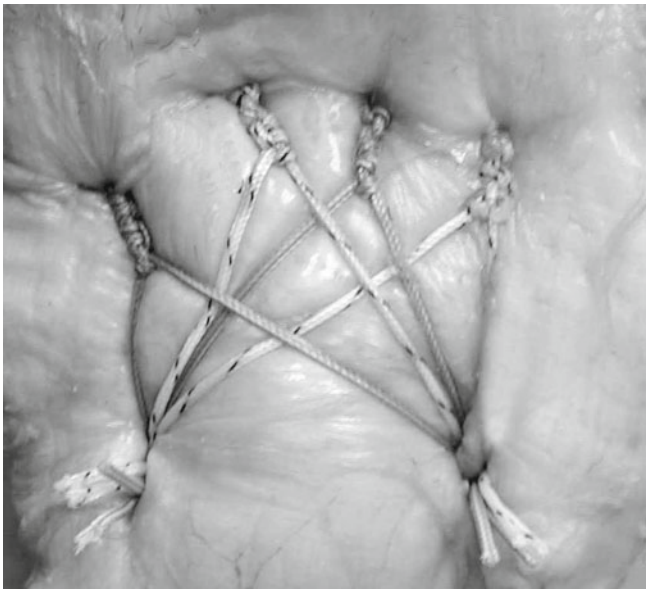

Fig. 12.7 Double row arthroscopic rotator cuff repairs often utilize a suture bridge technique. The suture strands from the four medial row knots from two anchors (*top* of the image) are tied securely and left long enough to be threaded through two lateral row knotless anchors. Once the sutures are tensioned appropriately, these lateral row anchors are implanted (*bottom* of the image), the excess suture material cut, and the suture bridge completed (Superior view of a cadaver specimen) (© F. Alan Barber MD reproduced with permission)

"orthogonal" or "anatomic" anchor position is felt by its proponents to be superior to placing the anchor in a "Deadman angle" at the edge of the greater tuberosity.

The third variety of anchor is the glenoid anchor. These anchors are principally designed for shoulder stabilization procedures in younger patients with better bone quality than those undergoing a rotator cuff repair. Instability rehabilitation programs generally call for shorter immobilization periods which results in stresses being applied sooner to the suture-tissue repair site than a cuff repair. On the other hand, the capsule-labral tissue and bone involved in a shoulder instability case are more robust than the tissues encountered in cuff repairs. Consequently the biomechanical properties and design features of an acceptable glenoid anchor will be different from the one used in the humeral tuberosity.

A glenoid anchor is smaller, has a low profile, and is designed to be inserted into cortical bone.

The anchors best suited for the glenoid rim are smaller in size, ranging from under 2 mm in diameter to as large as 3.5 mm. This smaller size meets the requirements of the confined space and dense glenoid rim. Toggle anchor designs that would not be as appropriate for a decorticated greater tuberosity can be used in the glenoid. Smaller and shorter anchors allow more options in the limited space of the glenohumeral joint. The shorter anchor is advantageous especially to avoid over-penetration of the inferior glenoid near the 6 o'clock position. A longer anchor drill could break through inferior to the shoulder capsule or into the axillary space and potentially injure the axillary nerve.

The trade-off is that smaller anchors generally have lower load to failure strengths than larger anchors. Therefore, a reasonable balance between anchor size and holding strength must be sought. Smaller glenoid anchors cannot accommodate as many sutures as the larger cuff anchors, and this must be considered in selecting an anchor for glenoid capsule-ligamentous repair. Glenoid anchors come in both knotless and knot-tying designs.

Over time increasingly effective anchor designs have been introduced and older designs retired. As these designs evolve, the previous emphasis on failure strength has been replaced by concerns about the anchor size, the number of accompanying sutures, the anchor material (biodegradable and biocomposite), and the surgeon's preferred technique. The following is a list of current anchors listed alphabetically by manufacturer. It is not meant to be comprehensive or endorse one anchor over another. Arthroscopic surgeons should assess the pros and cons of each anchor and choose the anchor best suited to the case at hand.

Arthrex (Naples, FL)

Corkscrew Family (Medial Row Anchor)

This anchor design (Fig. 12.8) can be made of PLLA (BioCorkscrew FT), PEEK (PEEK Corkscrew FT), β-TCP-PLLA blend (Biocomposite Corkscrew FT), or titanium (Corkscrew FT). The FT stands for fully threaded. In the Corkscrew family, the eyelet is not made from a polymer but from a #4 braided polyester suture loop that is molded into the body to create a distinctive suture eyelet that provides less suture abrasion when tying the attached sutures. The load to failure strength of the anchor in cancellous bone is acceptable. The Corkscrew family of anchors is available with two braided polyester #2 sutures, with two solid color #2 FiberWire sutures, or with two #2TigerTail (FiberWire with stripes) sutures. The anchor comes in three sizes: 4.5, 5.5, and 6.5 mm.

The original titanium Corkscrew FT anchor was revised to have two individual suture eyelets and two sutures (either #2 FiberWire or braided polyester). The anchor threads are widely spaced to work in cancellous bone, and the anchor comes in 4.5, 5.5, and 6.5 mm diameters. The anchor insertion shaft has a vertical laser mark on the distal part to indicate the suture eyelet orientation and is suitable for rotator cuff repair.

SwiveLock (Lateral Row Anchor)

SwiveLock anchors are knotless anchors meant for lateral row constructs (Fig. 12.8). They are made of PLLA, PEEK, or β-TCP-PLLA blend. Every anchor however has a distal eyelet that is made of PEEK regardless of the material in the anchor body. The SwiveLock anchors are available in 2.9, 3.5, and

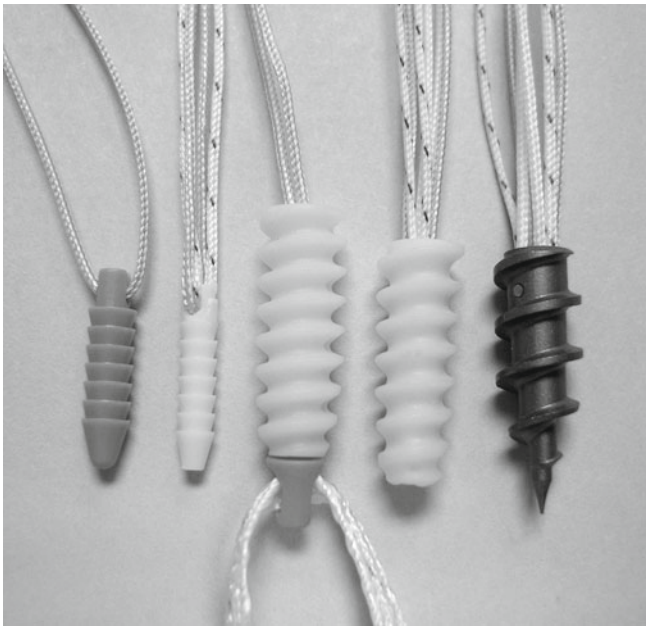

Fig. 12.8 Arthrex has several anchors of different types. These are loaded with FiberWire, usually fully threaded, and are available in PEEK, biocomposite, and titanium materials. *From left to right* are the PEEK SutureTak (single loaded), BioSutureTak (double loaded), Biocomposite SwiveLock (loaded distally with 2 mm FiberTape), BioCorkscrew FT (triple loaded), and the titanium Corkscrew FT (triple loaded) (© F. Alan Barber MD reproduced with permission)

4.5 mm sizes. They do not come with preloaded sutures but are meant to utilize the sutures from the tied knots of medial row anchors which can be threaded through the distal anchor eyelet. The suture is then tensioned to the desired amount and the anchor is screwed into a predrilled hole. This technique allows the surgeon to have some control over the suture tension.

SutureTak (Glenoid Anchor)

Like the other Arthrex anchors, the SutureTak comes in PLLA, PEEK, or β-TCP-PLLA blend varieties (Fig. 12.8). The PLLA and β-TCP-PLLA blend options are designed with the suture molded into the anchor. The sizes include 2.0, 2.4, 3.0, and 3.7 mm options. The 2 mm anchor comes single loaded with #1 FiberWire. The 2.4 and 3.7 mm anchors come single or double loaded with #2 FiberWire. The middle 3 mm option anchor comes either single or double preloaded with either #2 FiberWire or #2 TigerTail (FiberWire with stripes) sutures.

Biomet Sports Medicine (Warsaw, IN)

JuggerKnot (Glenoid Anchor)

This unique anchor is comprised completely of suture (Fig. 12.9). There are currently three different versions: a 1.4, 1.5, and 2.9 mm. The two smaller anchors are designed as glenoid anchors and the larger is designed as a rotator cuff

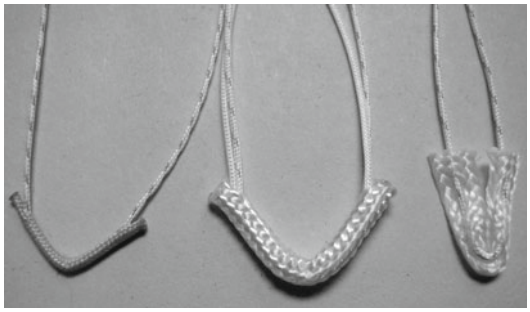

Fig. 12.9 Suture-based anchors are currently offered by both BioMet Sports Medicine and ConMedLinvatec. Three examples of these anchors are *from left to right* the Biomet JuggerKnot 1.4, JuggerKnot 2.9, and Linvatec Y-Knot (© F. Alan Barber MD reproduced with permission)

anchor. In the smaller anchors a #1 MaxBraid suture passes though a #5 polyester suture sleeve which deploys into a "V" configuration. The entire anchor measures 1.4 mm when it is inserted but expands much larger than this when it takes on the "V" configuration. This configuration prevents it from exiting the small drill hole when tensioned. An intact cortex is required to act as a backstop for this anchor deployment.

DePuy-Mitek (Raynham, MA)

Healix Anchor (Medial Row Anchor)

The Healix anchor is available made of PEEK, titanium, or β-TCP/PLGA (beta-tricalcium phosphate and poly(lactide co-glycolide)) biocomposite (Fig. 12.10). The PLGA copolymer is composed of 15 % PGA and 85 % PLLA. The advantage of the β-TCP is its osteoconductive property and the potential for bone ingrowth into the anchor location after degradation. The anchor is available in three sizes: 4.5, 5.5, and 6.5 mm diameters. These anchors are double or triple loaded with #2 OrthoCord. Recent modifications of this anchor include the Healix Advance and the Healix Knotless anchors. In the Healix Advance the screw threads are modified and the distal crossbar eyelet is protected to avoid breaking during insertion. The Knotless version has a side slot which captures the sutures, locking them in place.

VersaLok (Lateral Row Anchor)

This knotless anchor is an expanding bolt with two separate components. The pre-deployment outer diameter is 4.9 mm and the length is 27 mm. During deployment, the titanium pin is forced inside the outer PEEK outer sleeve. This expands the outer diameter of the implant to 6.3 mm and shortens the length of the device to 17 mm. For insertion the anchor is positioned over a hole prepared with a 2.9 mm awl. The anchor is then inserted into the hole by use of a mallet. The sutures are tensioned, and when adequate tension is achieved,

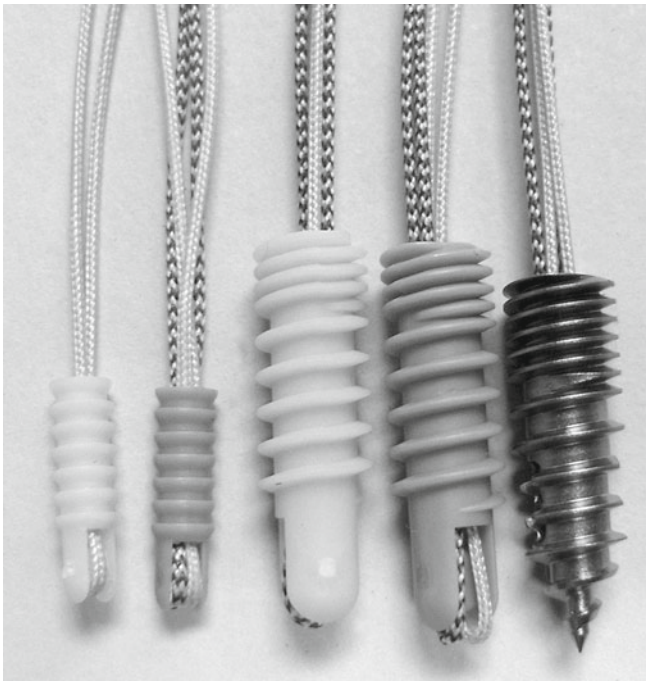

Fig. 12.10 DePuy-Mitek anchors, loaded with No. 2 OrthoCord, are available in PEEK, biocomposite (β-TCP-PLGA), and titanium material. *From left to right* are the Gryphon BR and Gryphon PEEK glenoid anchors followed by the Healixbiocomposite (β-TCP-PLGA), PEEK, and titanium anchors (© F. Alan Barber MD reproduced with permission)

the trigger on the applicator is pulled to lock the anchor and suture in place. Pulling the trigger causes the titanium pin to advance into the anchor sleeve. This serves two functions. It secures the sutures by pinching them between the inner titanium pin and the outer PEEK anchor sleeve, and the titanium pin expands against the anchor sleeve also causing the sleeve to expand as the pin is deployed. This also gives a press fit between the anchor and bone as the anchor expands.

Gryphon BR (Glenoid Anchor)

Gryphon BR (Fig. 12.10) is a push-in anchor with seven ribs composed of Biocryl Rapide: 30 % β-TCP/70 % PLGA. It comes with one or two No. 2 OrthoCord (UHMWPE and polydioxanone) sutures. The sutures pass down a central hollow core in the anchor and loop around a distal cross bar eyelet. This low-profile anchor is smaller in design to allow for placement in the glenoid. A hole is predrilled using a drill and the anchor is inserted with a mallet.

Smith and Nephew (Andover, MA)

TwinFix Ultra (Medial Row Anchor)

This screw-in anchor is made of PEEK, titanium, or PLLA/HA (poly-L-lactic acid and hydroxyapatite) (Fig. 12.11). During manufacturing of the biocomposite anchor, the PLLA and HA are blended together and then injected into a mold to form the anchor. The intent of the biocomposite design is so it will offer better integration into bone by taking advantage of the osteoconductive properties of the HA component. These anchors accommodate two or three sutures through dual eyelets located toward the upper region of the anchor and are either double loaded or triple loaded with #2 UltraBraid in various colors (blue suture, blue-cobraid, and black-cobraid). The TwinFix anchors are provided in three different sizes: 4.5, 5.5, and 6.5 mm diameter.

Footprint Anchor (Lateral Row Anchor)

This knotless anchor (Fig. 12.11) is intended to be used in conjunction with medial row anchors. It comes only made of PEEK and has a 5.5 and 6.5 mm diameter option. The long suture arms from the medial row knots are loaded through the eyelet and the anchor is inserted into a predrilled hole. The sutures are pulled to the desired tension then the knob at the end of the applicator is rotated clockwise. This action deploys and advances a central plug inside the anchor to pinch the sutures and maintain the tension in an interference fashion. If the tension of the sutures is too tight or too loose, the knob can be rotated counterclockwise to back the central plug off of the sutures. This allows the sutures to move freely once again and to be re-tensioned. Care should be made to back the central plug up only enough to allow the sutures to move freely, as excessive backing out of the central plug can disengage it from the anchor.

Raptor Anchor (Glenoid Anchor)

The BioRaptor was the first generation of this anchor. This is a non-screw push-in anchor with a series of raised ridges that run the entire length of the shaft. The eyelet is located at the midpoint (3 ridges below and 4 above) of the anchor and allows it to be press-fit into a drilled hole. It comes single or double loaded with two #2 UltraBraid (UMMWPE) sutures. These sutures pass through a single eyelet set transversely and positioned in the midportion of the anchor, thus avoiding a superior stump above the ribs. It comes in 2.3 and 2.9 mm diameters. In the 2.3 version, 5 ridges are found in the superior 2/3 of the anchor, and the eyelet is immediately below these. The numbers associated with the anchor name are deceptive since they relate only to the minor diameter of the anchor. Most other anchors are named for their major or largest diameter. For comparison, the major diameter of the BioRaptor 2.9 is actually 3.7 mm. The major diameter of the BioRaptor 2.3 is actually 3.0 mm. Also, the BioRaptor 2.3 is not biodegradable as its name suggests. It is actually a nonabsorbable anchor made from PEEK. Recently, a biodegradable biocomposite version of the BioRaptor (the OsteoRaptor) was introduced (Fig. 12.11). It shares the same design and size options but is made of a blend of PLLA/HA (hydroxyapatite).

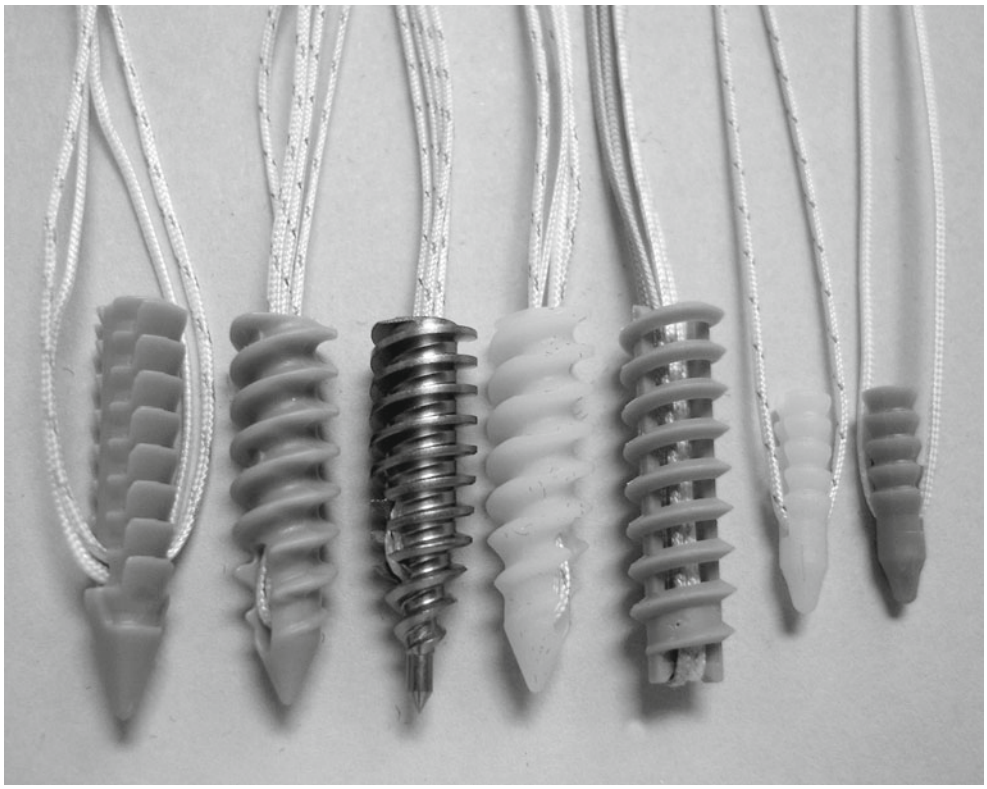

Fig. 12.11 Smith and Nephew anchors are made of PEEK, titanium, and a biocomposite (PLLA/HA). Anchors shown *from left to right* include the cuff anchors Footprint Ultra (a knotless anchor), three versions of the TwinFix Ultra (provided in 4.5, 5.5 – shown here, and 6.5 mm sizes) using different materials, the PEEK Healicoil 5.5, and the glenoid anchors OsteoRaptor 2.3 and PEEK BioRaptor 2.3 (© F. Alan Barber MD reproduced with permission)

ConMed-Linvatec (Largo, FL)

Super Revo FT (Medial Row Anchor)

This fully threaded self-drilling titanium screw anchor (Fig. 12.12) has an internal independent suture sliding eyelet. It is pre-threaded with two or three No. 2 Hi-Fi sutures and is designed for cortical fixation.

CrossFT (Medial Row Anchor)

This is a PEEK 5.5 mm diameter screw anchor (also available as a biocomposite anchor) with a distal crossbar eyelet and accommodates up to 3 No. 2 Hi-Fi sutures (Fig. 12.12). It is fully threaded with one thread running the entire length of the anchor and a second thread at the proximal end to maximize cortical compression.

Y-Knot (Glenoid Anchor)

This all-suture anchor comes with 1 blue-white braided UHMWPE suture (Fig. 12.9). In contrast to the Biomet JuggerKnot anchors, the Y-Knot anchor has a flat braided UHMWPE threaded with one #2 UHMWPE suture. It is inserted into a predrilled 1.3 mm hole.

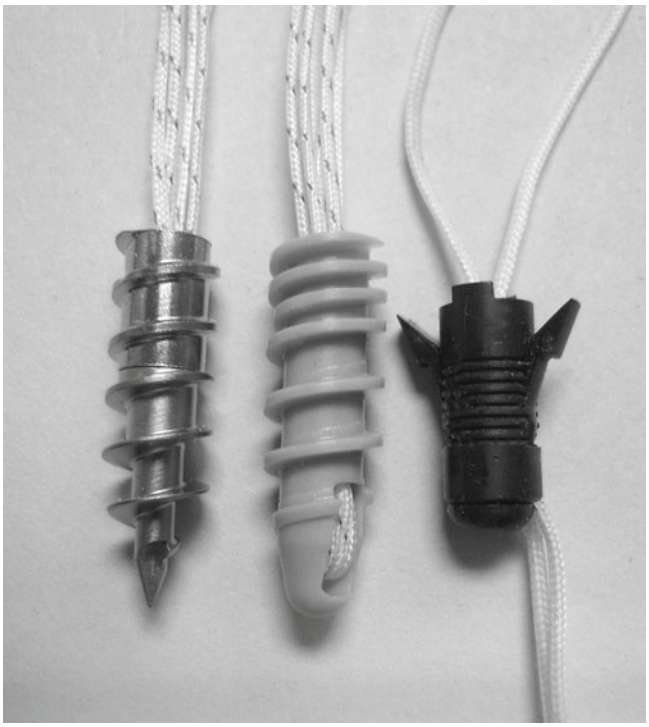

Fig. 12.12 Linvatec anchors are made of PEEK and titanium. Shown here *from left to right* are the SuperRevo FT (triple loaded), CrossFT (PEEK), and the knotless PopLok made from PEEK (© F. Alan Barber MD reproduced with permission)

Anchor Placement

The principal challenge of all repair techniques is to secure the tissue (tendon or ligament) to the normal bone attachment without more tension than is appropriate for physiologic healing. Repair concepts for the rotator cuff which have facilitated this goal include an appreciation of margin

convergence, the interval slide, Deadman angle, and variations in anchor location. Several options exist for placing anchors to achieve footprint attachment. These include locating the anchors at the edge of the articular humeral cartilage to reduce the tension on the tendon, locating the anchors more lateral to the articular cartilage but still on top of the tuberosity, locating the anchors on the lateral side of the humeral shaft (sometimes referred to as orthogonal) in the cortical bone while using a tension band or suture bridge to compress the cuff tendon to the prepared greater tuberosity site, or using a double row of anchors (one fixing the tendon near the articular cartilage and the second placed laterally fixing the rest of the rotator cuff tendon across the full extent of the normal cuff footprint).

A fundamental aspect of using suture anchors is the direction in which the anchor is inserted into the bone. This angle of insertion is especially important for rotator cuff repairs. The humeral head adjacent to a chronic cuff tear may undergo osteoporotic change and have an osteoporotic ("hollow") humeral head with fewer trabeculae than normal. The common term for the angle of insertion is the Deadman angle [17, 18]. However, the 45° angle should be the maximum acceptable angle for insertion rather than the ideal! More acute insertion angles often result in superior anchor placement because the anchor enters the denser subchondral bone with the more tangential angle. This provides better security for the rotator cuff repair. The depth of anchor insertion is also important as research has shown that those inserted deep fail by suture cutting through the bone or they displace by rotating and translating toward the cortical surface, whereas those inserted proud were associated with failure at the eyelet [19, 20].

Another concept relating to the effectiveness of anchor performance is the general bone density of the insertion site. Bone density varies with the location on the greater tuberosity. Tingart et al. have shown that the anterior area of the greater tuberosity is denser than the posterior area, and therefore, load to failure can be different in these two areas [21]. Better security is achieved if suture anchors are placed in the proximal-anterior and middle portions of the greater tuberosity to avoid increased anchor failure in areas of poor bone density [21]. Bone mineral density is a predictor of initial strength and final gap opening and pullout strength for all types of suture anchors. Therefore as predicted, studies have confirmed that gapping is more common over the posterior aspect of the tuberosity compared to the anterior aspect [22, 23]. Likewise, anchors fail earlier in older bone as compared to younger bone due to osteoporosis [24].

Failure Mechanisms

Failure of a surgical repair is multifaceted and certainly frustrating. Failures can occur from biological or mechanical causes. Biological failure causes include blocks to tissue healing from smoking, metabolic disorders, malnutrition, and vascular disease. Biomechanical failure can occur because of anchor failure (anchor pullout, breakage, eyelet failure), suture failure (breaking or knot slipping), or tissue failure (suture cutting out). Biological failure is both complex and beyond the scope of this chapter. This discussion will focus on the mechanical causes.

In general, repair construct failure can occur at the tissue-suture interface, the suture (slipping or breaking), suture-anchor interface, or at the anchor-bone interface (anchor pulling out, anchor breaking, or anchor moving in the bone). However, it must be remembered that clinically, the principal mode of repair failure for rotator cuff repair is at the suture-tendon interface.

Anchor Pullout

Modern anchors are exceedingly strong with respect to resisting pullout. Anchor pullout strength is a function of the contact surface area between the bone and the anchor and the resultant resistant frictional forces from the bone-anchor interface. Thus, the greater the anchor surface area, or the more resistant to failure the bone is at this interface (denser bone), the higher the forces needed to cause anchor pullout. Biomechanical studies consistently show that glenoid anchors exhibit lower pullout strengths than rotator cuff anchors. This is due in part to the cuff anchors often being larger, with deeper threads compared to glenoid anchors. Interestingly, the larger treaded rotator cuff anchors (i.e., 5.5 or 6.5 mm) do not demonstrate a significant difference in their failure loads [25, 26]. It also seems that a fully threaded design rather than absolute anchor size is the more critical factor in anchor pullout strength.

Bone density and anchor location also play a role. Another study showed that a 50 % increase in trabecular bone density resulted in a 53 % increase in pullout strength [27]. Both bone density and anchor configuration can play a part in increasing pullout strength; however, as mentioned previously, anchor pullout is seldom the mode of failure.

Anchor Breakage

Anchor breakage which occurs during insertion is frustrating to any surgeon attempting a repair. However, once implanted the anchor rarely breaks. Anecdotally, we have experienced and have heard other surgeons experience anchor breakage during implantation. Several explanations exist for anchor breakage during installation. The principal issue is the anchor material. Biodegradable anchors are clearly more likely to break than metal or PEEK anchors. Misaligning the anchor during the insertion into a drilled hole results in the loss of orientation and the increased likelihood that the anchor will

break during insertion. Poor visualization due to inadequate exposure or bleeding can also result in insertional anchor breaking. Attempting to put an anchor into bone without a predrilled hole can result in both the anchor breaking and damage to the adjacent bone and soft tissues. For anchors that are screwed into place, failure to tap a hole can result in increased sheer stresses and torque on the anchor, especially in very hard bone, which can result in breakage. The result may be an anchor that is only partially inserted and any attempt to advance the anchor results in its breaking. Careful attention to technique and increased surgeon experience with a particular anchor system can help mitigate these mishaps.

Anchor Eyelet Failure

The anchor eyelet allows the sutures to pass through and attach to the anchor. Various eyelet designs exist. The classic proximal post eyelet is being replaced with newer designs including eyelets located in the main anchor body or a distal crossbar reached through a hollow central anchor core. Some anchors offer an internal independent eyelet that allows the suture to reside completely within the anchor body. Each design has its own strengths and weaknesses. Barber et al. [25] showed that the distal crossbar eyelet fails consistently at the crossbar protecting the anchor body from pulling out of the bone. In that same study they showed that for the biodegradable anchors tested, eyelet failure was the predominant mode of failure. The rise of anchor eyelet failure may be in part due to the increasing strength of newer generation sutures.

Suture Breakage

The introduction of ultrahigh molecular weight polyethylene (UHMWPE)-containing sutures was a watershed event resulting in a significant change in cuff repair strength and suture performance. Any biomechanical comparison of load to failure testing using UHMWPE sutures with older studies that did not use these high-strength sutures is comparing apples to oranges.

Suture breakage with today's products is usually iatrogenic. Despite the strength of newer generation sutures, poor surgical technique can weaken the suture leading to suture breakage during not tying or after anchor implantation. Clamping or abrading the suture can weaken it. Nicking the suture with a sharp instrument or suture passer will have an obvious effect on the suture's integrity. If the region of suture compromise is close to the anchor, then the surgeon must adjust the suture lengths and the knot choice to avoid suture breaking during knot tying. Sometimes the damage to the suture can go unnoticed until breakage occurs usually during knot tying. If the surgeon is able to identify a defect before this event, he still has a few options. If the anchor is loaded with multiple sutures, he may be able to dispose of the damaged suture and rely on the remaining suture. Additionally, with the appropriate anchor eyelet, the remaining suture can be used to shuttle a replacement to reload the anchor. Lastly an additional anchor can be used.

Suture abrasion has always been thought to be a cause of suture failure. Depth, angle, and rotation of anchor insertion can play a role in this. If a suture anchor is placed too deep, one of two failure methods can occur depending on the density of the bone. A deep anchor in good bone can result in suture wearing against the bone at the level of the cortex. Cycling of the suture over this bony edge leads to failure. Secondly, a deep anchor in poor bone can result in the suture wearing a channel through the adjacent cortex leading to anchor migration [19, 28].

The angle and rotation of the anchor are also critical. Ideal anchor insertion places the angle of the anchor so that the line of suture pull parallels the eyelet [29]. Angling the anchor so that it is not aligned with the pull of the suture causes the suture to lever and rub over the eyelet. Likewise rotation of the eyelet away from the suture puts strain on the suture as it levers over the eyelet opening. Both anchor angulation and rotation can lead to suture abrasion and failure. Careful attention to surgical technique in terms of suture handling and anchor placement decreases the chances of suture breakage.

Suture-Tendon Cutout

Suture cutout at the suture-tissue interface is the leading cause of failure for soft tissue repairs [23]. This is particularly true in older patients with chronic tears. Should suture cutout occur during the arthroscopic procedure, the tissue should be carefully assessed before a second attempt at suture passage is made in the same location. Utilizing a "rip-stop" stitch or selecting a different site may be required. If the tissue was thought to be of good quality, the surgeon should evaluate the technical aspects of how the suture was passed. For instance, maceration of high quality tissue can occur if too many attempts are made at suture passage. Evaluate carefully where you would like your suture and place it therewith one pass. The size of your suture passing device may also pay a role. Intuitively, rotator cuff repair quality is negatively affected by suture passing devices that create large holes in the cuff [30]. Shoulder position postoperatively can also have an effect on suture-tendon cutout. Excessive tension on the repair before healing completely puts the repair at risk. Internal rotation will tension posterior structures, while external rotation tensions anterior structures. Rotational movement can uncover the footprint of a

rotator cuff repair and should be guarded against. A careful assessment of the anatomy being repaired should be coupled with appropriate postoperative joint position to avoid unneeded stress on the repair.

Repair Types: Single Row Versus Double Row

A controversy currently exists concerning the conflicting approaches of firmly fixing the cuff footprint (using a double row with a suture bridge) or attaching the cuff tendon and providing marrow vents to encourage the extension of the tendon during the subsequent healing over the remainder of the greater tuberosity (single row and microfracture of the greater tuberosity). How these two rows of anchors function and share the load is important.

Khoury et al. recently reported that medial row anchors are subjected to two-thirds of the total stress of a double row cuff repair. Meanwhile, the lateral row anchors in a suture bridge see only 33 % of that load [31]. This 2 to 1 loading ratio of the medial row may result in the tendency of the medial row to fail first both clinically [32] and biomechanically [33]. Since the medial row sees the stress first and is first to fail, it seems prudent that these anchors be as strong as possible. A construct which achieves greater lateral fixation strength makes failure more problematic. Voigt recently reported that 13 % of all suture bridge cuff repairs demonstrated medially ruptured tendons with a healed footprint [34]. Ruptures at the musculotendinous junction leave very few options for a revision [34].

It should be remembered that a suture bridge technique uses knotless lateral anchors. The associated UHMWPE-containing sutures tend to elongate and are more likely to slip than previously tested and used braided polyester sutures [7]. A knotless lateral row anchor which does not rely on a knot for security must have a very effective suture-locking mechanism when used in conjunction with UHMWPE suture.

What are the arguments for using suture bridging constructs? Larger, stronger suture constructs (Fiber-Tape, Arthrex; Fiber-Chain, Arthrex) [35] may perform differently than "classic double row" constructs and triple loaded constructs. While certainly stronger, these tissue crossing larger sutures may place pressure over a larger area of an already degenerative cuff tendon and potentially compromises the vascular supply to some areas of the tendon. Could these crossing sutures actually strangulate the tissue? Additionally, increasing the number of UHMWPE-containing sutures also increases the repair strength until eventually the weakest link in the repair is the tissue and not the suture. Introducing more and more suture material into a repair may reach the point where the increased strength is irrelevant and quite possibly the physiology of healing will be impaired.

There is increasing recognition that holding the cuff tendon footprint fixed over a greater area leads to repair failure at the musculotendinous junction in rotator cuff muscles with less fatty degeneration or muscle atrophy. Cho described two different cuff repair failure modes: Cho type 1 (failure at the original repair site) and Cho type 2 (failure around the medial row) [36, 37]. As noted, failure at the musculotendinous junction was previously described by Voigt et al. [34]. It is of special concern that this Cho type 2 failure (musculotendinous junction tear) occurred with double row repairs in 59 % of Cho's failure cases. It is also interesting that the percentage of the Cho type 1 retears increased with the severity of fatty degeneration or muscle atrophy. This suggests that the healthier tissue may be more likely to tear in the more catastrophic Cho type 2 manner (at the musculotendinous junction).

Summary

Advances in sutures and suture anchors offer improved techniques for arthroscopic glenohumeral instability surgery and arthroscopic rotator cuff repair. Metallic anchors have been mainly replaced by nonmetallic anchors including bioabsorbable, the newer biocomposite, and PEEK anchors. The biocompatible absorbable anchors are just as strong and durable as the metallic and plastic anchors and facilitate easier postoperative imaging and revision surgery. Any suture anchor selected should maintain the soft tissue in close enough proximity to the bone until natural biologic healing of the tissue to the bone has occurred. The larger cuff anchors tolerate higher loads, can hold more sutures, and work better in osteoporotic bone of the greater tuberosity than the smaller anchors designed for glenoid fixation.

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Arthroscopic Suture Management

13

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Shoulder arthroscopy is a complex surgery, and its success depends on a wide range of seemingly minute details. The correct position of the hands and proper utilization of all available instruments is a good starting point.

Suture management is fundamental because it is easy to find oneself with many sutures in a very small space without being able to distinguish between the two strands of the same suture or without knowing which of the sutures to knot first.

The sutures can be passed through the tissues in different ways, depending on the type of lesion and surgical procedure to be performed, which determine the type of instrument that will be utilized. Suture passages can be performed with direct and indirect techniques. Direct suture passages are further subdivided into antegrade and retrograde. Direct passage occurs when the sutures are passed directly in the tendon whereas indirect passage requires a suture shuttle. To correctly manage any type of suture, specific instrumentation is necessary, which consists of the following tools: operative cannula (at least 8.0 mm), retrieval cannula (5.5 mm), direct antegrade suture passer, sharp-end suture passers with various angles of curvature for direct retrograde passages, series of suture hooks with different curvatures preloaded with a shuttle suture for indirect passages, suture grasper, and grasping forceps (see Chap. 7).

Before examining the specifics, it is important to note that the tips and techniques presented in this chapter represent the personal opinion of the authors and are based on their individual experiences. It is necessary, therefore, to consider variations to the techniques outlined below; these variations might be specifically designed for certain procedures or they might reflect the preferences of the individual surgeon.

We want to further highlight that reading this chapter is far from sufficient to really understand suture management. Rather, an extensive period of study will be needed. First and foremost, one must have a clear idea of the type of lesion to be treated and the best method to approach it (first step: have an idea!). Once the objective is fully understood, the second step involves careful and repeated observation of every single maneuver for all the techniques. As soon as we understand exactly what happens inside the operating field, the third step requires practical "hands-on" experience to acquire surgical skill ("I know what I want to do but how do I do it?"). This final step involves achieving full competence in cannula and portal management, the proper order of the various steps, as well as the correct way to hold and utilize the instruments (Box 13.1). A useful exercise to solidify what we have learned, besides practicing the maneuvers on an anatomical model, is to repeat each procedure step-by-step in one's mind or on a piece of paper. Now we are ready to begin!

Box 13.1: Tips and Tricks

In the hands of an expert, suture management seems natural and repetitive. In reality, very few precautions must be taken. We have nevertheless included some lessons learned from our experience:

- Using cannulas, especially for young and/or inexperienced surgeons, prevents the creation of false routes during the passage of the instruments and the interposition of soft tissues during the passage and retrieval of sutures.
- To prevent sutures from becoming entangled, the operative cannula must always be free of sutures that we have already passed or that do not have to pass in the tissue.
- If cannulas are not used for the operative and retrieval portals, instruments must be introduced and extracted with closed jaws to avoid creating additional paths.

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- When we want to retrieve a suture from a portal, we must always observe the anchor to be sure that it is only the suture limb that we want to retrieve that is sliding! If the sutures slide in the anchor, it means that we are retrieving the wrong limb. We must stop immediately to not run the risk of extracting the suture from the anchor completely and having it get lost. A little trick to avoid this problem is to block the loop with a hemostat; nevertheless, if one does not look away from the operating field, there will not be any need for the hemostat!
- When we retrieve the two limbs of a suture to knot them from another portal, it is advisable to retrieve one limb at a time, being careful to not unthread the suture from the anchor or from the tendon.
- The knots must be in contact with the repaired tissue and not with the bone. In the case of rotator cuff repairs, the knot is on the repaired tendon. In the case of instability, the knot must be on the capsular tissue, not on the glenoid. This avoids the risk of cartilaginous abrasion and irritating articular pops and catching.
- After knotting the sutures, do not cut the two ends too close to the knot because it could slacken.
- Never look at your hands while suturing; instruments in the operating field are our fingers!

Rotator Cuff Repair

Rotator cuff lesions can vary in terms of size, shape, retraction, and mobility; therefore, repair techniques also vary and depend on the characteristics of the lesion. Suture management will be described below with the difficulty level of the procedures increasing as we proceed.

One Anchor Preloaded with Two Sutures

Imagine a supraspinatus tendon tear that can be managed with only one anchor. In this case, we will use direct antegrade passages.

Position the arthroscope in the posterior portal, the operative cannula in the lateral portal, and the retrieval cannula in the anterosuperior portal. The anchor is placed, through a superolateral accessory portal near the cartilaginous superior edge of the humeral head, in the center of the lesion with a 45° angle relative to the direction of the tendon.

For correct suture management, it is fundamental to ascertain the proper direction of the sutures. Preloaded anchors generally have a laser marker that indicates the position of the eyelet through which the sutures are threaded. In this case, we will align the sutures in such a way that they are perpendicular to the edge of the lesion. The anchor is preloaded with two permanent high-strength braided sutures of different colors to easily distinguish one from the other (Fig. 13.1).

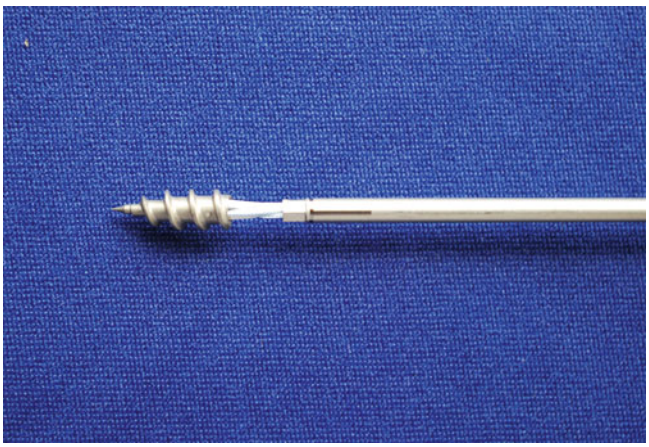


Fig. 13.1 Anchor is preloaded with two permanent high-strength braided sutures of different colors. Laser marker indicates the position of the eyelet through which sutures are threaded

Once the anchor has been positioned, we will pass the sutures through the tendons following a precise order: from anterior to posterior. We introduce the suture grasper in the operative cannula to take hold of the medial limb of the most anterior suture.

The suture grasper and the suture are extracted. Because the grasper takes hold of the suture near the anchor and because of the considerable length of the suture, the free limb of the suture that we have grasped will often still be in the superolateral portal even when the grasper has already been extracted from the joint, and we will find a loop in the lateral portal. Therefore, we have to know which end of the loop to pull to completely remove the suture limb. This is a particularly delicate task because if we pull the wrong end of the loop, the suture will be completely extracted from the anchor eyelet and it will be lost (Fig. 13.2). There are two ways to avoid this problem: tag the limb which should not be extracted with a hemostat or point the arthroscope towards the anchor eyelet and watch how the suture behaves while it is being extracted from the lateral portal. If the wrong end of the loop is pulled, the suture slides towards the anchor eyelet. On the other hand, if the suture does not move towards the anchor eyelet, it is an indication that the correct end of the loop is being pulled and, hence, that the suture limb will be correctly extracted.

Once the suture limb is removed from the lateral portal, we load the suture into a direct antegrade suture passer preloaded with a single-use needle, which will be used throughout the entire operation. We go back into the operative cannula with the passer closed and we pass the suture in the tendon. This instrument has jaws that enable an adequate and steady grasp, ensuring a constant distance between the free edge of the rotator cuff and the sutures (Fig. 13.3). To correctly pass the

sutures (from anterior to posterior), the suture passer jaws have to be directed towards the anterior edge of the tear.

After passing the suture through the tendon, we open the jaws of the suture passer and, at the same time, delicately remove it from the cannula with closed jaws. The suture strand is retrieved from the superolateral percutaneous portal, which enables all the suture limbs to be contained in the same portal rather than being dispersed in various directions in the field of view (Fig. 13.4).

At this point, we have to repeat the same sequence with the other strand but this time the suture passer jaws will be directed towards the posterior margin of the lesion. To achieve homogeneous tension throughout the lesion, the distance between the sutures has to be similar to the distance between each suture and the contiguous edge of the lesion.

After passing both the sutures in the lesion, we will close and knot the sutures following a specific order: from posterior to anterior!

Now we introduce the suture grasper in the operative cannula and retrieve the most posterior suture. After retrieving both limbs, it will be fundamental to ascertain which is the "post" and which is the "loop." The post is the limb around which the knot will be tied. The loop is the limb that will be tied around the post during the execution of the knot. In order to achieve the greatest tension and proper attachment of the

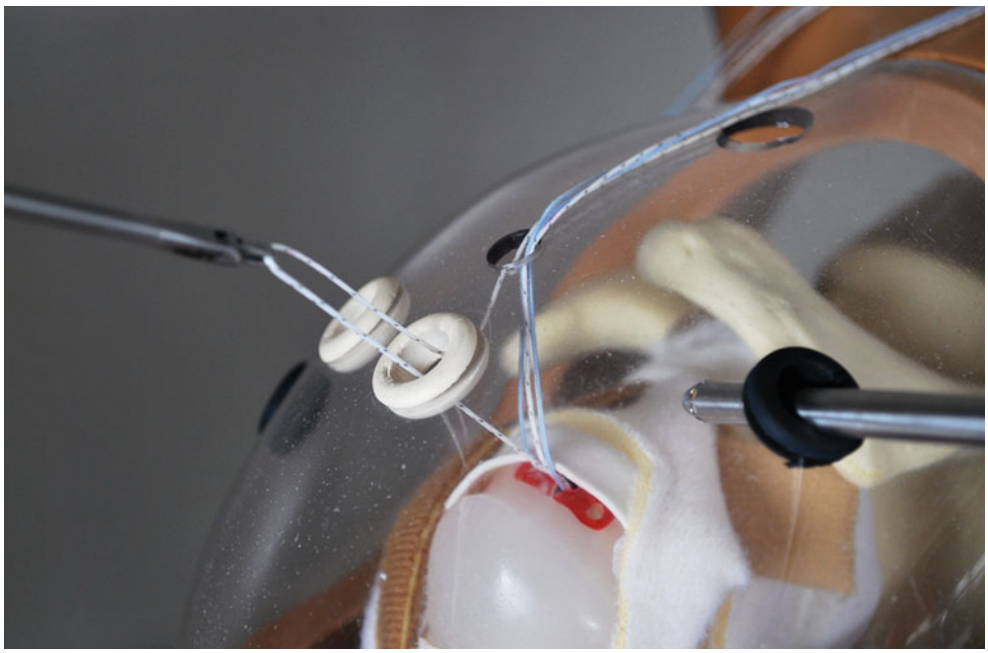


Fig. 13.2 The medial strand of the suture is retrieved from lateral portal using a grasper. It is important not remove the suture from the anchor eyelet

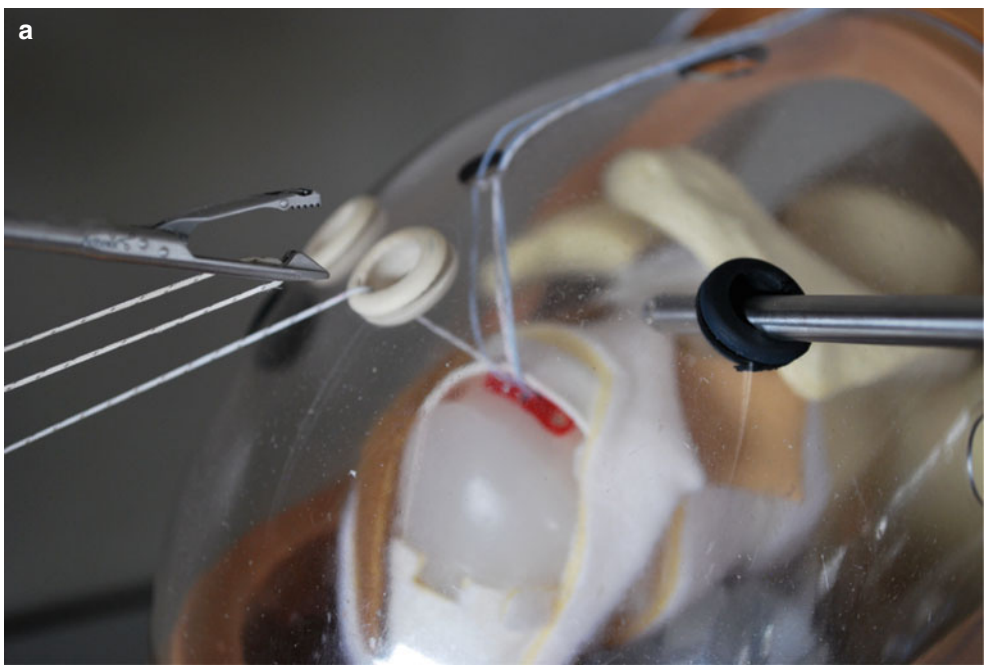
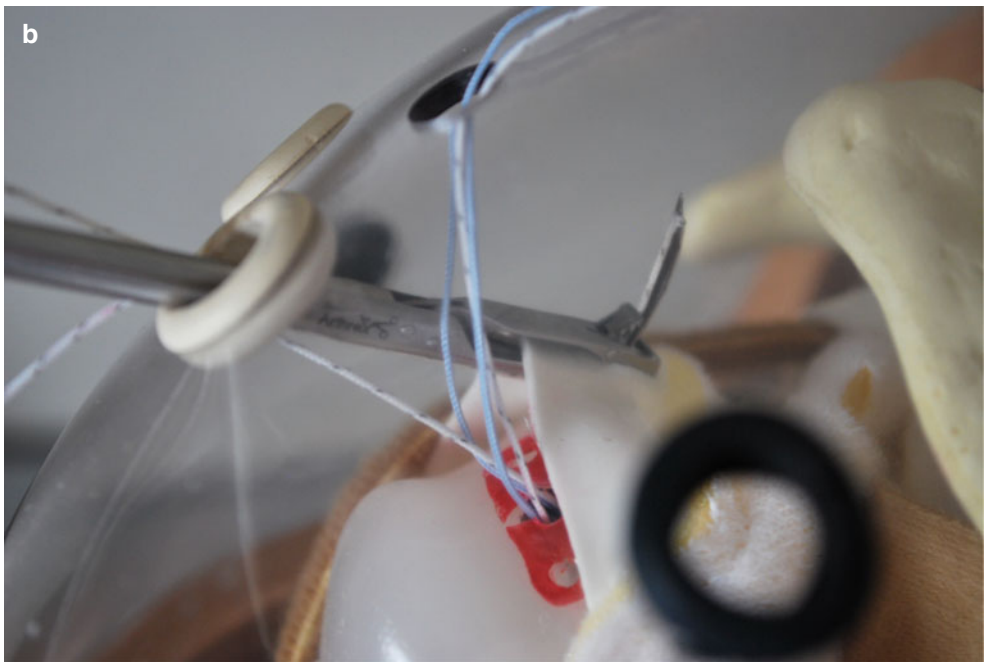


Fig. 13.3 Direct suture passer for anterograde passages. (**a**) The suture is loaded on the passer's bite. (**b**) The preloaded suture is passed through the tendon

Fig. 13.3 (continued)



tendon to the bone during knot tying, the limb that passed in the tendon is used as the post (see Chap. 14).

There are two ways to clearly differentiate between the two limbs:

- Gently slide one of the two limbs.
- Use a knot pusher: identify the post by pulling one of the two limbs, insert the post in the knot pusher, and advance the knot pusher in the cannula until it reaches the lesion, avoiding any twisting of the sutures (Fig. 13.5).

The only remaining steps are to evaluate the suture's ability to slide in the tissue and to choose the most appropriate knot (see Chap. 14). After tying the knot, a cutter is utilized to cut the suture limbs. We introduce a suture grasper in the operative cannula, retrieve the most anterior suture, and then repeat the same steps. The lesion is repaired.

Now we are ready to remove the operative cannula from the lateral portal and introduce the arthroscope in this portal. This allows us to assess the repair and the distribution of the sutures with an "en face" view. We insert the switching stick into the anterosuperior portal in order to evaluate the tension of the repair (see Chap. 23).

In the example described above, we used the lateral portal to knot both sutures. Nevertheless, there is another precise rule: the sutures have to follow the lines of force of the lesion to make the repair as anatomic as possible; therefore, based on the lines of force of the lesion (i.e., a posterosuperior lesion), it is not uncommon to knot the most posterior sutures from the anterosuperior portal and the superior sutures from the lateral portal. This case is described in the subsequent paragraph.

Two Anchors Preloaded with Two Sutures Each

Imagine a full thickness lesion of the supraspinatus and infraspinatus tendons of the left shoulder. For the most posterior sutures, it would be difficult to use a direct antegrade suture passer in the lateral portal, maintaining the arthroscope in the posterior portal, because the field of view would be too close to the operating field. This would result in a rather compromised view in terms of width, depth, and perception of real proportions. In this case, alternative solutions can be adopted, such as moving the arthroscope to the anterosuperior portal, moving the arthroscope to the lateral portal and the operative cannula to the anterosuperior portal, or moving the arthroscope to the lateral portal and using the posterior portal as the operating portal to execute either indirect sutures (with a suture hook) or direct retrograde sutures. Of all these options, we have found the last one to be the easiest, fastest, and most reliable; in our experience, it is the solution that is most commonly utilized.

We insert the arthroscope in the posterior portal, the operative cannula in the lateral portal, and the retrieval cannula in the anterosuperior portal. The anchors are placed from the superolateral portal, as previously described. In this case, however, we are repairing a larger lesion. Therefore, we will use more than one anchor. Anchor placement will follow the same order as that outlined for the sutures in the previous scenario: from anterior to posterior.

We place the first anchor near the cartilaginous edge close to the anterior edge of the tear and, like in the previous

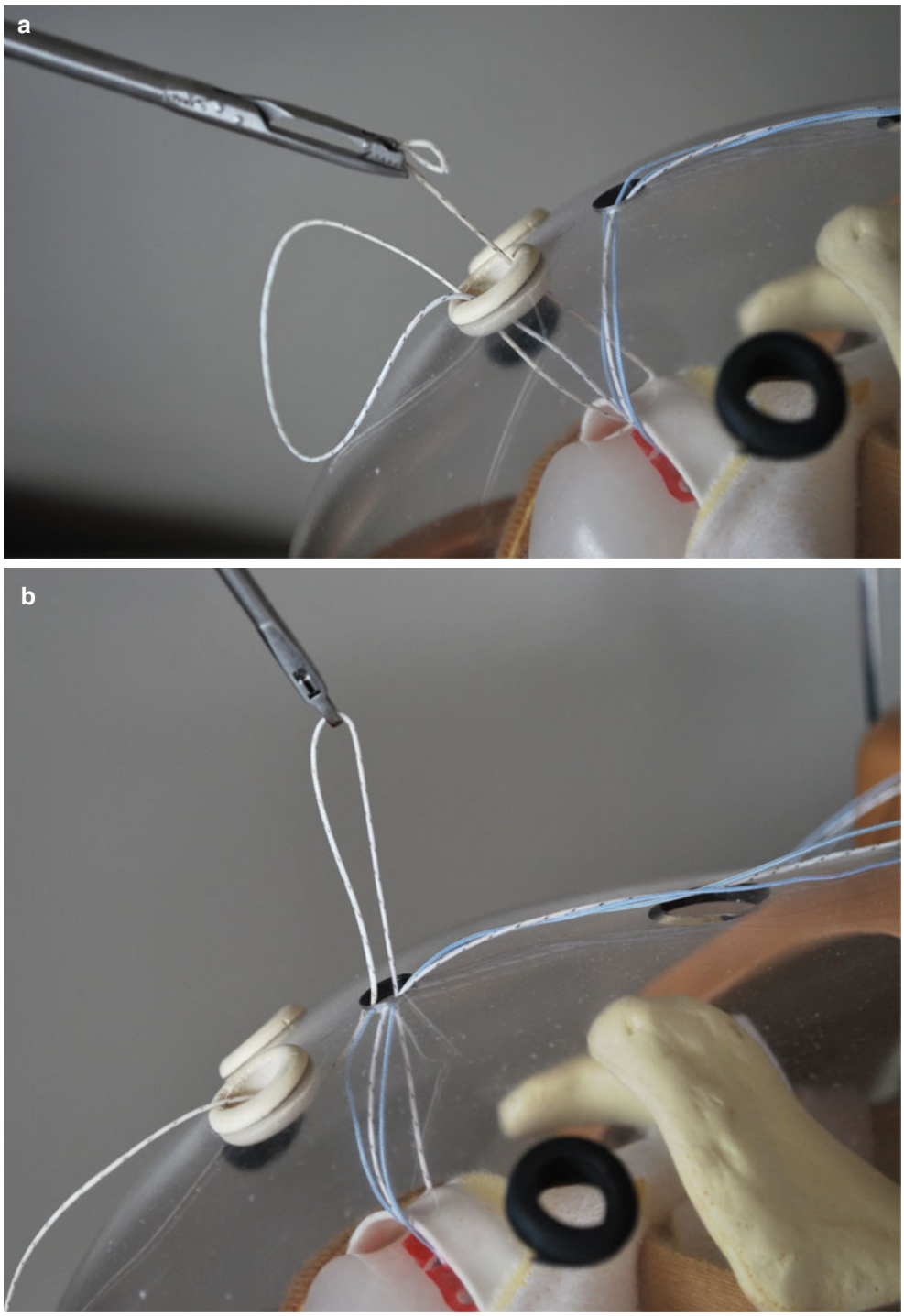


Fig. 13.4 Direct suture passer for anterograde passages. (**a**) Suture passer is delicately removed from the cannula with closed jaws. (**b**) Suture strand is recovered from the superolateral portal

scenario, at a 45° angle relative to the direction of the tendon.

We pass the suture passer in the same order as seen previously. In this case, the most anterior suture will be passed in the most anterior portion of the lesion, and the second suture will be passed in the most posterior portion. How posterior? Like the previous case, we need to consider that the sutures (4 total, assuming 2 anchors) have to be distributed along the anteroposterior length in such a way as to be equidistant from each other. Therefore, if the perception of the size and shape of the lesion is not reliable with the view from the posterior portal, it is preferable to take a look from the lateral portal before starting to repair the lesion. This additional perspective enables us to more effectively assess the shape and size of the tear. Furthermore, using the grasper from the posterior and anterosuperior portals, we can test reducibility and

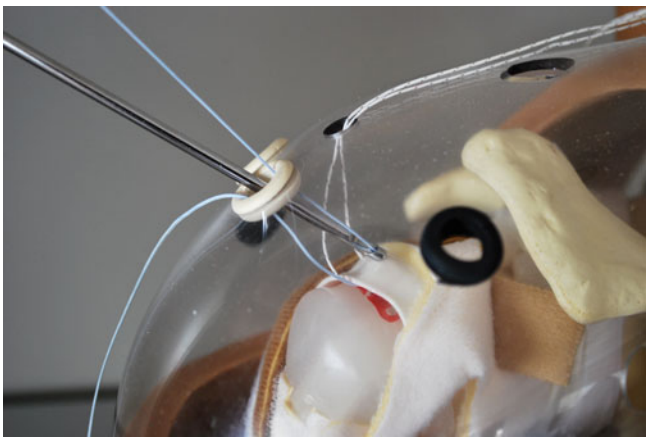


Fig. 13.5 A knot pusher is used to identify the post by pulling one of the two limbs of the suture and to avoid any twisting of the sutures

the direction of the force vectors (ideal segment between the point of anchor insertion and the point in which the suture is passed in the tendon), which is most effective to achieve a complete repair with minimal and homogeneous tension along the entire lesion (see Chap. 23).

After passing the sutures of the first anchor using a direct antegrade suture passer, the four suture limbs of the two sutures come out of the superolateral portal (see previous paragraph).

Before positioning the second anchor, we utilize a hemostat to block the four suture limbs of the first anchor. This will enable us to distinguish between the sutures of the first anchor and those of the second anchor when the moment arrives to tie the knots (after the placement of the second anchor).

To place the second anchor, the arthroscope is maintained posteriorly. The anchor must be positioned near the posterior edge of the lesion. If the lesion is too far posterior, it could be difficult to identify the correct placement; therefore, the assistant will help by reducing arm elevation and internally rotating the limb to expose the exact point in which the anchor should be placed. Alternatively, a grasper can be used from the anterosuperior portal to elevate the posterior edge of the rotator cuff in order to distance it from the field of view and from the point in which we intend to insert the second anchor. Or yet another alternative would be to move the arthroscope to the lateral or anterosuperior portal.

Once the anchor has been placed, we must pass the sutures. We follow the same order: we will pass the most anterior suture through the tissue followed by the most posterior suture.

Since we must work on the posterior rotator cuff, we will utilize the direct retrograde suturing method. Hence, we position the arthroscope in the lateral portal and we use the posterior portal as the operating portal.

We are visualizing the lesion en face. We introduce a grasper in the retrieval (anterosuperior) portal that will help us to elevate the posterior rotator cuff to understand where and how to pass the sutures; a direct retrograde suture passer is inserted into the posterior portal. We use a suture passer with sharp tip, which facilitates passage of the instrument through the tissue, and with a loop at the end, which allows for atraumatic retrieval of the suture. It is important to remember that posterior rotator cuff lesions are often delaminated, so we must remember to include all the layers in the suture (see Chap. 23).

We must pass the first suture in the most central part of the lesion, maintaining a constant distance between the various sutures, as well as between the outermost sutures (anterior and posterior) and the edges of the lesion.

Before proceeding, we use the grasper to reduce the tear and check where we passed the second suture of the first anchor, and we pass the suture passer through the tendon from the bursal to the articular side. If there are additional layers, the grasping forceps will help us to distinguish between them and to pass the suture passer through all the layers. The sharp tip of the suture passer punctures the tendon; we push the suture passer delicately in the rotator cuff with small, semicircular movements of the wrist until we have reached the articular side, and we are able to open its jaws. We direct the pointed tip of the suture passer towards the second anchor. With the grasper, we select the medial limb of the most anterior suture, open the suture passer jaws, retrieve the suture, and then repeat (in inverted order) the same movements of the wrist to enable the suture to pass through the tissue by retrograde passage until it comes out the posterior portal (Fig. 13.6). As we mentioned earlier, it is important to not remove the suture from the anchor eyelet during this step!

We repeat the same procedure for the second suture, remembering that it has to pass through the area corresponding to the posterior edge of the tear.

As previously highlighted, we close our sutures in accordance with the lines of force of the lesion and following a precise order: from posterior to anterior.

We introduce the suture grasper in the anterosuperior portal and retrieve the two limbs (one at a time) of the most posterior suture. The lesion is posterior, and therefore, it is correct to exert traction from the anterosuperior portal (Fig. 13.7). At this point, the remaining steps include distinguishing between the sutures, evaluating how they slide in the tissue, and selecting the most appropriate knot. After tying the knot, we must cut the two suture limbs with a cutter and proceed to the second suture. Because we are still in the posterior area, we will tie the knot from the anterosuperior portal.

The sutures of the posterior anchor are now tied.

Before proceeding with the sutures of the anterior anchor, we change the position of the arthroscope again because now we must tie the knot of the most anterior sutures. Therefore, we will achieve the best view looking through the

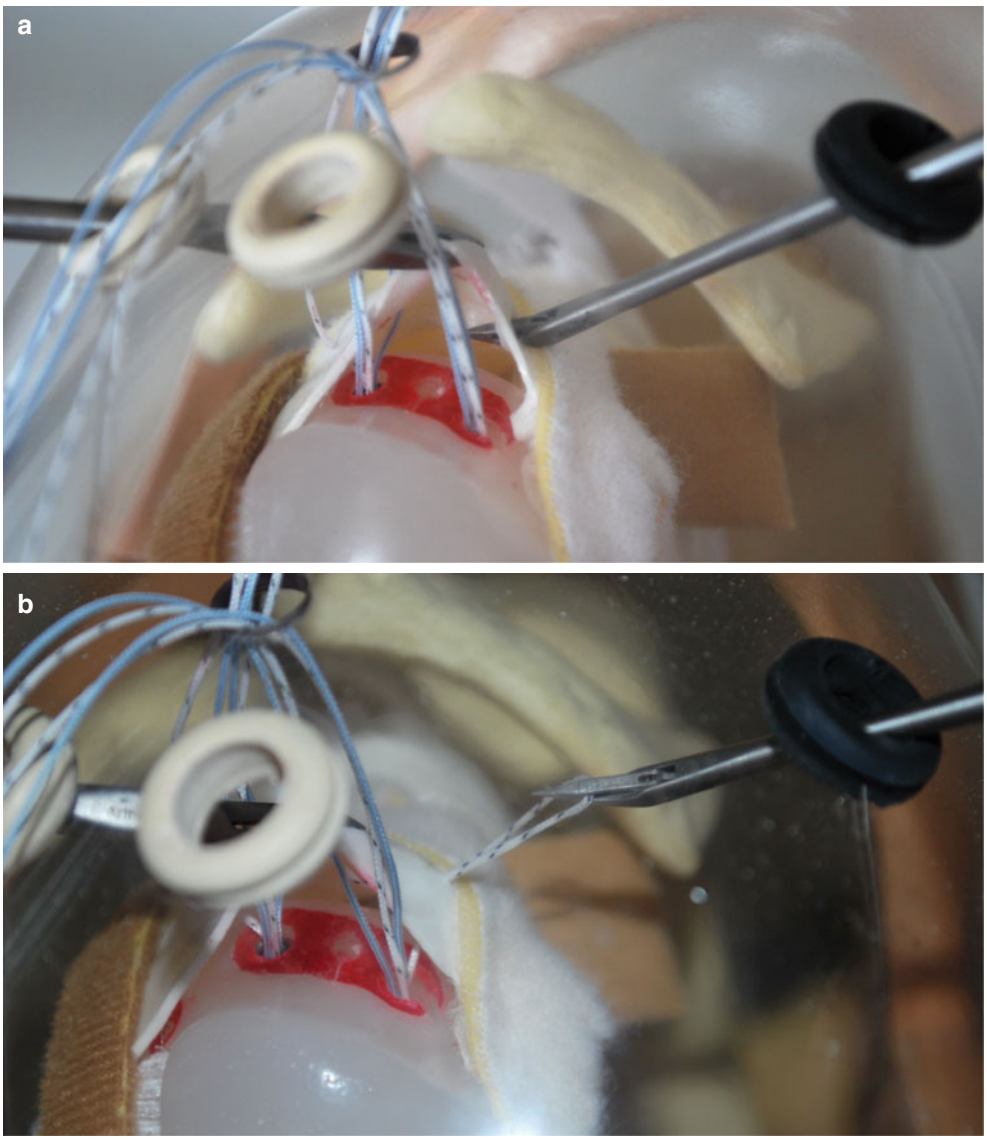


Fig. 13.6 After anchor placement, posterior suture is passed using a direct retrograde suture passer through the posterior portal. (**a**) The suture passer is delicately pushed through the rotator cuff from the bursal to the articular side. (**b**) The medial limb of the most anterior suture of the posterior anchor is recovered and passed through the tendon

posterior portal, but even more importantly, we will be able to use the lateral portal to knot the sutures, thereby respecting the lines of force of the superior portion of the lesion while knotting, from medial to lateral.

The arthroscope is then placed in the posterior portal and the operative cannula in the lateral portal. We must remember to remove the hemostat, which was used to isolate the sutures of the first anchor; we utilize the suture grasper to retrieve the sutures, as described previously.

Once these final sutures are knotted and cut, we move the arthroscope back to the lateral portal, and with the switching stick inserted in the anterosuperior portal, we evaluate the repair.

In summary, we placed the first anchor and passed its sutures; we positioned the second anchor and passed its sutures, and finally, we knotted all the sutures at the end. This method enables us to evenly distribute the tension of the repair on the sutures and anchors.

Thus far, we have only described suture management techniques which involve single-row repair. In reality, there are various techniques, such as double-row and transosseous repair, which can be used for rotator cuff repair. A detailed description of these alternatives is nevertheless beyond the scope of this chapter (see Chap. 23).

To correctly manage the sutures regardless of the repair technique, we need to have an outline in mind, which always includes the following steps:

- Evaluation of tear size and shape and selection of the most appropriate type of repair.
- Positioning the anterior and posterior anchors in such a way as to enable the sutures to be properly aligned for the repair selected.

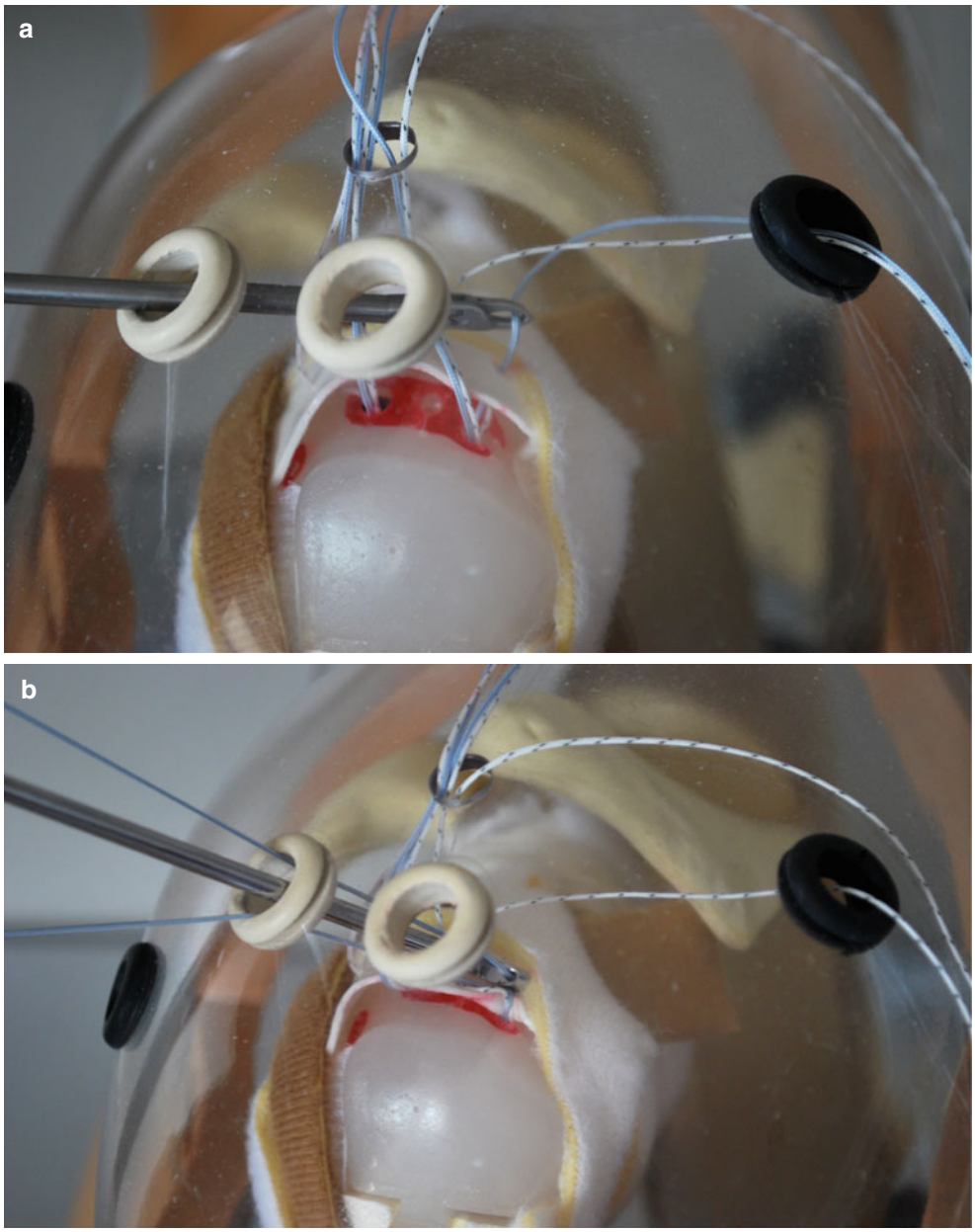


Fig. 13.7 (**a**) The most posterior sutures are retrieved from the anterosuperior portal. (**b**) The sutures are tied from the anterosuperior portal, according to the direction of the force vectors

- Passage of the sutures using the most effective method (direct or indirect) from anterior to posterior.
- Closure of the sutures according to the tear's lines of force, from the most posterior to the most anterior suture.

Generally speaking, the outline above can also be applied to larger lesions, which require the use of three anchors. One must consider that some surgeons prefer to utilize a sequence in which the positioning of the anchors precedes suture passage and knotting (outline for two anchors from anterior to posterior: anchors 1–2; sutures 1–4; knots 4–1). We prefer to pass the sutures of each anchor before positioning the subsequent anchor to make the procedure more orderly and to reduce the risk of confusing the sutures of the different anchors (outline for two anchors from anterior to posterior: anchor 1, sutures 1–2; anchor 2, sutures 3–4; knots 4–1).

Side-to-Side Repair

The shape of the lesion influences the type of repair. The objective is always to mobilize the lesion as much as possible and to follow its natural shape in order to execute a repair without tension.

There are various techniques to reduce the tension of a repair. For example, when repairing a tear with a longitudinal component (L or reverse L shaped), it is important to close this component with side-to-side sutures before proceeding to the positioning of the anchors. Furthermore, very large V- or U-shaped lesions can be reduced in size by positioning side-to-side sutures in the most medial area of the lesion to transform them into crescent-shaped lesions; in other words, the objective is to achieve a functional repair which reduces the area of the lesion as much as possible

(margin convergence technique). We will describe suture management during a rotator cuff repair with a side-to-side technique.

Imagine repairing an L-shaped rotator cuff lesion of the left shoulder. We will use a retrograde suturing approach. We position the arthroscope in the lateral portal and the operative cannula in the anterosuperior portal. We introduce a right-curved suture hook preloaded with a shuttling monofilament or wire loop in the operative cannula with the left hand and introduce a grasper in the posterior portal with the right hand. We mobilize the anterior edge of the rotator cuff tear with the grasper and identify (with precision) the apex of the tear. The hook must pass through the anterior edge of the tear at its apex.

The hook is maneuvered with small, circular movements of the wrist that augment the curvature of the instrument, allowing it to delicately pass through the depth of the rotator cuff.

Once the tip of the hook has crossed the entire depth of the rotator cuff from the bursal to the articular side, we let the shuttling loop slide. When there is a sufficient amount of the loop in the joint, we retrieve it with the grasper and bring it out through the posterior portal (retrieval portal).

We insert a braided, non-resorbable #2 suture (i.e., FiberWire) in the suture shuttle loop that we will use for the repair.

We remove the hook with delicate rotating movements of the wrist. Once the hook is extracted, we retrieve the suture shuttle.

We introduce the suture grasper in the anterosuperior portal and retrieve the other suture limb. In this way, we have cleared the posterior portal.

Now we must pass the suture in the posterior edge of the cuff tear. The following sequence must be executed: introduce the suture grasper in the anterosuperior portal, mobilize the posterior edge, and identify the apex of the lesion and the point in which we passed the suture on the anterior edge. Next we introduce a direct retrograde suture passer in the posterior portal and pass through the entire depth of the posterior edge of the rotator cuff at a more medial point relative to the passage previously executed on the anterior edge. The stitch will, therefore, have an oblique direction from medial to lateral, from anterior to posterior. This will ensure a repair along the line of force that will be more effective in reducing the tear.

With the help of the suture grasper, we isolate the suture limb that comes out from the articular side of the anterior edge of the lesion and grasp it with the suture passer; extract the suture passer allowing the suture to slide through the posterior edge from the articular side towards the bursal side. We must remember that delamination of the rotator cuff is generally posterior; therefore, we must be careful to pass the suture through all the layers (see Chap. 23).

Before closing the suture, we must understand how to do it; we must always follow the lines of force of the lesion. When repairing an L-shaped lesion, the larger anterior edge will likely be more mobile and will be closed on the posterior edge. Therefore, we can close the suture from the posterior portal. We introduce the suture grasper from the posterior portal and retrieve the anterior suture limb. Now the only remaining steps include distinguishing between the suture strands and knotting the sutures, remembering that the post must be the limb around which the knot is tied (the limb which passes through the tissue that we want to reduce during the knotting). In this case, the post will be the limb that passed in the anterior edge (Fig. 13.8).

If necessary, we can pass other sutures, repeating the same steps until a complete reduction of the lesion is achieved, resulting in a crescent shape, in order to manage the lesion with the anchors and with the least amount of tension.

As seen previously with the anchors, it is important to have a clear objective: reduce the tension. With this goal in mind, the side-to-side sutures will be passed from medial to lateral, from the apex towards the lateral edge of the lesion, and they will be knotted one after the other, giving us the ability to visually evaluate the result obtained each time (outline for two sutures from medial to lateral: suture 1, knot 1; suture 2, knot 2).

Arthroscopic Capsuloplasty (Bankart Repair): Three Anchors Loaded with a Single Suture

Imagine repairing an anteroinferior Bankart lesion of the left shoulder. We will manage the sutures using indirect passages.

Position the arthroscope in the posterior portal, the retrieval cannula in the anterosuperior portal, and the operative cannula in the anterior midglenoid portal.

Through the anterior portal, we position the first anchor in the most distal portion of the anterior edge of the glenoid. The anchor eyelet must be parallel to the edge of the glenoid so the suture will be oriented perpendicularly to the glenoid labrum being repaired. We introduce the suture grasper in the anterosuperior portal and retrieve the anterior suture limb (the limb that is found on the capsulolabral tissue side).

We introduce a left-curved suture hook, held in the right hand, in the operative cannula and execute a reduction and a distal-proximal shift of the capsulolabral tissue using a grasper, held in the left hand, introduced through the retrieval cannula. The assistant, in the meantime, will hold the arthroscope.

We maneuver the suture hook, as explained previously, so that it passes through the capsular tissue and below the glenoid labrum to retighten the shoulder capsule and to bring the glenoid labrum back into place. For a more detailed description, refer to the chapters dedicated to this procedure (Chaps. 15 and 16).

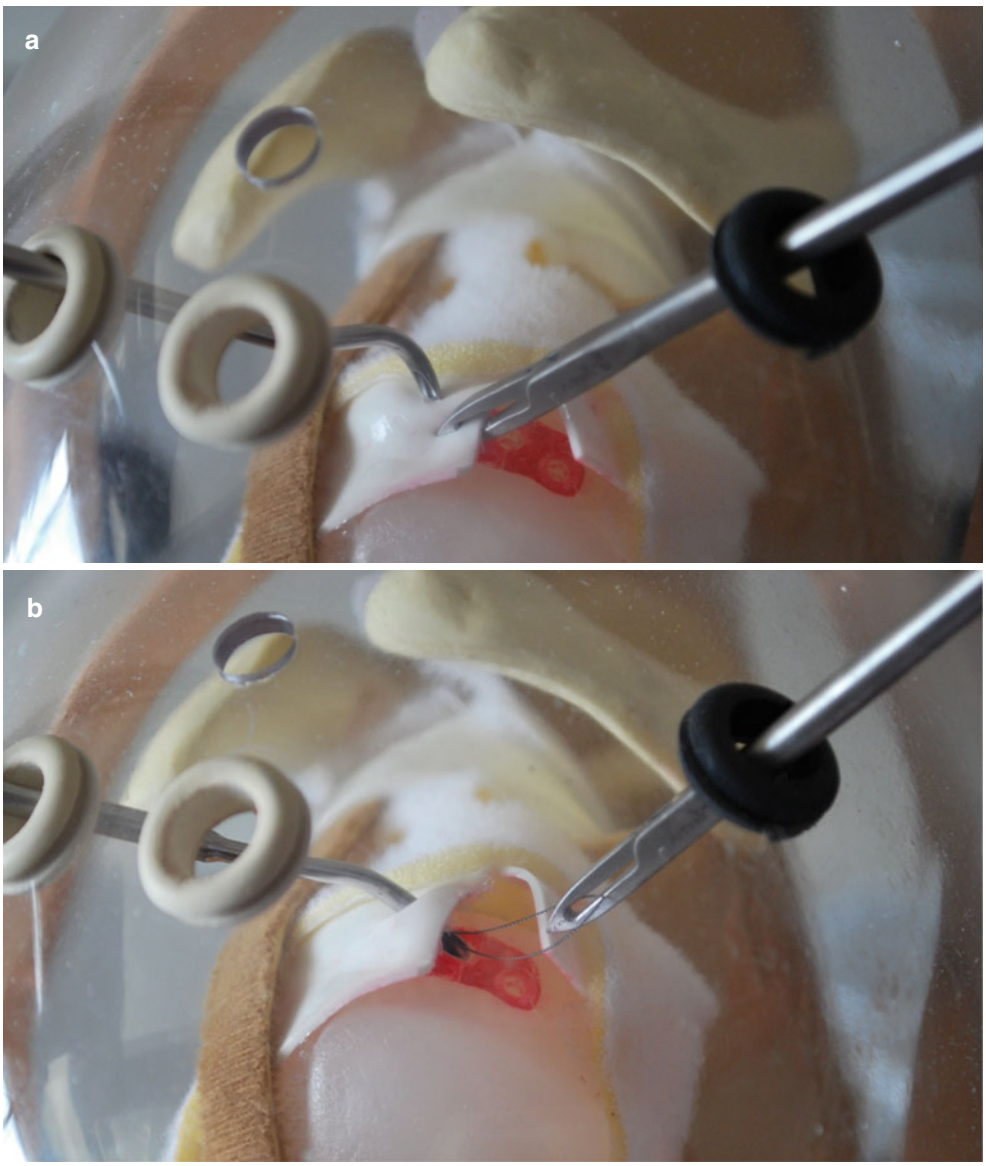


Fig. 13.8 The indirect suture passer is used to manage an L-shaped lesion of the left shoulder. (**a**) A right-curved suture hook preloaded with a monofilament is introduced in the anterosuperior portal and passed through the anterior edge of the tear from the bursal to the articular side. (**b**) The shuttle loop is retrieved from the posterior portal using a grasper. (**c**) A high-strength permanent suture is loaded in the shuttle loop. (**d**) Sutures are retrieved and tied from the posterior portal, according to the direction of the force vectors

After passing the suture hook, we allow the shuttling loop to slide ahead; we retrieve it with the suture grasper from the anterosuperior portal; we load the suture limb (that we just brought through the same portal) into the suture shuttle loop; we extract the suture hook, retrieve the suture shuttle and, hence, allow the suture to slide through the tissue in retrograde fashion (Fig. 13.9).

We have executed a simple suture. If we intend, rather, to pass mattress sutures, we must orient the anchor eyelet so that it is perpendicular to the anterior edge of the glenoid. The suture will then be parallel to the glenoid labrum. In this case, after passing the most distal suture limb, we retrieve the other limb of the same strand from the anterosuperior portal and repeat the same steps, being careful to pass the proximal limb 3–4 mm superior to the distal limb.

After all the sutures of the first anchor have been passed, the only remaining step is to knot the sutures from the anteroinferior portal.

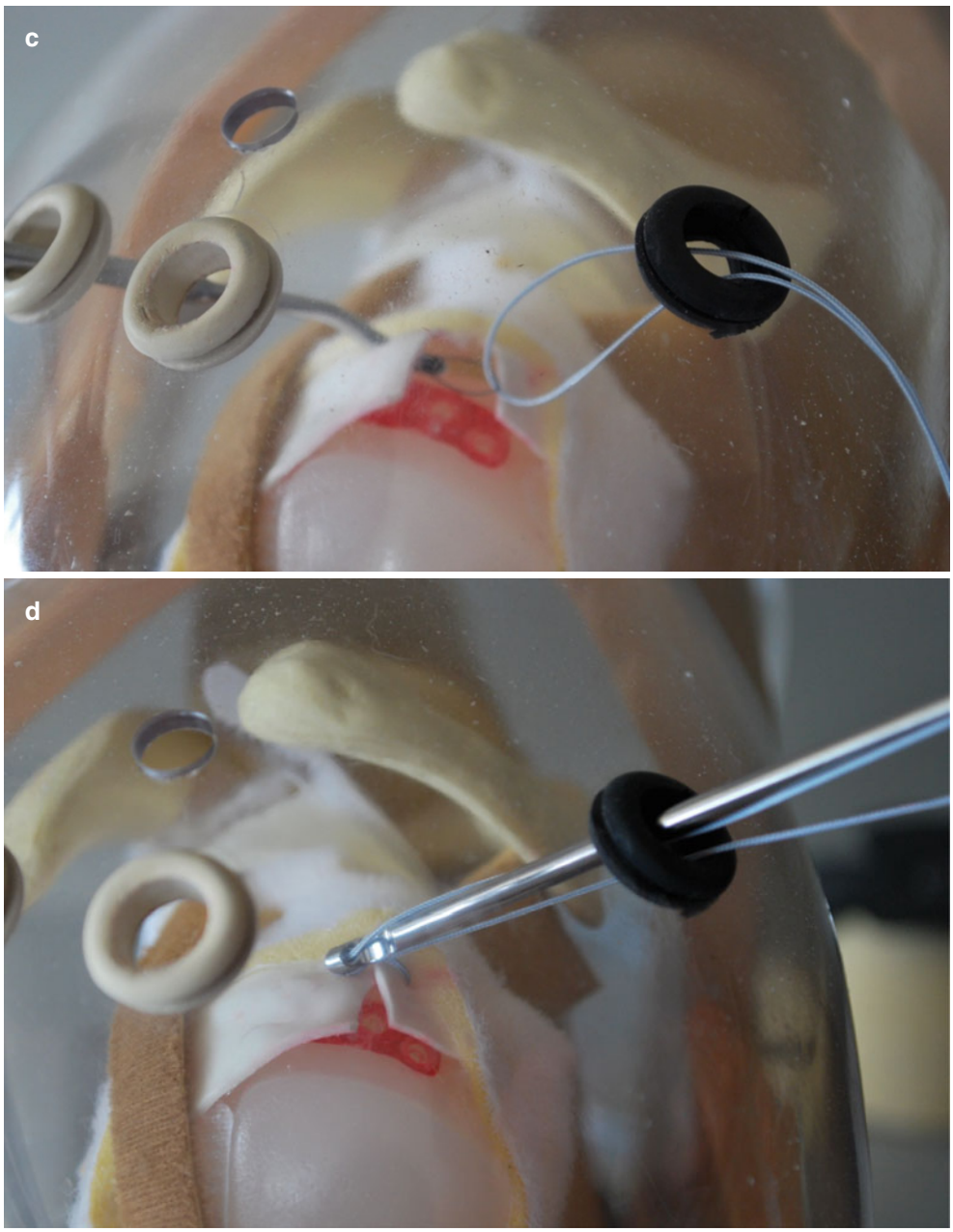
Once the sutures have been tied and cut, we introduce a switching stick in the anterosuperior portal to test the strength of the sutures and the result obtained.

Unlike what was seen in the rotator cuff repairs, the sutures are knotted each time before implanting the subsequent anchor. The subsequent anchors will be placed proximal to the first and the sutures will be managed in the same way.

Suture management during a Bankart repair establishes the following sequence: (outline for three anchors from distal to proximal: anchor 1, suture 1, knot 1; anchor 2, suture 2, knot 2; anchor 3, suture 3, knot 3).

Various surgical techniques and different types of anchors are utilized for this operation. If we decide to use knotless anchors, the suture management process would not change. In this case, we would pass the suture through the tissue first, like we have seen, and then we would implant the anchor.

Fig. 13.8 (continued)



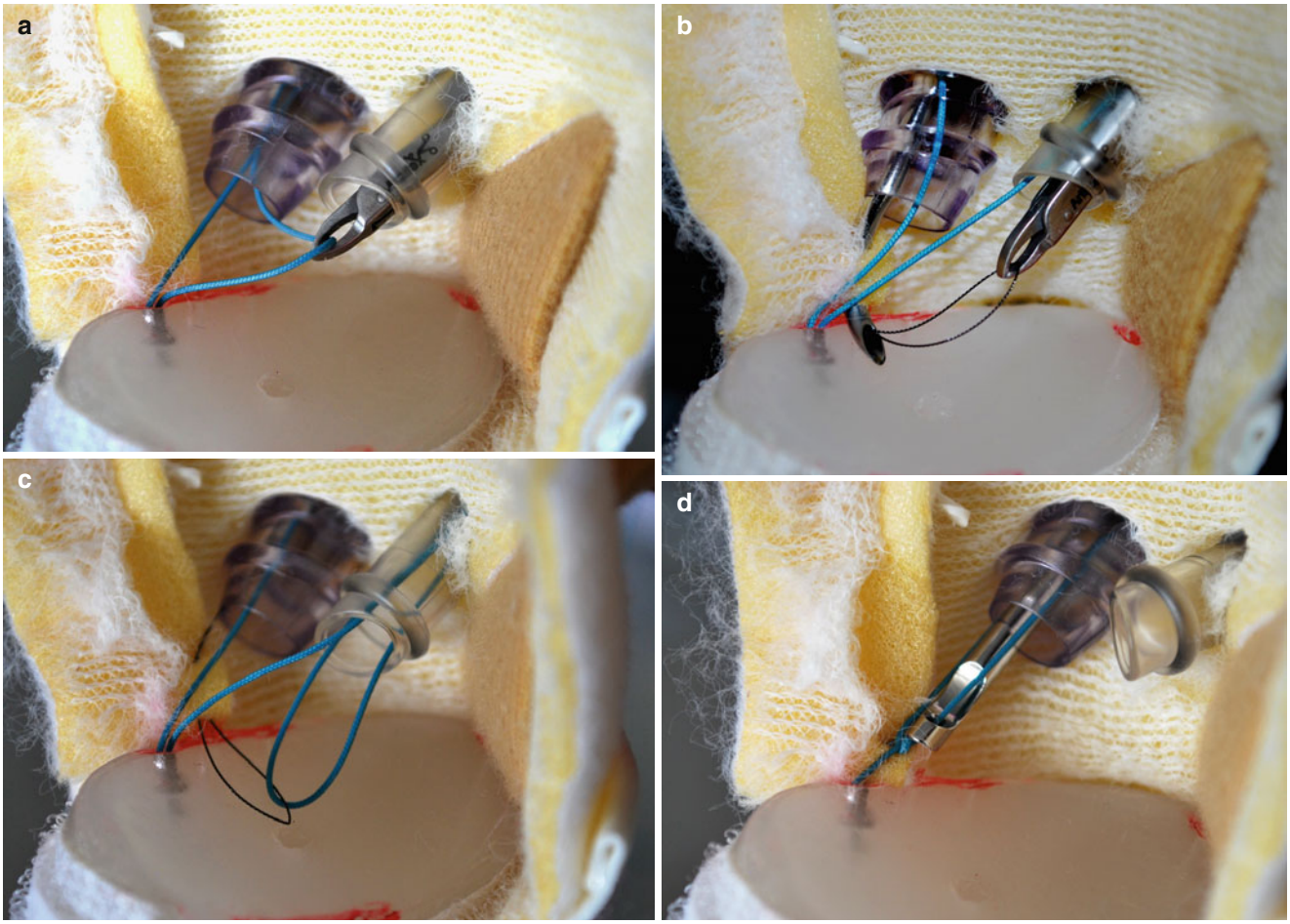


Fig. 13.9 Arthroscopic capsulolabral repair using an indirect suture passer. (**a**) The anterior suture limb of the suture anchor is retrieved through the anterosuperior portal. (**b**) A left-curved suture hook is passed through the capsular tissue and below the glenoid labrum. (**c**) The anterior suture limb is loaded in the shuttle loop of the suture hook. (**d**) Sutures are tied from the anterior midglenoid portal. The knot is tied over the capsular tissue

Arthroscopic Knot Tying

14

Jae-Chul Yoo and Young Eun Park

Introduction

Arthroscopy has evolved from a merely diagnostic procedure to a therapeutic procedure, with most reconstructive procedures being done by arthroscopy. Suturing has been the cornerstone of all reconstructive surgeries to hold the tissues together and without excessive tension to facilitate healing and knot tying has been practiced for centuries. Arthroscopic shoulder reconstructive procedures also employ sutures and knots, and every shoulder surgeon must understand the principle and learn the technique of arthroscopic knots. Though the principles of repair are the same, arthroscopic reconstruction is different from open techniques in that suturing is much more difficult in arthroscopic procedures. The surgeon has to suture tissues at a distance, under the constraint of cannula and in a wet field. This necessitates the use of knot pusher and placement of knot with asymmetric tensioning of suture limbs. Surgeon does not have the benefit of tactile feedback as in tying open knots. Furthermore, the knot is often tightened at an acute angle to suture and not perpendicular to defect, an ideal situation described in open knotting. Though squaring of knots with crossing the suture limbs and applying equal tension to both limbs has been described [1], it is difficult and the knots placed in arthroscopic surgeries tend to become a series of half hitches rather than square [2]. The difficulty is compounded by the fact that the tissues that are being sutured are not always of good quality.

Since the introduction of arthroscopic shoulder surgery, surgeons have been constantly seeking better and stronger knot-tying materials and methods to overcome these problems. Hence, there are many researches and innovations in suture materials, knot configurations, and techniques. Unfortunately this has also led to a proliferation of techniques and methods, and there seems to be much variation in preference for knot-tying configurations and suture methods that surgeons use. The aim of this chapter is to clarify the issue on arthroscopic knot placement with particular reference to shoulder surgery. The principles of knot placement and recent advances in knot configurations and suture materials will be discussed. There are numerous knot configurations and techniques that have been described, and it is beyond the scope of this chapter to discuss them all. We have chosen several knots that have been commonly used or described in the literature and will focus on these knots in detail.

Arthroscopically sutured knots in shoulder surgery can fail in several circumstances. Common scenarios are cut through of suture from the tissue, pull out of anchors from bone, breakage of suture material, knot slipping, and loosely placed knot that does not approximate the tissue. Though there is no objective data, loosening of knot that allows tissue separation of more than 3 mm is described as knot failure. This chapter mainly focuses on the knot-tying principles hoping to avoid or minimize the untying of a knot (knot security) and placing the knot without proper approximation of tissues (loop security).

Several studies [3–5] have pointed out important factors when considering knot tying: material properties, material tensile strength, coating of the suture material, friction between suture limbs, internal interference of the knot, the tension applied to make a tight knot, the knot configuration, slack between throws of the knot, the tying instruments including knot-pusher design, and surgeon's skill. Therefore, in tying an arthroscopic knot, it is essential to understand the following factors: (1) suture material factor, (2) knot pusher factor, (3) knot configuration factor, and, last but not the least, (4) the surgeon's factor.

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Terminology

Before we proceed further, we must be familiar with the relevant nomenclature that is commonly used in the knot-tying community. The following definitions will be used throughout this article:

- *Suture limbs*: When a suture is passed through tissue or an anchor, it creates two ends to be tied and these are the suture limbs.
- *Turn* refers to the number of twists in a given throw.
- *Throw* refers to a specific step or layer of the knot. A throw can either be underhand or overhand (Fig. 14.1).
- *Reversing throw* refers to alternating underhand with overhand and vice versa.
- *Post* (limb or strand): Of the two suture limbs, one limb is kept under tension and this limb is called the post limb. It is around this limb that the other limb will be wrapped. As discussed later in the section on knot pushers, post limb is the one under tension and not necessarily the limb on which the knot pusher is placed. The post is typically the limb that is away from the bone (or away from the center of the joint) and through the soft tissue. This allows the tissue edges to be approximated under tension; as the knot slides down the post, the tissue to be approximated will be pushed ahead of the knot (Fig. 14.2).
- *Wrapping* or *loop* limb (or strand) refers to a portion of the suture limb that one makes a loop around the post.
- *Post switching* or *reversing* is alternating the post limb for each successive loop.
- *Slack* refers to the loose configuration of loop or compound loop, which slides in around the post.
- *Half-hitch knot* describes the simplest of all the sliding knots, consisting of a single turn around the post limb making a loop. It can be both used in sliding or non-sliding conditions.
- The terms *proximal* and *distal* are in relation to the surgeon; portion of suture limb near to surgeon is proximal and away from him and near to tissues is distal.
- *Slipknot* refers to a knot that is formed by sliding the loop strand on the post strand.
- *Square throw* refers to a simple two throws, which makes square knot, with each throw having one turn. When tying a square knot, the strands are pulled down in a line, perpendicular to the axis of the knot.
- *Knot security* is the ability of knot to resist slippage (Fig. 14.3), and *loop security* is the ability of suture to hold the tissues together (Fig. 14.4). Slippage of more than 3 mm is conventionally regarded as failure.

Knot Configuration Symbols

Tera and Aberg [6] introduced standardized symbols for knots, and Trimbos [7] applied it to the slipknot with modification in 1984. Since then, there were slight variations among authors in use of such symbols as #, or // being same as //x. However, the most widely accepted and reasonable (codification) symbolization nomenclature seems to be the one Loutzenheiser et al. [4, 8] and Burkhart et al. [3, 9] used:

S refers to single sliding throw (half-hitch) knots.

= refers to identical throw, loop direction same around the same post.

x refers to nonidentical throw, loop reversed around the same post.

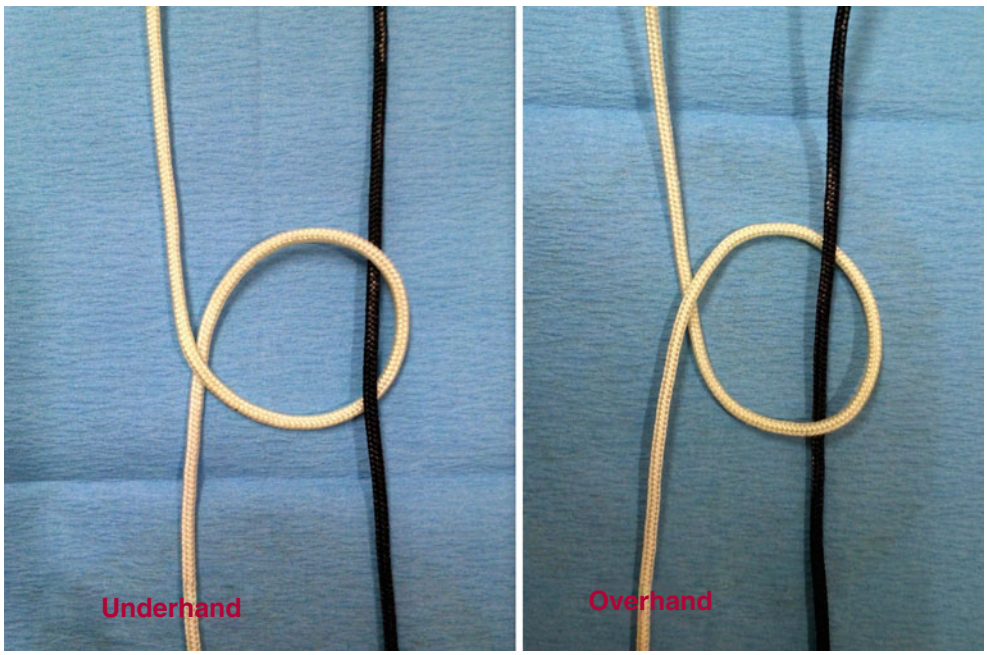

Fig. 14.1 In overhand throw, the loop is passed on the top of the post and below the post in underhand throw. Note that the loop (*white strand*) passes either over or under the post strand (*black*)

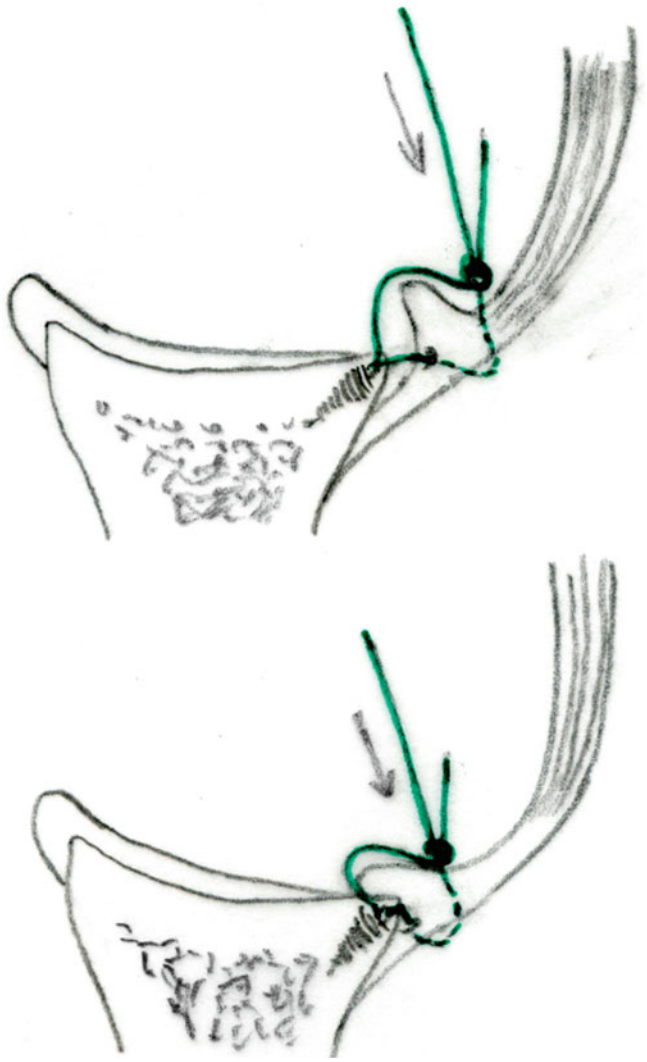

Fig. 14.2 The post limb is placed on the tissue side, so the tissue is pushed to the bone on tightening the knot

// refers to alternating post between throw, throws are identical.
//x refers to alternating post between throws, which are non-identical or reversed each time.

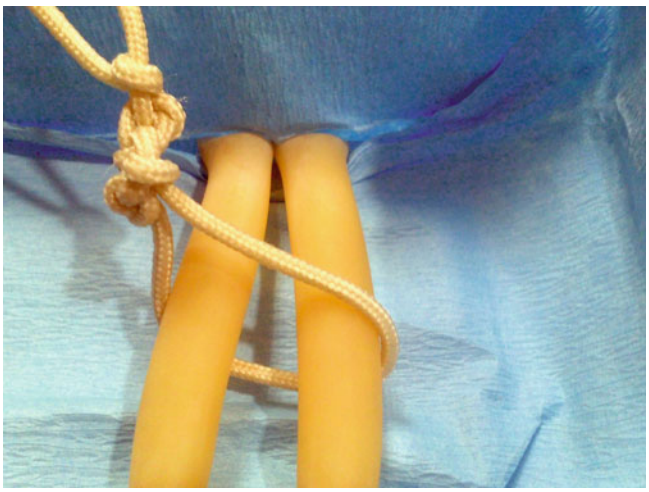

Fig. 14.3 The loop security is the ability of the loop suture that is passed through the tissue to maintain its length and tension till the knot is tied. It cannot be improved after the knot is tied. This picture shows poor loop security

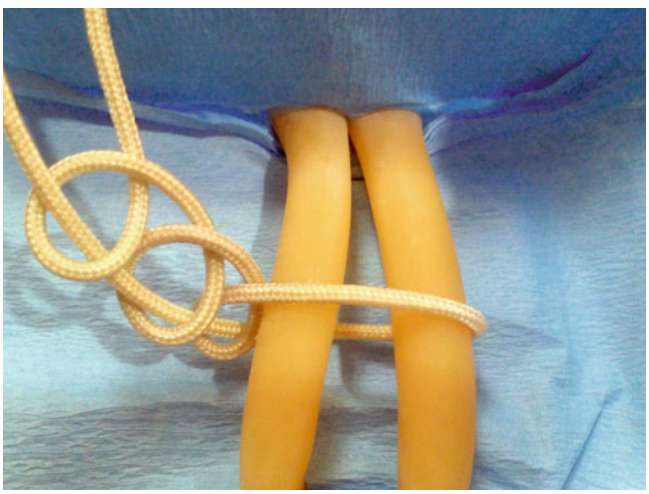

Fig. 14.4 The knot security is the ability of the completed knot to resist slippage and consequent loosening. This picture shows poor knot security

Suture Material Factors

Numerous suture materials have been described for use in arthroscopy surgeries. Suture materials are classified by their ability to be absorbed and whether it is monofilament or polyfilament (braided). The factors that a surgeon should consider while choosing a suture material are its strength, biocompatibility, knot holding, sliding properties and the ease of tying knot, and finally its susceptibility to infection. Absorbable sutures by design lose their strength with time, while the concern with nonabsorbable sutures is that they are permanent and may cause mechanical or abrasive effects. Braided sutures are easier to handle and knot settles well, but they tend to fray with handling and may injure tissues when passed. Monofilament sutures are stiffer to work with and knot security is difficult to achieve.

While the most commonly used and studied suture material in relation to arthroscopy surgery were the No. 1 PDS monofilament (absorbable) and the No. 2 ETHIBOND (a braided polyester suture from Ethicon, Somerville, New Jersey) sutures, the previous decade has the emergence of Polyblend sutures, which promise higher tensile strength and better handling and knot characteristics compared to traditional suture materials. These new-generation sutures have an inner core made of ultrahigh molecular weight polyethylene

(UHMWPE), which gives its extra strength and characteristic coating determining the handling properties. FiberWire (Arthrex, Naples, FL), ORTHOCORD, and MaxBraid can be listed among these sutures [10].

FiberWire consists of a core of many filaments of UHMWPE surrounded by braided polyester. This UHMWPE core resists elongation and is protected by the polyester jacket [10]. ORTHOCORD is another new suture that is made from a combination of 38 % UHMWPE and 62 % PDS. The ORTHOCORD suture is different from other super sutures because it consists of a PDS core with a UHMWPE sleeve and is coated with polyglactin 910 (Vicryl) for better suture handling characteristics. This material combination is designed to provide a low-profile suture once the PDS degrades while retaining some strength from the outer sleeve of UHMWPE [11]. Many studies have documented the supremacy of these newer, Polyblend braided sutures over older sutures [11–13].

Many shoulder surgeries employ suture anchors for secure fixation of suture to bone, and in this circumstance, the anchor eyelet is an additional factor to be considered. Studies comparing FiberWire with ETHIBOND have found FiberWire to have superior properties at the anchor eyelet interface [14, 15]. However, it must be noted that these newer sutures and all braided sutures may cause tissue damage when tying a sliding knot or may be at increased risk for suture damage and weakening when tying a sliding knot through a suture anchor. Coating on the newer braided sutures may reduce tissue drag but also reduce knot security.

Knot Pusher Factors

Most surgeons prefer to construct the knots extra-articularly and then push them with a knot pusher through a cannula to its intended position in the joint. Though direct intra-articular construction has been described, it is not commonly followed. Several problems can be encountered while pushing the knot with knot pushers. The knot pusher can easily slip through the loop and not advance at all. Pushing the knot may lead to fraying of the sutures. When passing half hitches it is much easier to pull the knot rather than push it. Also, the surgeon should understand the difference between the post limb, which is the limb under tension, and the limb which is threaded in the knot pusher. Though conventionally the limb that is threaded in the knot pusher is kept under tension and is identified as the post limb, it is possible that the other limb is in fact the one under tension and consequently the post limb. Such a situation occurs while past pointing the knot and can be used by the surgeon to alternate the posts without actually rethreading the knot pusher.

There are many different knot pushers available but the single-holed knot pusher has been the preferred choice of many surgeons. Most systems have knot pushers of only one size. However, a recent study [16] has pointed out the risk of knot pusher riding onto the arthroscopy knot thereby causing loosening or damage to the knot. The author has suggested the use of knot pushers of varying internal diameters for different sutures, small enough to avoid snagging of the knot yet big enough to allow passing of suture freely.

There are several "specialty" knot pushers, which represent unique designs by their respective companies [17]. The Nordt (Arthrotek), 6th Finger (Arthrex), FiberWire Tensioner (Arthrex), and the CrabClaw knot pusher (Arthrex) are some of the unique designs available. The Nordt (Arthrotek) is a mechanical spreading device. The closed spreader pushes the knot into place; activating the spreading mechanism applies equal and opposite tension to the two suture strands to tighten the knot. The 6th Finger Knot Pusher (Arthrex) consists of a small tube inside a larger tube. The surgeon can apply and maintain tension on the first throw with the inner tube while advancing subsequent throws with the sliding outer tube. The inner tube allows "past pointing" for knot tensioning. The FiberWire Tensioner (Arthrex) is a device designed specifically for the new Arthrex suture FiberWire. Once the chosen sliding knot is tied and advanced to the tissue level, the post limb is advanced up through the cannulated FiberWire Tensioner shaft and loaded into a slot and locking post on the tensioning wheel. As the wheel is turned counterclockwise, a tensiometer reads the tension obtained. When the desired tension is reached, three reverse half hitches can be thrown down the barrel of the tensioner to secure the fixation. The CrabClaw knot pusher has an opening mechanism in its ends. Hence, half-hitched can be loosely pre-tied outside the cannula. The opening jaw mechanism of the CrabClaw allows the surgeon to place the knot pusher behind each knot to advance them independently into the joint.

Knot Configuration Factors

A plethora of knot configurations and classifications have been described (Table 14.1) [35]. The simplest classification would be to classify them into sliding and non-sliding knots. Non-sliding knots are employed when the suture does not pass freely on the anchor or tissue or when the surgeon is worried about the tissue trauma from sliding sutures. Examples of non-sliding knots include square knot, Revo knot, and Snyder knot. Tissue has to be held approximated, while the knot is being placed as these knots by definition do not slide to provide further compression of the repair. Use of non-sliding knot is not recommended when the tissues are under tension.

In sliding knots, knot can slide on the post limb providing further approximation of the repair. To achieve this, knot must be placed with the post limb away from the bone, so that the knot can slide on the post and push tissues to bone and thereby provide a good approximation of repair. The initial loop limb must be at least twice the length of post limb

Table 14.1 Knot configurations

	Sliding knots		
Non-sliding knots	Slipknots	Ratchet knots	Locking (flip) knots
Stacked half hitches [18]	Duncan's loop [19]	Nicky's knot [20]	SMC knot [21]
Arthroscopic square knot [1]	French knot [22]	Modified taught line hitch [8]	Weston knot [23]
Revo knot (Snyder knot) [19, 24]	Roeder knot [25, 26]		Giant knot [27]
	Tennessee slider [24]		Field knot [28]
	Midship knot [29]		Dines knot [30]
	Inverse knot [31]		HU knot [32]
			Triad knot [33]
			Tuckahoe knot [34]

so that loop does not slide into the cannula as the knot is being pushed. Sliding knots can slide backward after being pushed into its position and thereby compromise the loop security. Two methods have been described to avoid this occurrence, and sliding knots have been classified into two types accordingly. In some sliding knots like Duncan's loop, French knot, Roeder knot, Tennessee slider, and Lafosse knot, friction of the suture provides a temporary restraint to back sliding, and the surgeon has to place additional half hitches to provide good loop security. Some sliding knots called ratchet knots (like Nicky's knot, modified taut-line hitch) preferably slide in only one direction. However, there remains a risk of knot backing out as the surgeon places additional half hitches if adequate is not taken.

Locking knots were described to overcome this difficulty. In these knots, once the knot is placed in position with good tissue approximation, pulling on the loop limb causes the knot to flip and the loop strand now becomes the post. Tensioning the other limb causes the knot to lock securely. Locking knots have been classified based on the region of flipping. Some knots like the Weston knot flip distally, while some like Nicky's knot flip proximally [36]. Theoretically, distal locking can prevent knot slippage better than proximal locking but is difficult to lock when tension in the knot loop is high. The proximal locking knot can easily be locked under the desired loop tension, but it can also easily lose tension during additional locking half hitches. The SMC knot is a middle-locking knot configuration. Due to its unique middle-locking ability, it provides the advantages of both proximal and distal locking knots. Middle-locking knots prevent easy slippage of loop security like distal locking and also can easily be locked like proximal locking knots, even with high loop tension [37].

Even though these knots are described as locking, authors describe placement of at least 3 reverse half hitches on alternate posts to secure the knot [38]. Examples of locking sliding knots include Weston knot, field knot, SMC knot, and giant knot. Though these locking sliding knots have the advantage of facilitating the desired approximation of tissues and the ability to be locked without losing loop security, they are not without shortcomings. These knots are more complicated and difficult to learn than non-sliding knots and have difficulties like premature locking as the knot is being pushed through the cannula. Untying of knot in this situation could be tiring. Furthermore, all sliding knots have an inherent risk of tissue drag and consequent tissue injury by cut through effect and consequent poor fixation. Braided sutures have much more tissue drag than monofilament sutures. Coating of braided sutures may reduce tissue drag and injury but may also decrease the knot security. The propensity to cut through tissues is compounded by the fact that tissue quality is suboptimal in most patients. This is a potential disadvantage with sliding knots, and the properties of suture material must be considered while choosing the configuration of knot.

In the recent years many studies have been conducted on the combinations of newer suture materials with various knot configurations [12, 13, 30, 39–42]. Interpretation of these studies is difficult as there is no uniformity in the knot configurations and suture materials used. No study has compared all the combinations on available suture materials with all described knot configuration in a standardized protocol. However, they seem to suggest that all the routinely used knots with these sutures are stable with respect to cyclic loading though a few studies report difference between the knot configurations and suggest some to be better than others.

Heat treatment has been suggested as a way to improve knot security. In a laboratory study, Williams et al. [43] have found that application of heat performed by use of the Mitek VAPR 3 electrosurgical unit and VAPR S90 electrode (DePuy, Mitek), ORTHOCORD, and FiberWire tolerated heat extremely well. Knot security was found to be increased.

In a study comparing the ease of tying knots with older suture materials like ETHIBOND No. 1 PDS II sutures, the Tennessee slider, Revo knot, Duncan loop, and Nicky's knot were found to be the easiest arthroscopic knots to learn to tie. Also knots tied with ETHIBOND suture were easier to tie than those tied with PDS II suture. Hence, the authors recommended that novice surgeons should consider using ETHIBOND suture and the Revo knot when developing their arthroscopic skills [44]. We feel that due to inherit weakness of ETHIBOND compared to recent strong suture material, we recommend using stronger materials and the Revo knots for beginners.

Knot-Tying Technique: General Principles

Portal Placement

The preparation for optimal suture tying begins with portal placement. Portals must be made with care so as to reach the area to be tied as closely as possible and avoid soft tissue interposition. In many instances anatomic constraints may preclude direct access to the tissues to be repaired, but every effort must be made towards it.

Cannulas

Use of transparent cannula placed directly over the proposed knot area offers several benefits. It avoids troublesome soft tissue interposition. Transparent cannulas offer visualization of knot as it being pushed inside the joint and also show any entwining of suture limbs. Placement of cannula in line with the suture limbs avoids soft tissue chaffing at its inner tip. Use of threaded cannula prevents fluid leakage at the portal site. The diameter of cannula is determined by the instruments that are required to be passed. The cannula should not come out of the joint during the entire sequence of knot tying. Otherwise it may reenter at another location and suture loops might get entangled in soft tissues.

Anchor Orientation and Suture Passage

Anchors must be placed perpendicular to the bone surface as far as possible. Eyelet face must be perpendicular to the path of the suture through the tissue. If the eyelet is rotated by 90°, the suture can be frayed. The hole made for inserting anchor must be funnel shaped. The suture limb exiting the eyelet of the anchor closest to the tissue must be passed into the tissue. This will avoid twisting of suture in the eyelet. This suture limb must be used as the initial post so that, as the knot is being pushed in, tissue will be approximated to the bone. If a double- or triple-loaded anchor is used, surgeon must be aware of eyelet design to avoid overlapping of sutures. Most such suture anchors have different colors for each suture for easy identification. When more than one anchor is used, the author prefers use of two dissimilar anchors so that all suture strands are of different colors.

One Suture in the Cannula

Only one set of suture limbs should be inside the cannula at the time of knot tying, delivering, and tightening. If there is more than one set, the other sets must be temporarily "parked" in some other ports or even outside the cannula in the same portal.

Avoidance of Twists

Prior to placement of knots any twist in the suture must be identified and removed. Double-holed knot pusher is the best instrument to remove any twists, but most surgeons use a single-holed knot pusher to identify twists. If sliding knots are being planned, the surgeon should confirm the free sliding of suture.

Specific Knot-Tying Technique

Many different configurations have been described but it is not essential or possible to learn them all. Every surgeon should practice and perfect a few knots that she/he is comfortable with. It is beyond the scope of this chapter to describe all the knots. The description of some common and widely used knot follows. Readers are referred to original articles for the description of the rest.

Half Hitch

Half hitch is the work horse of knot tying. Half hitches may be stacked on each other to become of non-sliding knot. They are also used to secure sliding knots, both locking and non-locking. A half hitch is made by following these steps:

- *Underhand half hitch*: One limb is identified as the post and other as the loop limb. The post limb is held under tension and the loop limb is initially passed under the post limb, passed distally, then above the post, and finally brought proximal under the loop.
- *Overhand half hitch*: One limb is identified as the post and other as the loop limb. The post limb is held under tension and the loop limb is initially passed over the post limb, passed distally, then under the post, and finally brought proximal over the loop.

Revo Knot (Fig. 14.5)

At the minimum, the arthroscopist must be able to tie a non-sliding knot, because at times when the suture limbs do not pass freely, it might be the only knot possible. Revo knot is the most common non-sliding arthroscopic knot used. All the general principles should be adhered to. In particular the arthroscopist must make sure that the suture limbs are not twisted. As described earlier, the suture limb of the tissue side is identified as the first post.

The steps involved are:

- The post limb is kept short and the knot pusher is placed on the post limb.
- One underhand half hitch done and pushed to the operative site.
- Tension maintained on the post and a second underhand half hitch done and pushed to the tissues.

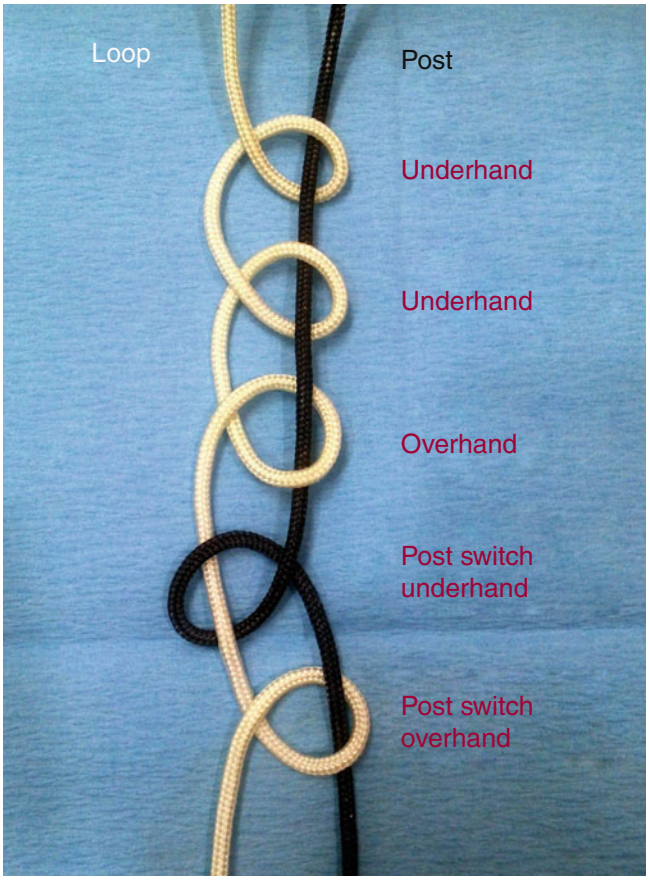

Fig. 14.5 The Revo knot configuration. This knot consists of multiple half hitches made by alternating the post and direction of half hitches

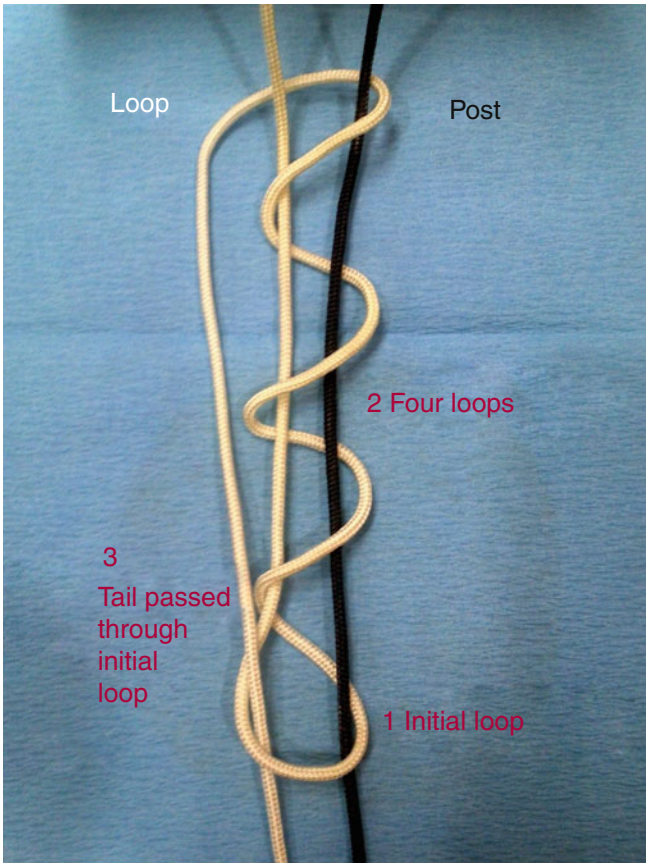

Fig. 14.6 The Duncan loop configuration. This knot must be secured with additional locking half hitches to prevent slippage

- An overhand half hitch done.
- Post switching: Loop will now be the post and knot pusher is placed on this strand and an underhand half hitch done.
- Tension is applied.
- Post switching: Knot pusher is placed on the original post and an overhand half hitch is thrown and tensioned.
- Tension is maintained on the post and additional half hitches are thrown.
- The first half hitch is passed underhand.
- Post-switched and the second half hitch thrown overhand.
- A total of 4 half hitches thrown by post switching and reversing the throw between consecutive hitches.

Duncan's Loop (Fig. 14.6)

Duncan's loop is the most common sliding knot configuration used. It is tied thus:

All the general principles should be adhered to. In particular, the arthroscopist must make sure that the suture limbs are not twisted. As described earlier, the suture limb of the tissue side is identified as the first post. The steps are:

- Loop strand is kept twice as long as the post.
- Loop strand is passed over the post to create the initial loop.
- Four further loops are passed over both strands.
- The tail of loop strand is passed through the initial loop.
- Tension on the tail of the loop will compact the knot.
- The loop is delivered to the operative site by tension on the post and simultaneously pushing with a knot pusher.

SMC Knot (Fig. 14.7)

- Post suture is kept short.
- Loop strand is crossed over the post.
- The loop strand is crossed under and then over both the strands to form a triangular interval with the post.
- The loop strand is passed under and then over post strand.
- The free is end passed from bottom to top through the triangular interval.
- This locking loop is held with a finger to avoid premature locking.
- The knot is pushed with a knot pusher while the post in under tension and the knot is pushed to the tissue.
- The loop strand is pulled to lock the suture.
- Pressure is maintained on the knot to avoid slipping.
- The knot is further secured with half hitches.

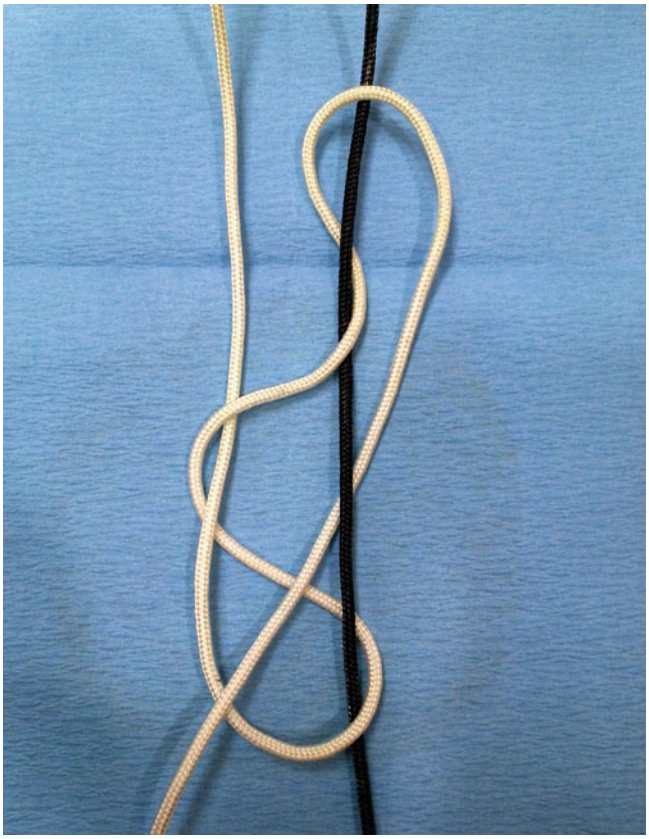

Fig. 14.7 The SMC knot configuration. The round loop formed on the *right side* of the picture is the locking loop. By pulling the lock loop strand, the internal locking mechanism is applied which prevents reverse slippage

Surgeon Factors

Consistent knot tying requires practice. Aspiring surgeons should initially practice with large chords, and once he or she is confident about configuration, knotting must be practiced with sutures. Use of cannula and knot pusher is next practiced. The final step is practicing in shoulder models using anchors and wet sutures. Practice in cadavers will facilitate learning of correct placement of cannulas and practice in surgery situations. Common problems such as suture twisting, soft-tissue entrapment, loss of tissue tension, and loose knots should be addressed and corrected before attempting shoulder surgeries. Many ingenious apparatus have been described [45] and may be used for practice.

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Part III

Major Shoulder Problems and Related Arthoscopic Procedures

Acute Traumatic Anterior Shoulder Instability

15

Patrick N. Siparsky and Dean C. Taylor

Introduction

The complex relationship between increased range of motion and decreased stability subjects the shoulder to more episodes of subluxation and dislocation than other joints in the body. Traumatic anterior glenohumeral dislocation remains a common problem not only in young athletes but also for older individuals after a fall. The most common mechanism of injury remains shoulder abduction with forced external rotation.

The shoulder is a complex joint composed of static and dynamic stabilizers. During an episode of shoulder abduction and forced external rotation, the humeral head places significant stress on the anterior-inferior labrum. At the time of dislocation, the humeral head experiences axial loading, external rotation, and anterior translation. The pathoanatomy of the traumatic anterior dislocation in young patients is typically the Bankart lesion (also known as the Perthes-Bankart lesion), where the anterior-inferior capsulolabral complex is forcefully pulled from the glenoid during the dislocation [1, 2]. With the capsulolabral complex stripped from the glenoid, the humeral head is no longer stabilized by the deepening effect of the labrum. This injury can also present with a bony fragment attached to the capsulolabral complex resulting in additional instability due to decreased glenohumeral articulation.

In addition to glenoid and labral damage, an impaction fracture on the posterolateral aspect of the humeral head is commonly present. Often referred to as the Hill-Sachs lesion, this defect results from the humeral head being forcefully externally rotated in abduction until the posterolateral aspect of the humeral head hits the glenoid [3]. A sizeable defect can complicate treatment if the shoulder arc of motion is interrupted by engagement on the glenoid articulation.

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While approximately 80 % of traumatic anterior glenohumeral dislocations result in soft tissue or bony Bankart lesions, several other soft tissue injuries can lead to recurrent instability. When the Bankart lesion is not the offending pathologic entity, the typical injury occurs to the capsule or capsular attachments to the labrum or humerus. These injuries include the humeral avulsion of the glenohumeral ligaments (HAGL), the reverse HAGL (RHAGL), the bony HAGL (BHAGL), and the glenoid avulsion of the glenohumeral ligaments (GAGL) [4–6]. Each of these injuries can contribute to anterior instability and significant effort must be made by the clinician to recognize them.

Arguably, the greatest challenge of dealing with first-time traumatic anterior instability is deciding which patients may benefit from surgical intervention. Despite controversy regarding exact recurrence rates, many authors agree that younger (age <25) males have the highest recurrence rates when treated nonoperatively [7–23]. This population is also most likely to suffer a traumatic anterior dislocation. Recurrence rates in this population can approach 100 %.

The high rate of recurrence was previously thought to be isolated to military personnel and high-level contact athletes due to the nature of the involved work or sport; however, now it is clear that this applies to all young males. For males less than 25 years old treated nonoperatively, Robinson et al. reported a 77 % chance of recurrent dislocation for athletes and an 81 % for nonathletes at 2 years. By 5-year post-dislocation, both groups had an 85 % chance of recurrent instability [20]. It is also clear that there is more to successful treatment of instability than simply avoiding recurrence. Sachs et al. showed that patients who cope with instability (and do not achieve early stability) have lower functional outcome scores than those who undergo surgical stabilization of a Bankart lesion [21]. This benefit spread across multiple different scoring systems including the American Shoulder and Elbow Society scale (ASES), Constant-Murley scale, and the Western Ontario Shoulder Instability Index (WOSI) scores. This outcome suggests that while avoidance of recurrence is imperative, each patient's ability to deal with

work, sport, social, lifestyle, and emotional aspects of the injury is also important.

This chapter will focus on treatment of the active, young patient with a first-time traumatic anterior glenohumeral dislocation. History and physical, imaging, and surgical versus nonsurgical treatment options will be discussed. A description of relevant factors to consider for each patient will help delineate treatment options. Our operative technique and postoperative rehabilitation protocols are also presented.

History and Clinical Examination

Acute traumatic dislocations are rarely subtle in nature. In most cases, the patient will recall a significant event and the onset of immediate pain. It can be helpful to understand the exact mechanism of injury, specifically the position of the arm with respect to abduction and rotation, as well as contact or a non-contact injury pattern [16]. When the shoulder is dislocated anteriorly, the arm is often held by the contralateral hand in a position of internal rotation or with the arm folded across the belly. There may be a bulge over the anterior-inferior aspect of the shoulder. Even if the diagnosis of dislocation is clear, a neurovascular exam should be done on the patient prior to reduction. At a minimum, the sensory and motor function at the hand (radial, median, and ulnar nerves), elbow flexion strength (musculocutaneous nerve), and sensory function over the lateral aspect of the upper arm (axillary nerve) should be fully tested.

At the time of injury or initial evaluation, it is helpful to discuss any past shoulder injuries or operations. Should the patient describe prior history of laxity, the examining physician must establish whether this laxity (asymptomatic hypermobility of the joint) results in symptomatic instability (abnormal translation leading to symptoms). An exam of the contralateral shoulder should be part of the routine evaluation of any shoulder dislocation.

Once the shoulder is reduced, or if the patient comes to the office with the shoulder already reduced, it is important to perform another neurovascular exam. Test both the deltoid sensory and motor function to determine if any axillary nerve injury has occurred. Between 20 and 50 % of patients with traumatic anterior dislocations experience some type of neurologic deficit, though this is often neuropraxia and will resolve [24]. Documentation of a thorough neurologic exam is critical, and discussion with the patient regarding expectations may help in the recovery process.

Once the shoulder has been relocated, there are several specific tests for instability that should be completed to help evaluate each patient that has sustained a traumatic shoulder dislocation. While the focus of this testing is clearly directed at stability, the physician should evaluate the entirety of shoulder function to rule out any other associated injuries. These tests focus on evaluating range of motion, strength, and stability. Range of motion of both shoulders should be assessed while also testing strength to confirm that no injury to the rotator cuff has occurred. In any patient over 40 years of age with a shoulder dislocation, a rotator cuff tear should be presumed until proven otherwise by exam, MRI, or both.

The sulcus sign, apprehension test, relocation test, surprise test, and the load-and-shift tests are all helpful in assessing shoulder laxity. The sulcus sign is performed by simply applying longitudinal traction to the arm while at the patient's side [16, 25]. We stabilize the humeral head in the anterior-posterior direction while pulling longitudinal traction in order to avoid confusion between inferior translation of the humeral head and anterior-inferior subluxation. The test is measured as displacement of the humeral head from the inferior aspect of the acromion.

The (anterior) apprehension, relocation, and surprise tests are a constellation of tests done together to assess anterior glenohumeral stability. These tests are best done with the patient in the supine position and the shoulder in a position of abduction and external rotation. With progressive external rotation, the patient feels a sense of apprehension as the humeral head begins to subluxate over the anterior rim of the glenoid (anterior apprehension test). The relocation test counters this maneuver by applying a posteriorly directed force from the examiner's hand directly over the humeral head. This relocation force should alleviate the patient's sense of apprehension and should also allow additional external rotation without pain. The surprise test is removal of the posteriorly directed force from the relocation maneuver resulting in reproduction of the patient's symptoms as the humeral head translates anteriorly without the opposing force [26].

Finally, the load-and-shift assesses laxity of the glenohumeral joint in various directions and helps delineate the location of the soft tissue lesion. In the clinical setting, this test looks for reproduction of symptoms based on forced translation as described below. In the operating room setting, this test assesses laxity through the amount of humeral head translation on the glenoid with the patient relaxed. This test is done with one hand on the patient's elbow, applying an axial load to center the humeral head on the glenoid. Then anterior, posterior, and inferior forces are applied separately at varying degrees of shoulder abduction. Typically, this test is done at 0°, 45°, and 90° of abduction. With progressively increasing shoulder abduction, a positive test indicates superior, middle, and inferior glenohumeral ligament laxity or injury. The translation can be graded as well. Grade 1 indicates any humeral head translation to the glenoid rim. Grade 2 indicates humeral head translation over the glenoid rim, but with spontaneous reduction. Grade 3 indicates humeral head translation over the glenoid rim that does not spontaneously reduce.

Imaging

For the initial stages of treatment after dislocation, plain radiographs are utilized. Anterior-posterior (AP), axillary lateral, and scapular Y views are routinely ordered. The West Point axillary view is also useful in identifying any anterior-inferior glenoid abnormalities (Fig. 15.1). With the patient positioned prone on the x-ray table with a pillow under the affected shoulder, the shoulder is lifted approximately 8 cm off the table. The elbow is flexed to 90° and hung off the table. The x-ray cassette is positioned on the superior aspect of the shoulder, and the x-ray beam is then aimed 25° to the patient's midline and 25° to the table surface [27].

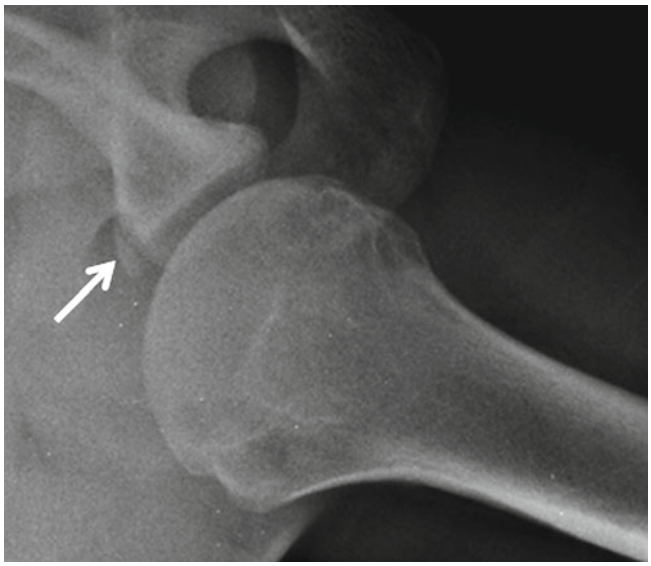

Fig. 15.1 West Point axillary view showing anterior glenoid rim fracture (*arrow*) of Bankart lesion

It is important to determine the presence of any of the following bony abnormalities while looking at the plain radiographs: Hill-Sachs lesion, greater tuberosity fracture, and anterior-inferior glenoid avulsion-type fracture (bony Bankart lesion). Typically, an MRI helps to visualize any further soft tissue injury. While hemarthrosis may provide an adequate early contrast for looking at labral injury on MRI, an arthrogram can assist in defining any labral injuries (Fig. 15.2). In the absence of a clear Bankart lesion, the clinician should suspect the presence of an injury to one of the glenohumeral ligaments, specifically the humeral avulsion of the glenohumeral ligaments (HAGL) lesion.

A CT scan should be reserved for evaluation of significant glenoid bone loss. 3-D reconstructions are sometimes helpful in defining the exact location and shape of the bone loss. With greater than 25 % bone loss, it is unlikely that an arthroscopic Bankart repair will be sufficient to restore glenohumeral stability. The CT scan can also help with characterization of a Hill-Sachs lesion or with any possible greater tuberosity fractures.

Indications and Contraindications for Surgical Stabilization

Though this is an area of long-standing controversy, multiple studies have shown that young males are the best candidates for surgical stabilization for anterior traumatic instability because they have high rates of recurrent dislocation. It should be emphasized that the decision to utilize surgical intervention is on an individual patient basis. There is no

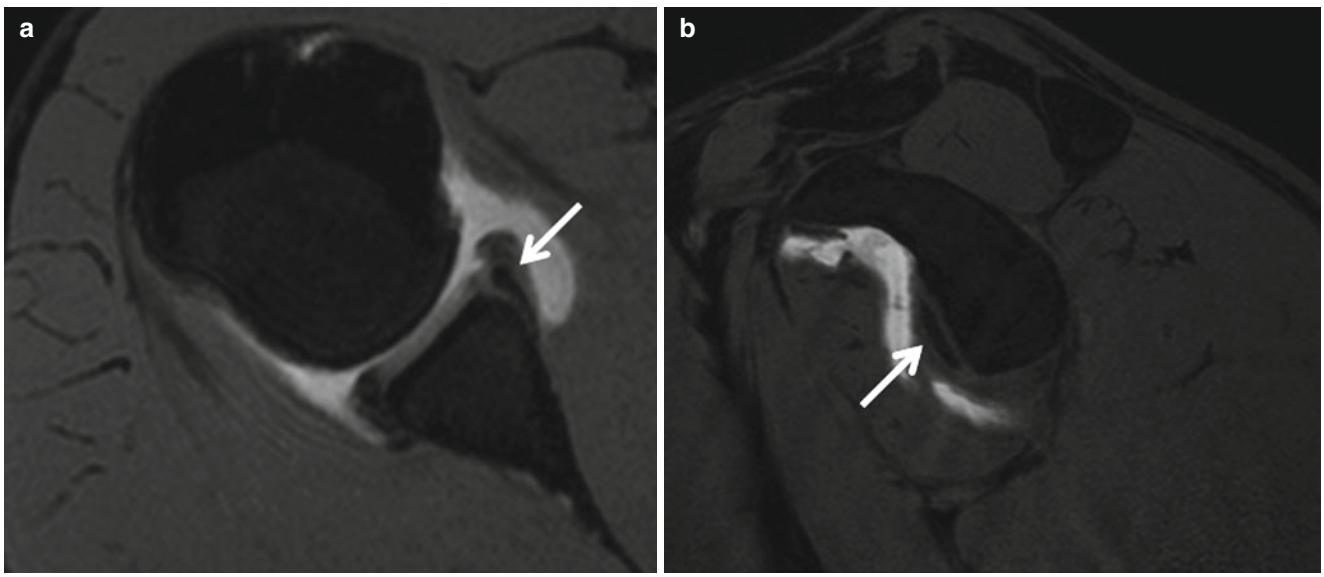

Fig. 15.2 Axial (**a**) and sagittal (**b**) MRI arthrogram showing large anterior bony Bankart (*arrow*) with capsulolabral attachment

infallible decision making plan for this injury; however, there are sufficient data now to support early surgical stabilization following first-time traumatic anterior shoulder dislocation. The decision to operate should be based on many factors including age, activity level, work status (overhead versus non-overhead), potential for lost wages, contact sport participation, and ability of the patient to cope with the instability.

While surgical stabilization for traumatic anterior instability remains elective, surgery is strongly recommended in several situations. We feel surgery should be strongly recommended if there is an irreducible dislocation, fracture requiring surgical stabilization (e.g., a displaced greater tuberosity fracture), nonconcentric glenohumeral reduction, tissue interposition blocking adequate reduction, humeral head articular defect >25 %, or an associated rotator cuff tear (>50 % cuff tear). If the dislocation occurs during the season for a high-level athlete, the inability to participate in normal sport-specific drills with intention to return to sport in the future would be another strong indication for surgery. Relative indications for surgery include multiple dislocations within the same athletic season, contact sports activity, and age <20 years [16].

There are few contraindications to early surgical stabilization of a first-time shoulder dislocation. Surgery should be avoided if the patient is unable to comply with postoperative rehabilitation and restrictions. This general rule is not limited to elderly patients. Similarly, a patient with significant medical comorbidities limiting surgical intervention or postoperative rehabilitation should not undergo surgical stabilization due to associated risks and complications.

Treatment Models

With a growing body of evidence-based literature regarding traumatic anterior shoulder instability, models have been created to provide outcome information for operative versus nonoperative treatment for patients of varying ages, activity levels, etc. The value of this modeling is that it can apply subjective patient-derived factors with objective functional data to stratify treatment options. Mather et al. [13] have designed a Decision Analysis Model that utilizes the validated WOSI [28] score as the primary outcome measure, with secondary measures including risk of 1 year and overall instability, stability at 10 years, risk of future surgery, and risk of revision surgery. All of the data utilized to create these models are from level I or II studies only [7–10, 12, 18, 19].

In the future, this will be a publically available tool for patients and physicians to become more informed regarding potential surgical outcomes based on individual information. Using a computer program, the physician can enter information into the model to help assess factors such as rate of recurrent dislocation. For example, the previously mentioned model has shown that an 18-year-old male treated nonoperatively has a 77 % risk of dislocation within the first year and only a 32 % chance of having a stable shoulder at 10 years. When treated operatively, the recurrence rate is only 17 %. Conversely, a 30-year-old female painter (significant overhead activity) also treated nonoperatively has a 34 % chance of recurrent instability at 1 year and a 62 % chance of having a stable shoulder at 10 years [13]. Her recurrence rate if treated operatively is 23 %. This modeling system provides personalized patient care, allowing various factors to help make the best decision for each patient.

Author's Preferred Surgical Technique

While controversy remains over the benefits of open versus arthroscopic stabilization [11], the author's preferred treatment method, if possible, is arthroscopic. Even if an open stabilization is indicated, a diagnostic arthroscopic evaluation of the glenohumeral joint can be helpful to visualize shoulder anatomy, specifically areas that are more difficult to visualize and repair using an open technique, such as superior labral tears or detachments.

For most instability cases, we utilize the lateral decubitus position. The sitting position is a viable option as well that allows for easier conversion to a deltopectoral approach if open surgery is indicated. The lateral decubitus position allows for traction and abduction to be easily applied allowing for improved visualization of the joint space as well as access to the glenoid and labrum.

After induction of general or regional anesthesia, a thorough examination under anesthesia (EUA) is performed. The patient is placed in the lateral decubitus position, supported by a bean bag. EUA includes assessing range of motion, as well as anterior, posterior, and inferior load-and-shift tests. The shoulder and upper extremity are then prepped and draped in sterile fashion. The upper extremity is placed in traction and approximately 40° of abduction. A standard posterior portal is made, and a full diagnostic arthroscopy is performed. Anterior superior and anterior-inferior portals are routinely utilized. The arthroscope should routinely be placed in the anterior superior portal for further evaluation of the glenohumeral joint. The posterior labrum, glenoid cartilage, and glenohumeral ligaments are often better visualized through this portal. This step will also help avoid missing a HAGL or other associated ligamentous injury. Patient positioning and placement for each portal are visualized in Fig. 15.3.

Soft Tissue Bankart Repair

Soft tissue Bankart repair requires careful soft tissue handling and good suture management. When the capsulolabral

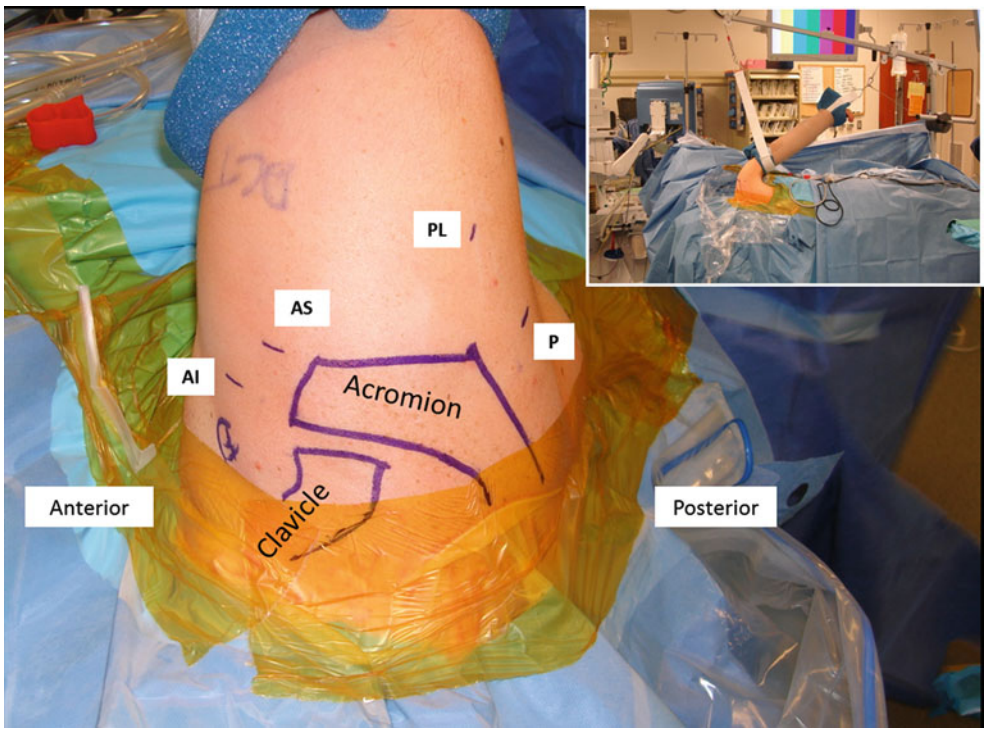

Fig. 15.3 Patient positioning and portal placement. Right shoulder in lateral decubitus position (inset photo) with *AI* (anterior inferior), *AS* (anterior superior), *PL* (posterolateral), and *P* (posterior) portals

complex is located, it must be elevated and mobilized off the glenoid neck. After this tissue is free, the glenoid edge should be cleared of any scar tissue. The glenoid rim and scapular neck are then abraded to create a bleeding healing surface. This abrasion can be done with an arthroscopic rasp, shaver, or burr. Avoid excessive bone removal and damage to the glenoid cartilage. Similarly, the undersurface of the freed labrum can be roughened with a meniscal rasp to promote healing.

Next, careful anchor placement is completed along the glenoid face. Anchors are placed 1–2 mm onto the articular cartilage surface. If the glenoid is pictured as a clock face, usually one anchor is utilized for each number on the clock face where the labrum is being repaired to the glenoid. Positioning the anchor too far medially will malreduce the labrum off the glenoid. Positioning too far onto the glenoid articular margin risks skiving under the cartilage and creation of a chondral flap. The anchor trochar is placed approximately 45° to the surface of the glenoid. While there are different ways to pass sutures from the anchor through the detached capsulolabral complex, we usually use a curved suture lasso or suture hook to pass sutures. Capsular redundancy can be addressed by passing the sutures separately through the capsule and the labrum. This effectively creates tucks in the capsular tissue which ultimately results in a tighter repair and more stability.

Once the suture has been shuttled through the capsulolabral tissue, a grasper is used to hold the repair tissue in the planned fixation location. The sutures are tied arthroscopically using sliding locking knots. All knots are kept as far from the articular margin as possible to avoid irritation and cartilage scuffing. Finally, shoulder stability is tested under direct visualization of the repaired tissue. Of note, we use suture anchors and nonabsorbable sutures, but knotless anchors, absorbable sutures, or both also work well [29, 30].

Bony Bankart Repair

For bony Bankart lesions, the bony fragment is isolated and mobilized with arthroscopic elevators. Typically, the fragment is depressed medially from the normal glenoid, separated by a scar tissue layer. Care should be taken not to damage the bony fragment, as it can be quite thin. It is often useful to keep this fragment for bone-on-bone healing with the repair. Once the fragment is free, the repair technique mirrors that of the soft tissue Bankart. The surgeon should make effort to bring the bone up to its original location while still creating a good bumper with the capsulolabral complex. Finally, shoulder stability is tested under direct visualization of the repaired tissue. A case demonstrating bony Bankart repair is shown in Fig. 15.4.

Postoperative Care

Following arthroscopic stabilization, the patient is placed into a shoulder immobilizer. We have a postoperative protocol with three distinct phases working towards full return to activity. Stage I is from 0 to 6 weeks postoperatively. For the

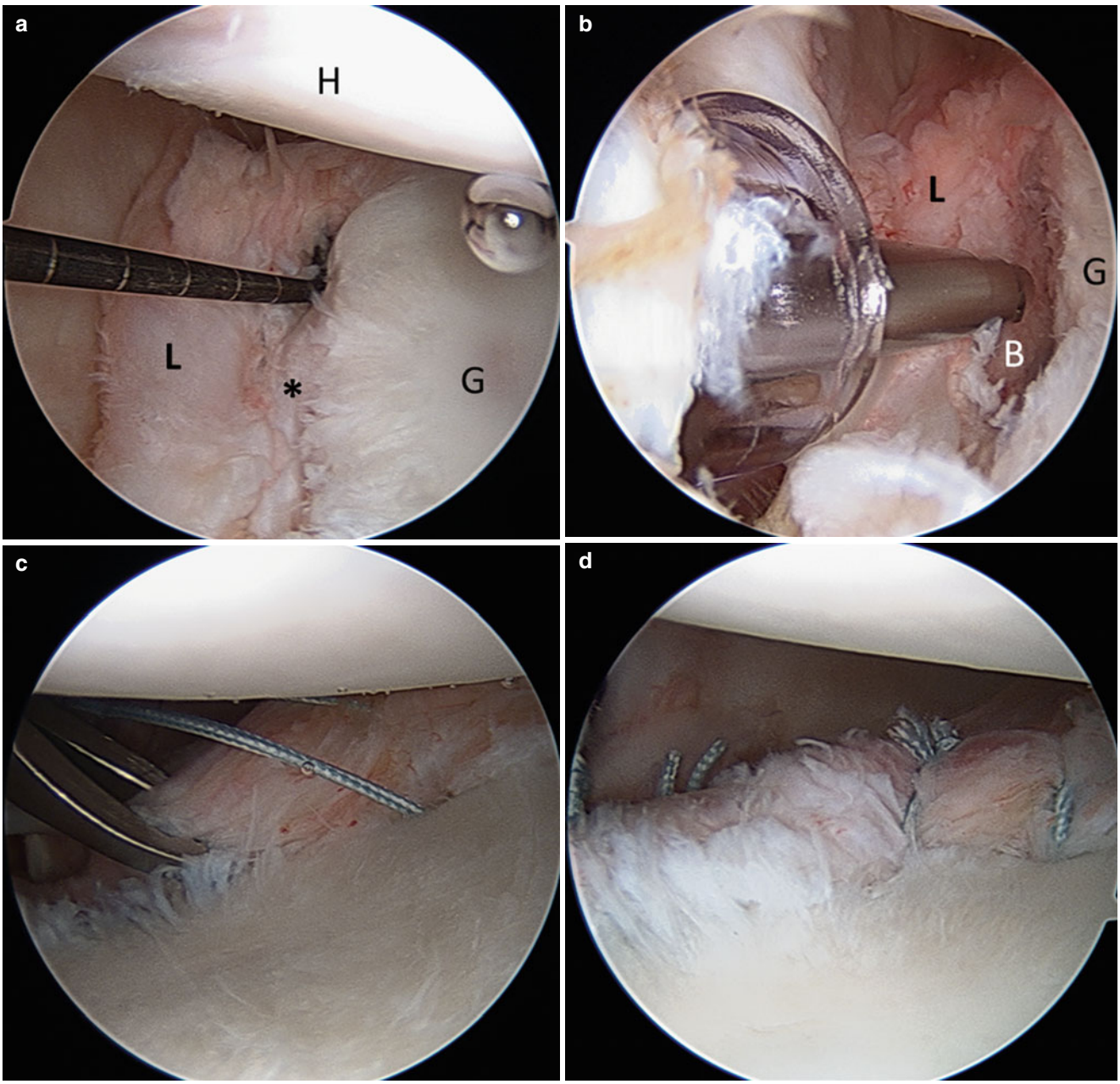

Fig. 15.4 Bony Bankart repair in right shoulder in lateral decubitus position. (**a**) Bony Bankart fragment (*) attached to labrum (*L*), scarred medially off glenoid (*G*) face (H represent humeral head). (**b**) Elevated bony Bankart (*B*) with labrum (*L*). Abrasion of glenoid (*G*) edge promotes healing. (**c**) Suture from suture anchor around capsulolabral complex. Grasper used to pull tissue to final attachment position. (**d**) Completed Bankart repair. Sutures off glenoid face

first 4 weeks, the patient remains in the shoulder immobilizer. During this time, scapular stabilization, supported pendulum, and internal and external rotation exercises with the arm at the side are done under direct supervision of a physical therapy team. We routinely use water therapy between 2 and 4 weeks postoperatively (when wounds are healed) to work on early range of motion. Between 4 and 6 weeks postoperatively, the patient can discontinue sling wear as comfort allows.

Stage II is from 6 to 12 weeks after surgery. The goals of this phase are to gently increase glenohumeral range of motion, minimize shoulder pain, and progress from active-assisted motion to active motion. Stage III (3–6 months postoperative) focuses on maximizing the strength of shoulder stabilizers, functional training for safe return to sport or work activity, and full range of motion. Usual return to full activity is at approximately 6 months.

Complications

There are several potential complications of traumatic anterior shoulder dislocations. In addition, there are several potential complications associated with surgical stabilization. Following dislocation, approximately 20–50 % of patients will have some type of neurologic complication [24]. Visser et al. showed 42 % of patients had electromyographic evidence of axillary nerve damage in a study of 77 anterior dislocations [24]. For the most part, this is a stretch-induced neuropraxia. In this study, other commonly injured nerves included the suprascapular, musculocutaneous, and radial nerves.

Two avoidable errors in managing shoulder dislocations remain failure to get adequate imaging to identify the dislocation (often from poor imaging due to patient discomfort) and failure to radiographically confirm reduction. Identifying fractures associated with dislocation is also critical. Missing a greater tuberosity fracture on x-ray may cause significant disability if the bone heals with the rotator cuff musculature in a shortened position.

In general, surgical complication rates for open and arthroscopic shoulder instability surgery are low. Kang et al. have grouped the surgical complications into perioperative, intraoperative, and postoperative complications [31]. Perioperative complications include misdiagnosis, inadequate imaging, inadequate history and physical, and failure to identify concomitant injuries. It is critical that the surgeon know the nature of the dislocation and the associated physical exam limitations that may indicate other pathologic changes such as rotator cuff or superior labral tears [17, 32, 33].

Intraoperative complications include nerve injury, failure to appropriately tension the repair, misdiagnosis of the injury causing the instability, and hardware failure. The most commonly injured nerves in open and arthroscopic instability repair remain the axillary and musculocutaneous nerves, mostly from improper retraction and excessive traction [34–36]. Tensioning the repair can be very difficult, especially if there is significant scaring of the labrum to the medial glenoid and capsular contraction. If the patient remains unstable, even with significant tension on the repair, the surgeon should look for other possible injuries such as glenohumeral ligament disruption. Similarly, if the patient continues to have anterior-inferior instability with the arm in adduction, capsular plication within the rotator interval may be warranted. Hardware failure is an uncommon complication. It is important that anchors are firmly secured within the bone and that they are not placed too flat relative to the cartilage surface. This can lead to chondral injury and failure.

Finally, postoperative complications include stiffness, pain, infection, and recurrence. Stiffness can occur as a function of tightening of the anterior capsule or as an adhesive capsulitis. Both of these are rare, and therapy can help prevent these entities. Most importantly, we recommend that therapy not be done beyond the point of discomfort. Aggressive physical therapy inducing significant pain is likely to cause increased stiffness and irritation to the joint, which is counterproductive. Infection after instability surgery is very uncommon. A series from the Mayo Clinic identified only six infections over 20 years of instability surgery [37]. Despite different treatment modes for the instability used in this study, the recommendations remained the same including appreciation of both early (<6 weeks) or late (>8 months) infections, as well as culturing for Propionibacterium acnes when working up all infections. Recurrence rates vary within the literature following stabilization. One recent systematic review [8] and one evidenced-based medicine review [38] suggest recurrence rates after surgery between 3 and 20 %, with increased likelihood of injury associated with young age and higher activity levels.

Summary

Traumatic anterior shoulder instability is a common and complex problem facing the orthopedic surgeon. This injury can result in significant disability and time lost from work or sport. Early stabilization in young patients has shown improved clinical outcomes. Decision Analysis Modeling continues to improve the surgeon's ability to predict outcomes such as recurrence while allowing the patient to participate in the decision to treat this injury operatively or nonoperatively. With good data to support these models, the discussion between patient and surgeon can now be done with more information to help project successful outcomes based on patient-specific factors, leading to more optimal outcomes and patient satisfaction.

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Recurrent Anterior Shoulder Instability

16

Mary K. Mulcahey, John W. McNeil II,
and Matthew T. Provencher

Epidemiology

The shoulder is the most commonly dislocating joint in the human body, often occurring in the anterior direction. Stability of the shoulder joint is provided by three primary mechanisms [1]: (1) concavity compression, (2) compression of the humeral head onto the glenoid by the rotator cuff muscles, and (3) glenohumeral ligaments and capsule. With regard to instability, however, the more relevant anatomy includes the static and dynamic stabilizers of the glenohumeral joint. The static stabilizers consist of the bony anatomy, glenoid labrum, capsular ligaments, and the rotator interval, whereas the rotator cuff and the scapular stabilizers comprise the dynamic stabilizers. The glenoid labrum contributes to shoulder stability in several ways. It serves as an attachment point for the capsule and the surrounding ligamentous structures, effectively anchoring them to the glenoid. The labrum also contributes to the concavity-compression mechanism by increasing the concavity of the glenoid [2]. Glenohumeral dislocation often results in soft tissue (e.g., labral tear, capsular stretching) or bony injuries (e.g., glenoid or humeral head bone loss) and is therefore frequently associated with persistent deficits of shoulder function and a high risk of subsequent instability episodes in young, active patients [3–10].

Recurrent instability can be atraumatic or may occur following a traumatic event and is frequently classified as a subsequent dislocation, subluxation event, or persistent apprehension [11, 12]. Patients with recurrent instability may present with chronic pain (<6 months) in abduction and external rotation as their only symptom [13]. In addition, the unstable painful shoulder (UPS) was described by Boileau et al. [14] in 2011 as an indication of unrecognized anteroinferior instability that causes persistent pain in young athletes. These patients have anatomic lesions suggestive of instability; however, there is often no history of recurrent instability episodes [14].

Recent studies report that the rates of recurrent anterior instability following arthroscopic stabilization procedures range from 4 to 18 % [15–20] versus 0–7 % in open stabilization procedures [16, 18], although there remains considerable debate about the optimal treatment strategy. Several risk factors contribute to the rate of recurrent anterior instability following operative stabilization. The most commonly reported risk factors include age of the patient (<age 30 = higher risk), capsular stretching, generalized ligamentous laxity, number of anchors used, and participation in contact sports [15, 20–26]. Glenoid or humeral head bone loss has also been identified as a risk factor for recurrent instability [15, 20, 21, 23, 24, 27]. A study by Boileau et al. in 2006 [15] elucidated several factors associated with recurrent instability following arthroscopic Bankart repair. Greater than 25 % loss of the glenoid surface, a large Hill-Sachs lesion, a stretched inferior glenohumeral ligament, and anterior hyperlaxity were all significantly related to failure. This study concluded that patients had a 75 % recurrence rate in the presence of a stretched inferior glenohumeral ligament, anterior hyperlaxity, or a glenoid compression fracture involving more than 25 % of the glenoid surface [15].

In 2007, the instability severity index score (ISIS) was proposed as a means of identifying risk factors associated with recurrent instability [21]. This study identified six risk factors for recurrent instability including age less than 20 years at the time of stabilization; participation in competitive sports, contact sports, or any athletics requiring persistent overhead activity; shoulder hyperlaxity; Hill-Sachs lesion visible on a plain anteroposterior (AP) radiograph with the arm in external

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rotation; or loss of the normal sclerosis on the inferior border of the glenoid on a plain AP radiograph. Patients with a score greater than 6 points had a recurrence rate exceeding 70 %, which led the authors to recommend arthroscopic anterior stabilization only for patients with a score of 6 or less. For those patients with a score of greater than 6 points, an open surgical procedure (i.e., Latarjet) was recommended because of the unacceptably high rate of recurrence [21].

Trauma is a significant risk factor associated with recurrent shoulder instability following stabilization [28–30]. This factor is especially relevant in contact athletes, with the highest rates of recurrence being reported for men's football, wrestling, and hockey [31]. Contact athletes suffer a much higher rate of failure of stabilization procedures compared to the general population [28, 30, 32]. Cho et al. [28] reported a recurrence rate of 28.6 % in collision athletes versus only 6.7 % in non-collision athletes following arthroscopic stabilization for anterior shoulder instability.

Recurrent instability may occur in the setting of major trauma after the initial repair or may result from minimal force. The etiology commonly involves a soft tissue or bone tissue, and sometimes both. The most commonly reported factors contributing to failure are diagnostic and technical failures, capsular or labral insufficiency, and glenoid or humeral head bone loss, or both [15, 23, 24, 26, 28, 32, 34, 35].

Pathophysiology

It is important to understand the pathology that is commonly associated with anterior glenohumeral instability and to appropriately address these lesions when they are diagnosed.

Lesions of the Glenoid Labrum and Ligamentous Attachments

Bankart Lesion

Injury to the glenoid labrum and associated ligamentous attachments commonly occur following anterior shoulder dislocation. Avulsion of the anterior labroligamentous structures from the anteroinferior glenoid rim is known as the Bankart lesion (Fig. 16.1). This is often considered the "essential lesion" of anterior shoulder instability, with 90 % of all anterior shoulder dislocations having associated Bankart lesions [36]. The labrum and attached ligaments are often found anterior to the glenoid rim. The inferior and middle glenohumeral ligaments are therefore unable to perform their stabilizing functions at end range of motion. Additionally, the labrum no longer serves to stabilize or deepen the glenoid socket. The force required to translate the humeral head anteriorly decreases by 50 % in the absence of the glenoid labrum [37].

Anterior Labroligamentous Periosteal Sleeve Avulsion (ALPSA)

This lesion was initially described by Neviaser in 1993 [38]. The labroligamentous complex heals on the medial aspect of the glenoid neck (Fig. 16.2); however, recurrent instability is possible given the incompetence of the anterior inferior glenohumeral ligament (IGHL). ALPSA lesions are not commonly associated with first-time anterior dislocations, rather "time-dependent" and "recurrence-dependent" etiologies have been proposed [39]. In 2007, Yiannakopoulos et al. compared intra-articular lesions present in acute and chronic shoulder instability and found that almost ALPSA lesions were found in shoulders with chronic instability [40].

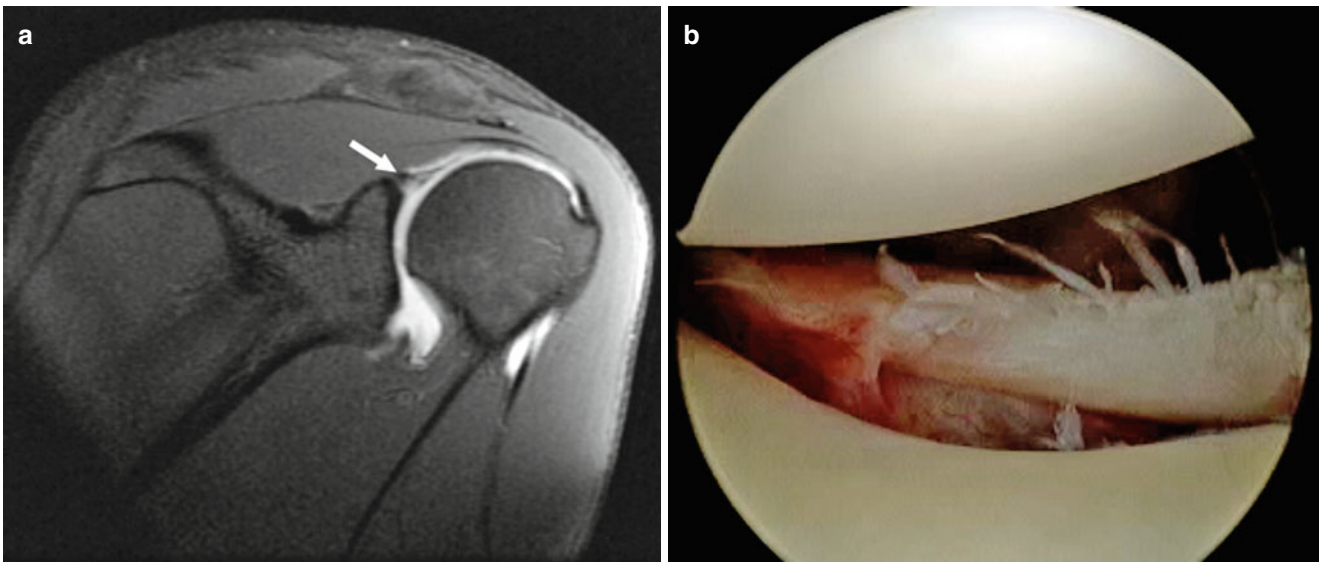

Fig. 16.1 Coronal MRI view (**a**) and arthroscopic image, *white arrow* shows superior extent of anterior labral tear, which extents inferiorly and torn off anterior glenoid (**b**) depicting a Bankart lesion

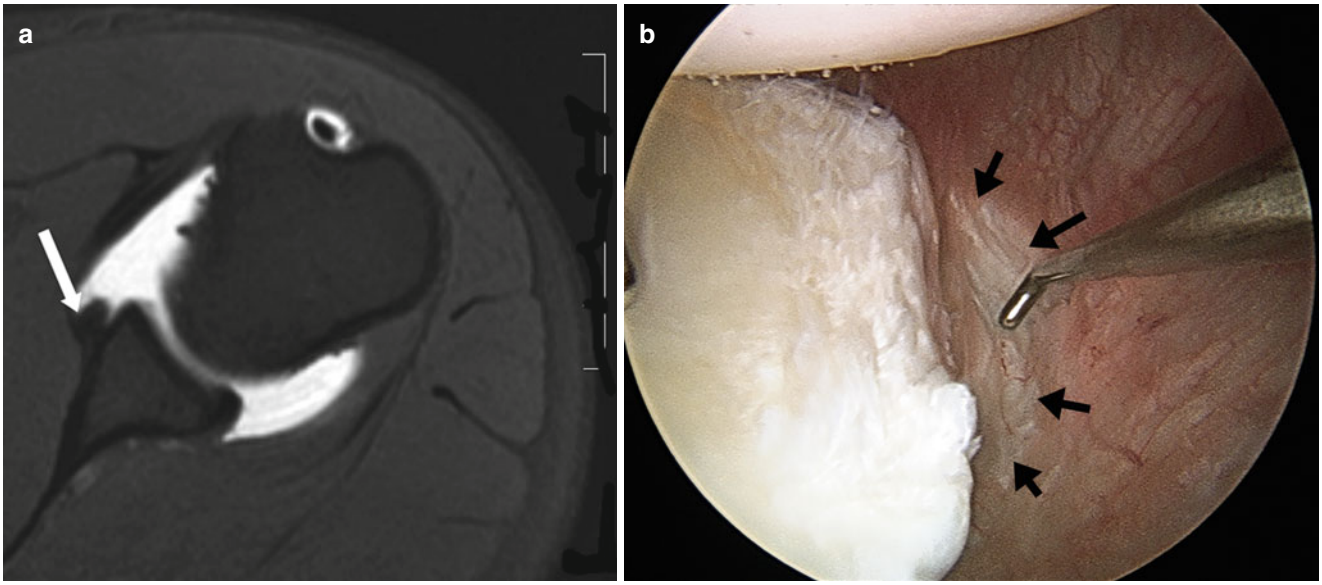

Fig. 16.2 Axial oblique MRA view, *white arrow* demonstrates ALPSA labral tear (**a**) and arthroscopic image (**b**) depicting an ALPSA lesion (*black arrows*)

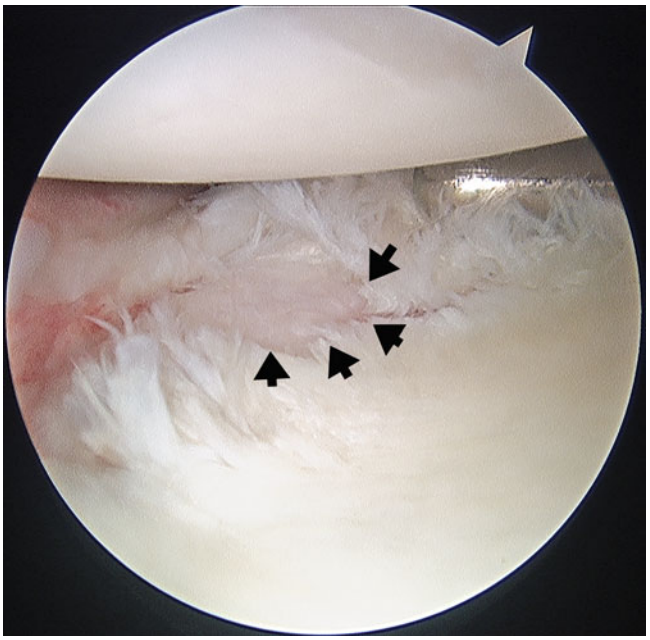

Fig. 16.3 Arthroscopic image demonstrating a GLAD lesion (*black arrows*)

Glenolabral Articular Disruption (GLAD)

Neviaser also described the GLAD lesion in 1993 [41]. This lesion consists of a shear injury to the articular cartilage on the anteroinferior aspect of the glenoid and the attached glenoid labrum (Fig. 16.3) and commonly occurs with forced adduction to an abducted, externally rotated arm.

Bony Bankart Lesion

The bony Bankart lesion occurs when an anterior glenohumeral dislocation of the humeral head causes a fracture of the anteroinferior portion of the glenoid rim (Fig. 16.4). Although the bony architecture of the glenoid is small, it serves a critical function in maintaining the stability of the glenohumeral joint [42]. Even a small fracture of the anterior glenoid allows the humeral head to easily subluxate anteriorly.

Humeral Avulsion of Glenohumeral Ligament (HAGL)

Humeral detachment of the glenohumeral ligaments (Fig. 16.5) was first noted by Bach et al. in 1988 [43]. A classic biomechanical study of the inferior glenohumeral ligament found that in 25 % of specimens, the ligaments were avulsed from the humerus [44]. The term "HAGL" was coined by Wolf et al. in 1995, in a study in which they reported a 1–9 % incidence of this lesion following anterior shoulder dislocation [45].

Isolated capsular injury is rare following anterior shoulder dislocation, accounting for only between 0 and 11 % of injury patterns. Capsular injuries, which are more often seen in recurrent instability, commonly occur in association with other pathologies [46].

Superior Labral Anterior and Posterior (SLAP) Tears

SLAP tears are not considered a primary lesion in anterior instability; however, this injury often occurs in patients following glenohumeral dislocation. Hintermann et al. identified a 7 % incidence of SLAP tears in a series of 212 patients treated arthroscopically for anterior shoulder instability [36]. Persistence of a SLAP tear may complicate the overall recurrence after instability repair.

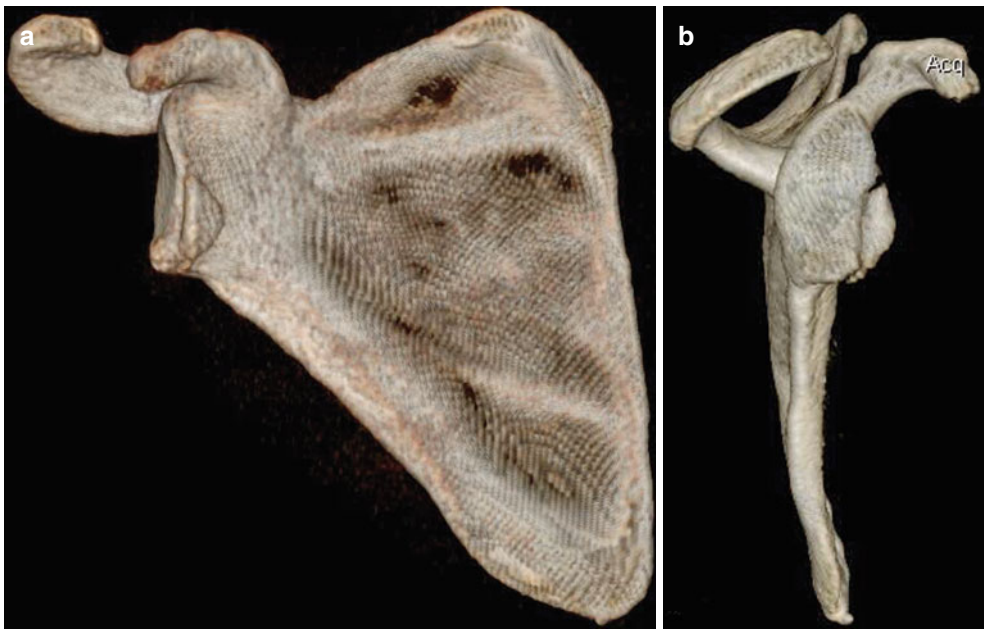

Fig. 16.4 Coronal (**a**) and sagittal (**b**) 3D CT images demonstrating a bony Bankart lesion

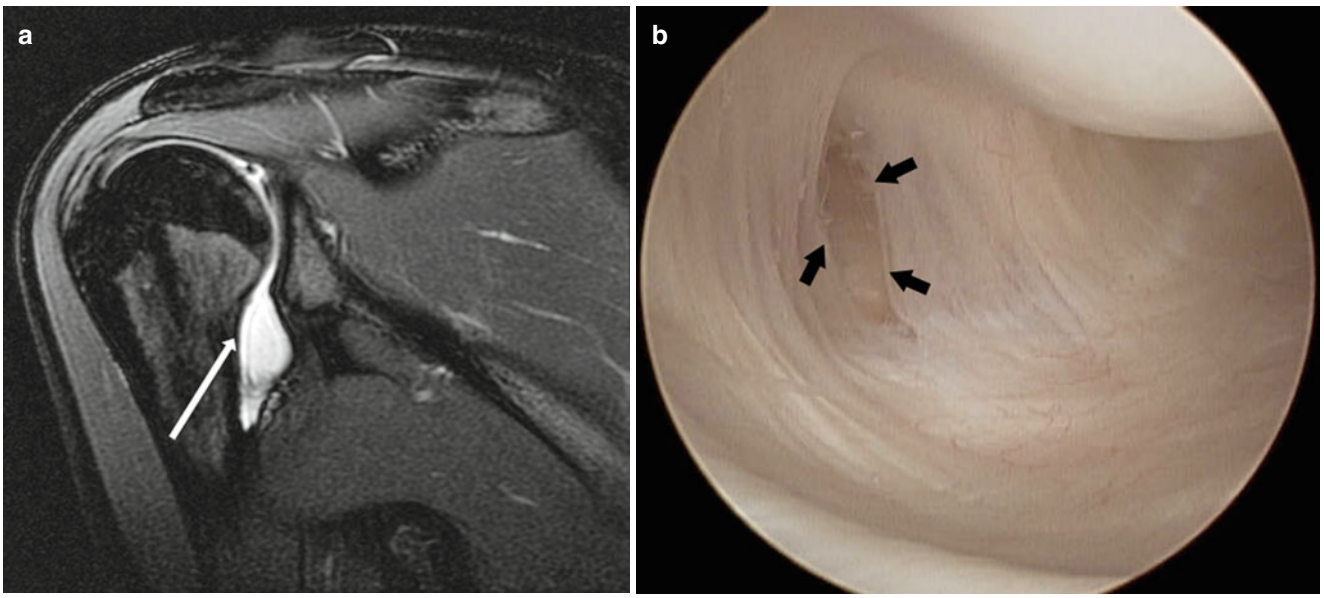

Fig. 16.5 Coronal MRA (**a**) and arthroscopic image, *white arrow* demonstrates HAGL tear (**b**) demonstrating a HAGL lesion (*black arrows*)

Bony Humeral Lesions

Hill-Sachs Lesion

The Hill-Sachs lesion is a compression fracture of the posterosuperolateral aspect of the humeral head that occurs as a result of impaction with the more dense anteroinferior glenoid during anterior dislocation of the glenohumeral joint (Fig. 16.6a).

Burkhart and De Beer [23] initially defined the Hill-Sachs lesion as "engaging" when the humeral head defect engages the rim of the glenoid while the shoulder is in a position of abduction and external rotation (Fig. 16.6b). Several studies support an association between an engaging Hill-Sachs lesion and anterior glenoid bone loss in some patients with recurrent anterior shoulder instability [23, 26, 47–49].

Hill-Sachs lesions are associated with 40–90 % of anterior shoulder instability events [40, 50–53], and the incidence may approach 100 % in patients with recurrent anterior instability [53]. Hill-Sachs lesions most commonly occur in association with anterior capsuloligamentous

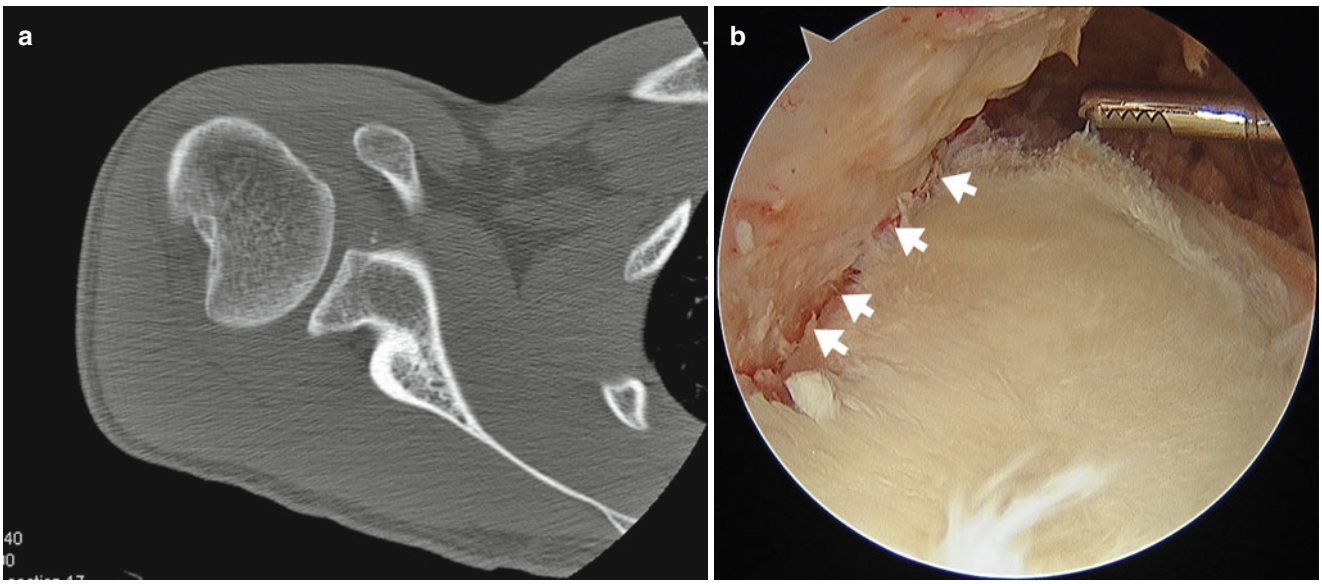

Fig. 16.6 CT image of a Hill-Sachs lesion (**a**) and arthroscopic view of the Hill-Sachs lesion that has easy "engagement" with the glenoid (**b**) shown by *white arrows* marking anterior glenoid and humeral head engagement over anterior rim

avulsion (i.e., Bankart lesion) [51] but may also be seen in association with anterior glenohumeral ligamentous pathology and glenoid bone loss (i.e., bony Bankart lesion) [48, 54]. Numerous classification and grading systems exist for Hill-Sachs lesions [26, 47, 50, 55, 56], although none of them have been deemed optimal for directing successful management. The most important factors to determine if a Hill-Sachs lesion is clinically significant are its size and whether it is engaging [23]. Lesions involving <20 % of the humeral head articular surface are rarely of clinical significance, while lesions >40 % of the articular surface are almost always clinically significant and are implicated as an underlying cause of recurrent instability [47, 57]. Management of midsize lesions (20–40 % of humeral head articular surface) is challenging.

Other factors to take into account when determining a management plan for Hill-Sachs lesions include the extent of concomitant glenoid bone loss, the extent of engagement with the glenoid, and the location and orientation of the lesion [49]. In midsize Hill-Sachs lesions, the injury is a bipolar problem with associated glenoid bone loss worsening the humeral-side defect and increasing the risk of instability. Yamamoto et al. [58] described the Hill-Sachs lesion based on the location and size of the humeral head defect and on the amount of glenoid bone loss. Using a cadaveric model, they determined that the distance from the contact area between the glenoid and the humeral head to the medial margin of the footprint was 84 % of the glenoid width. The authors concluded that a Hill-Sachs lesion outside of this glenoid track was at high risk for engagement and, consequently, recurrent instability [58]. Based on this model, large amounts of glenoid bone loss increase the significance of even small Hill-Sachs lesions [49].

History

Shoulder instability is defined as the inability to maintain the humeral head centered on the glenoid. When evaluating a patient with suspected shoulder instability, it is critical to obtain an accurate history. The patient should be asked to describe the position of the shoulder at the time of the initial dislocation as well as the mechanism of injury. It is also important to determine the frequency of dislocation episodes and the functional disruption that is caused by the instability [59]. A thorough history should also include the necessity for medically assisted reduction versus self-reduction, activity level (including contact versus noncontact sports), amount of time that has passed since the initial dislocation, and any treatment provided to the patient [60].

The provocative anterior instability position (typically with the shoulder abducted and externally rotated) as well as the amount of trauma required for instability to occur have significant implications for overall management. Dislocation with simple daily activities such as reaching overhead suggests different diagnoses (e.g., multidirectional instability and glenoid hypoplasia) than instability episodes that occur in the setting of more significant trauma [60].

Patients will often describe feelings of pain in extremes of motion or a sense of impending instability. The patient may

also report a history of subluxation or dislocation with the shoulder in certain positions (most often abduction and external rotation and with overhead activities). Although these are the most common symptoms, many patients will complain of numbness, transient sharp pain, or weakness as their only symptom of instability [60].

Clinical Examination

A thorough physical exam is paramount to making the correct diagnosis and for determining the appropriate treatment plan. It should be noted that instability of the glenohumeral joint is a clinical diagnosis that is confirmed with a careful history and examination. Furthermore, the direction, type, and classification of shoulder instability as well as operative plan are based mainly on supporting features from the history and physical examination. Evaluating a patient for laxity versus instability is imperative. It is important to remember that the presence of shoulder laxity does not necessarily signify associated instability. Instability is a perception experienced by patients during a dislocation or subluxation event. Laxity, on the other hand, is a normal finding of the glenohumeral joint, given the minimum obligate translation of the humeral head on the glenoid that is required for normal shoulder function [1, 61]. Shoulder laxity and instability are assessed by performing translation testing for laxity (anterior, posterior, and inferior sulcus) and symptomatic directional instability, which is an important indicator of shoulder instability [60]. External rotation with the arm at the side will often decrease the sulcus sign; failure to do so indicates a pathologic rotator interval [62, 63].

Most patients with shoulder instability will have normal range of motion, neurovascular exam, and strength of both the shoulder girdle and periscapular muscles [60]. Initially, the patient should be asked to demonstrate the position of the shoulder at the time of injury and the mechanism of injury. Examine the contralateral shoulder first to demonstrate the typical positions of instability as well as the specific physical exam tests to be performed, so the patient is able to anticipate what will happen during examination of the affected shoulder. There are three key components to the clinical exam: (1) anterior/posterior apprehension tests, (2) examining the glenoid concavity (e.g., load-and-shift test), and (3) assessing the muscles that compress the humeral head against the glenoid [59].

The anterior apprehension test is performed by placing the arm in abduction, extension, and external rotation. Conducting the posterior apprehension test involves placing the arm in adduction, midflexion, and internal rotation. Pain alone is insufficient evidence for instability. More revealing is confirmation from the patient that this position elicits the sensation that he/she has when she shoulder is ready to dislocate [59].

The status of the glenoid concavity can be assessed by having the seated patient relax and place the forearm on the thigh. Anterior and posterior humeral head translation is then evaluated as an indication of overall joint laxity. The humeral head is then pressed into the glenoid cavity while anterior followed by posterior translation is attempted (the load-and-shift test). Unrestricted translation of the humeral head while it is being pressed into the glenoid cavity suggests a deficiency of the glenoid lip in that direction [59]. It is imperative to note the point at which the humeral head begins to dislocate and engage on the glenoid. The presence of a significant engaging Hill-Sachs lesion or associated bone loss may be indicated by dislocation or engagement of the humeral head on the glenoid with the arm at the side, in 30° of external rotation [23] or in lesser degrees of abduction (45°) and external rotation. Shoulder instability in the midranges of abduction/external rotation is a common symptom in patients with engaging Hill-Sachs lesions [60] or in patients with glenoid bone loss.

Assessing the muscles that compress the humeral head into the glenoid includes evaluation of the isometric strength of the subscapularis, supraspinatus, and infraspinatus [59].

Imaging

Following a traumatic shoulder dislocation, plain radiographs should be obtained including true anteroposterior, axillary lateral, and scapular Y views. In patients with a history of recurrent anterior shoulder instability, or if there is suspicion for a bone defect, specialized views are indicated including apical oblique (Fig. 16.7a) [64, 65], West Point view (Fig. 16.7b) [66], or Didiee [67] views. For further evaluation of humeral head defects, including the Hill-Sachs lesion, the Stryker Notch view (Fig. 16.7c) [67] and a true anteroposterior in internal rotation should be obtained (Table 16.1) [68, 69].

Occasionally, surgeons may wish to obtain additional information regarding capsular and labral tissues, the bone, the rotator cuff, or the neurologic status of muscles. In these cases, further tests including magnetic resonance imaging (MRI), computed tomography (CT), electromyography, or diagnostic arthroscopy may be warranted [59]. Magnetic resonance arthrography (MRA), which involves injection of gadolinium into the glenohumeral joint, provides additional detail than that obtained with standard MRI. MRA is preferred by many surgeons over MRI as a diagnostic study for labral tears. The coronal oblique view

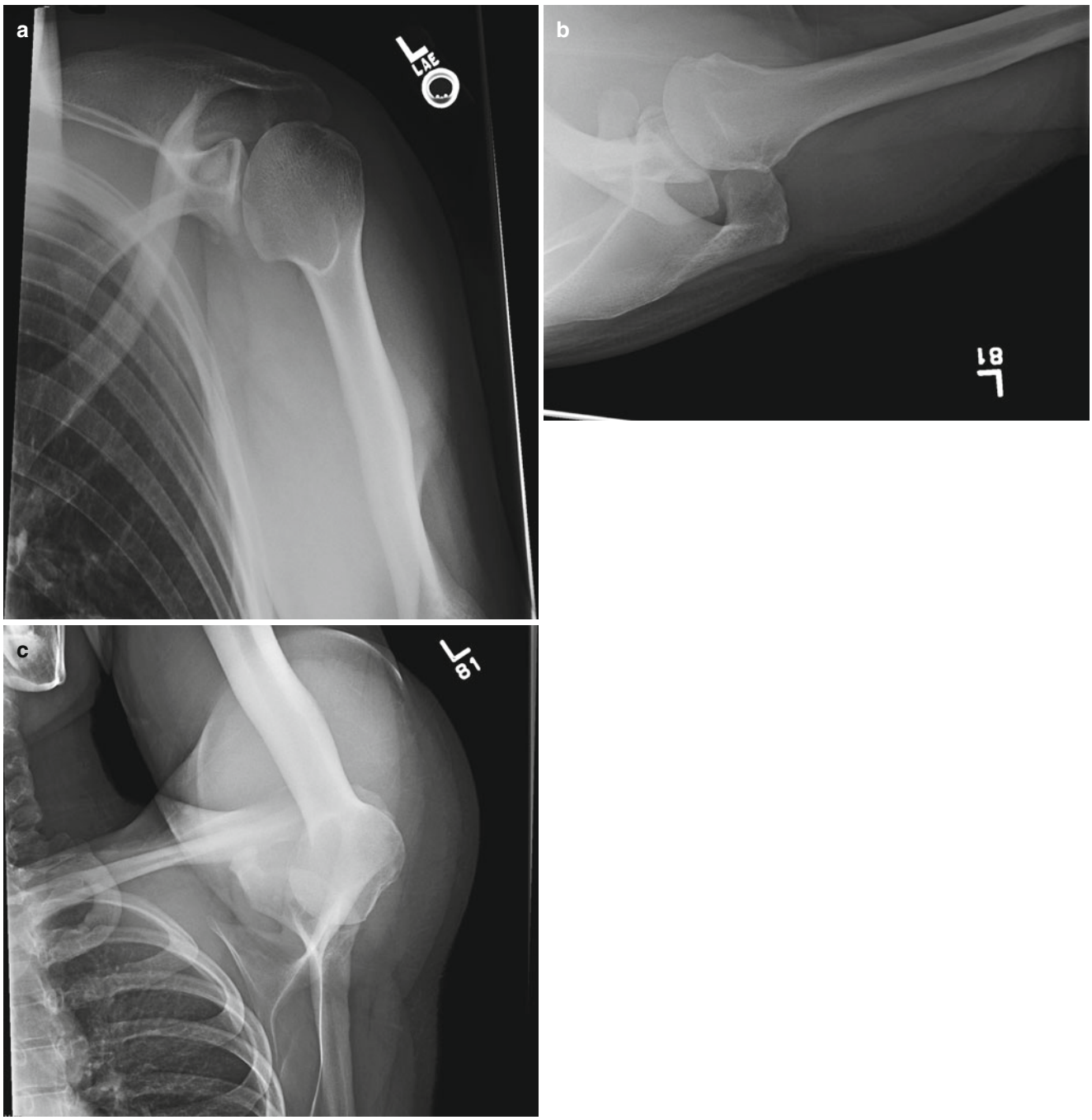

Fig. 16.7 Plain radiographs demonstrating a true AP (**a**), West Point view (**b**), and Stryker Notch view (**c**) of the shoulder

is the best view to identify SLAP tears, while the axial oblique view demonstrates anterior and posterior labral tears. CT is indicated to rule out bone deficiency (glenoid and humeral head). A 3D CT can also be ordered to more accurately identify and quantify bony deficiency. Additionally, the humeral head can be subtracted from the 3D CT, providing the best means of quantifying glenoid bone loss (Fig. 16.8) [46].

Treatment: Indications and Contraindications

Traumatic anterior dislocation of the glenohumeral joint is a very common injury and is associated with a high risk of recurrent instability episodes in young active patients. Numerous factors must be taken into account when deciding

Table 16.1 Specialized radiographic views used to evaluate patients with recurrent shoulder instability

Specialized views	Position of patient	Used to assess	Demonstrates
Apical oblique [68, 69]	Supine. Involved arm is flexed at the elbow and hand is placed across the chest. Injured shoulder is placed in the 45° posterior oblique position. Central beam is angled 45° caudad	Shoulder injuries	Glenoid rim fractures, Hill-Sachs lesions, humeral head subluxation, soft tissue calcification
West Point view [68, 69]	Prone. Involved shoulder on a pad raised 8 cm from tabletop. Head and neck are turned away from involved side. The cassette is placed at superior aspect of shoulder. X-ray beam is centered on the axilla with 25° downward angulation from the horizontal and 25° medial angulation from the midline	Lesions of the anteroinferior glenoid rim	Soft stissue calcification adjacent to anterior or anteroinferior rim of the glenoid or fracture of the glenoid rim
Didiee view [68, 69]	Prone. The cassette is placed under the shoulder. Arm parallel to the table top with a 7.5-cm pad under the elbow. Dorsum of hand on the hip with the thumb directed upward. Beam angled 45° lateromedially and is aimed at the humeral head	Anteroinferior margin of the glenoid	Hill-Sachs lesion
Stryker Notch view [68, 69]	Supine. Involved shoulder is raised vertically and the palm is placed behind the head. The elbow points toward the ceiling and the humerus is perpendicular to the table. The cassette is placed on the tabletop, directly below the shoulder	Posterolateral portion of the humeral head	Compression in posterolateral portion of the humeral head (i.e., Hill-Sachs lesion)

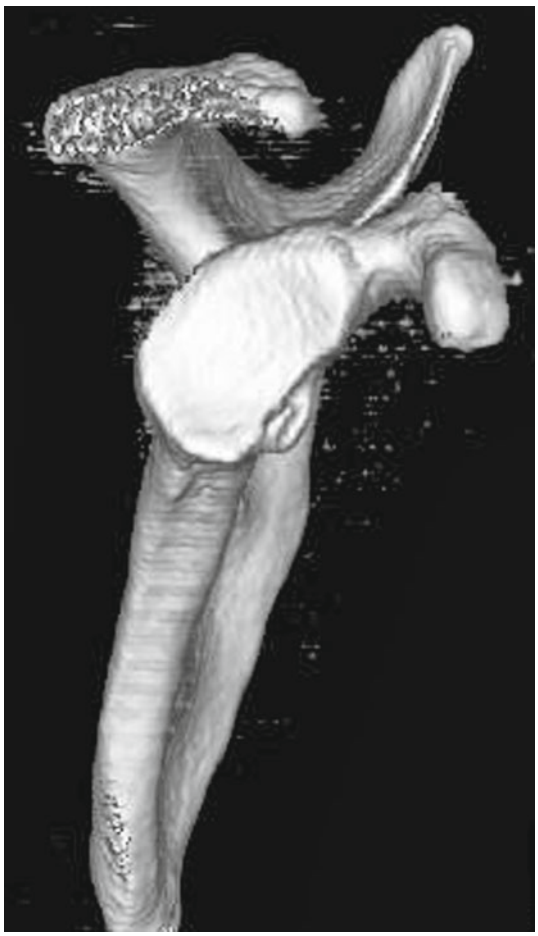

Fig. 16.8 3D CT with humeral head subtraction demonstrating attrition of the glenoid

on operative versus nonoperative management of anterior shoulder instability including etiology (e.g., traumatic versus atraumatic or generalized ligamentous laxity), age, frequency of instability episodes, activity level, and associated pathology (e.g., glenoid or humeral head bone loss, rotator cuff tear, SLAP tear) [70].

Nonoperative management of shoulder instability in active patients less than 30 years old results in a recurrence rate of 17–96 % versus 4–22 % in patients treated with arthroscopic stabilization procedures [71, 72]. This data supports early arthroscopic repair following first-time dislocation for young, active patients or those involved in overhead sports, although not all patients are ideal surgical candidates and the algorithm for optimal treatment continues to evolve.

Decision-Making Algorithm

Management of primary anterior shoulder dislocation continues to be an issue of considerable debate. The group at highest risk of recurrent dislocation has been defined as 18–30-year-old athletes participating in collision or overhand sports who sustain a dominant-side shoulder injury [73]. Numerous studies demonstrate age and sex to be two of the most important factors in determining the risk of recurrent instability [5–8, 10, 74]. Recurrence rates following first-time dislocation range from 17 to 96 % [73] and the risk of recurrence has been noted to be highest within the first 2 years following the initial dislocation [10].

The pathology associated with acute shoulder dislocation is significant and includes an 87 % incidence of Bankart lesions with 64 % also suffering a Hill-Sachs lesion and an 18 % incidence of both capsular tearing and rotator cuff injuries [75]. Multiple dislocations can lead to progressive intra-articular pathology including worsening bone loss (humeral head and anterior glenoid), capsular attenuation, damage to the rotator cuff, and superior labrum and biceps anchor injuries [73]. Quality of life is also an important consideration when determining whether to perform early surgical intervention. Studies by Kirkley et al. [76] and Robinson et al. [10] both suggest that recurrent shoulder instability negatively impacts a patient's quality of life by preventing return to preinjury level of play. Based on these data, it is reasonable to recommend surgical stabilization after first-time dislocation in young male athletes participating in collision or overhead sports. Open Bankart repair was originally considered the gold standard in the management of young athletes with shoulder instability; however, arthroscopic technique and surgeon experience have improved to such an extent that results of arthroscopic repair are equal to those of the open repair technique [77].

Patients with traumatic, recurrent anterior instability may also be candidates for arthroscopic stabilization; however, careful patient selection is imperative to maximize results. A thorough history and physical exam should be used to confirm anteroinferior laxity and adequate bone stock (glenoid and humeral head) to support arthroscopic repair. Advanced imaging including CT or 3D CT may also be warranted to visualize and quantify any bony deficiency. Patients with anteroinferior instability and no significant bone loss are candidates for arthroscopic repair; however, patients with glenoid bone loss >20 %, Hill-Sachs lesions >25–30 %, or engaging Hill-Sachs lesions may warrant an open procedure (i.e., Latarjet). Soft tissue injuries (e.g., HAGL lesion) may also require open repair [78].

Arthroscopic Treatment: Surgical Technique

Patient Positioning

Shoulder arthroscopy can be performed with general anesthesia, inter-scalene block, or a combination of the two depending on the preference of the surgical team and patient. Patients can be placed in either the lateral decubitus or beach-chair position. The beach-chair position has the advantage of providing easy access to the glenohumeral joint, ability to see the anterosuperior, inferior, and anterior aspects of the joint, and ease of conversion to an open procedure if necessary. Patient positioning is based on surgeon preference; however, for cases of shoulder instability, the authors prefer to place patients in the lateral decubitus position since it allows easy access to the entire glenoid, labrum, and capsule. With longitudinal and direct balanced suspension of the arm, this position allows for greater distraction of the glenohumeral joint and hence increased space for passing instruments during the repair. A limitation of lateral decubitus positioning is the difficulty of obtaining precise rotational control during instability repair. Appropriate tensioning of the capsule and inferior glenohumeral ligament is especially challenging in the lateral decubitus position and may result in stiffness and decreased external rotation postoperatively [60].

The examination under anesthesia (EUA) is a critical component of the procedure. It can provide information about the direction and extent of translation and may alter operative planning with regard to how much capsular plication to perform [60]. A patient's pain on physical exam in the office setting may lead to underestimation of the pathology or degree of instability of the shoulder. The range of motion should be assessed in elevation, external rotation with the arm adducted, and external and internal rotation with the arm abducted to 90°. Examining the shoulder for stability is performed by applying anterior, posterior, and inferior force while moving the arm throughout a range of abduction and rotation [79].

Portals

Proper portal placement is essential to perform an accurate diagnostic arthroscopy, appropriate soft tissue mobilization, and accurate placement of anchors. It is important to take sufficient time to mark out the location of the portal sites. Begin by clearly delineating the bone outlines of the acromion, distal clavicle, and coracoid with a surgical skin marker. Take care to mark out the inferior surfaces of the bone landmarks because portal entry points are measured from these surfaces [79].

After positioning the patient, standard posterior and anterosuperior portals are created and a thorough diagnostic arthroscopy is performed. If a Bankart lesion is identified, an additional mid-glenoid portal can be established at the 3 o'clock position on the glenoid using an 18-gauge needle to aid with localization slightly superior to the subscapularis tendon. Labral pathology at the 4–6 o'clock position can be difficult to address through these standard portals. Establishing a 7 o'clock portal approximately 2–3 cm lateral and 1 cm inferior to the posterior portal provides excellent access to the inferior aspect of the glenoid and may be used for percutaneous placement of anchors on the posterior and inferior aspects of the glenoid [60] (Fig. 16.9).

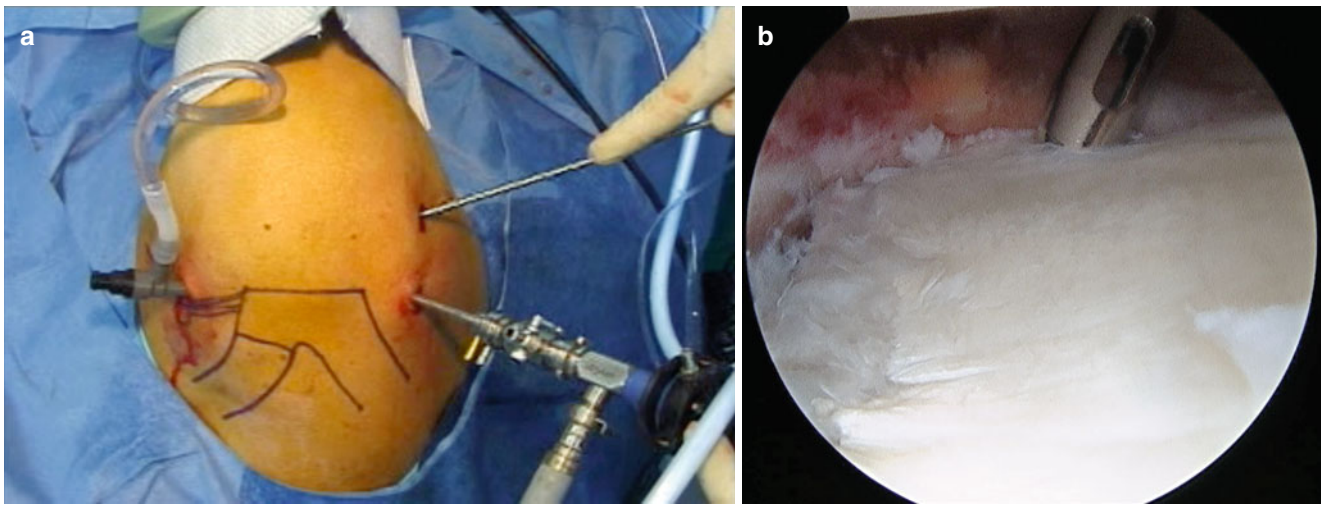

Fig. 16.9 Location of posterolateral portal in relation to standard anterior and posterior portals (**a**); arthroscopic view of instrumentation introduced through posterolateral portal (**b**)

The pathology of an anterior labral tear is best viewed from the anterosuperior portal. Visualization from this portal decreases the risk of the surgeon missing ALPSA lesions and allows one to more easily evaluate anteroinferior glenoid bone loss and the extent of the labral tear posteriorly [60]. With the arthroscope in the anterosuperior portal, it also affords excellent visibility while the surgeon works through the mid-glenoid portal, ensuring complete preparation of the tear and repair.

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

Diagnostic evaluation of the glenohumeral joint should be performed systematically in order to avoid overlooking any pathology. It is important to assess for pathology commonly associated with a Bankart lesion including a HAGL lesion, ALPSA lesion, and SLAP tears. Establish the standard posterior portal. After entering the joint, identify the biceps-labrum complex and rotate the camera to center the glenoid on the monitor screen. Inspect the rotator interval and superior glenohumeral ligament. The rotator interval may be widened or lax in patients with glenohumeral instability [33, 79].

Establish the anterior portal using the outside-in technique, with the goal of placing the portal in the center of the rotator interval. Evaluate the biceps tendon and pull the extra-articular portion of the tendon into the joint using a probe to inspect it for tearing or inflammation. Inspect the biceps-labrum complex using a probe inserted through the anterior cannula. Abduct and externally rotate the shoulder to determine whether the superior labrum elevates off the glenoid [79].

While maintaining the arthroscope in the posterior portal, evaluate the subscapularis recess and the superior border of the subscapularis tendon. Direct the arthroscope inferiorly to inspect the anterior labrum and the middle glenohumeral ligament. Evaluate the attachment of the anterior labrum to the glenoid using a probe passed through the anterior cannula. Fraying, tearing, or separation of the labrum from the glenoid may indicate instability [79]. With the arthroscope directed at the 5 o'clock position, inspect the inferior glenohumeral ligament. Assess both tension and insertion strength, using a probe.

Determine whether a "drive-through sign" is present inferiorly. This sign refers to the easy passage of the arthroscope between the humeral head and the glenoid, with the camera positioned at 6 o'clock. Recall that the drive-through sign indicates only glenohumeral laxity, not necessarily gross instability [79].

To view the posterior labrum from the posterior cannula, withdraw the arth roscope until it sits slightly anterior to the posterior capsule. Rotate the arthroscope until it points at the 6 o'clock position. Evaluate the posterior labrum for fraying, tears, or separation of the labrum. Continue inferiorly, to visualize the posterior-inferior glenohumeral ligament. Internally rotate the arm and note the tensioning of the ligament [79].

For thorough assessment of the rotator cuff tendons, direct the arthroscope superiorly. Abduct and externally rotate the shoulder until the anterior aspect of the supraspinatus tendon can be visualized. Inspect the cuff insertion from anterior to posterior and assess the insertion of the

supraspinatus on the humeral head. No exposed bone should be visible between the articular surface of the humeral head and the insertion of the supraspinatus tendon. Partial articular surface tears are present when some bone is exposed between the remaining intact supraspinatus tendon and the articular surface [79].

After evaluating the posterior insertion of the rotator cuff, direct the arthroscope inferiorly and externally rotate the shoulder. This allows visualization of the posterolateral humeral head and evaluation for the presence of a Hill-Sachs lesion [79]. Evaluate the humeral head and glenoid for signs of osteoarthritis.

Place the arthroscope in the anterior portal and reinspect the posterior labrum, capsule, and posterior rotator cuff. Position the shoulder in abduction and external rotation, to assess for internal impingement between the posterior-superior labrum and the posterior rotator cuff and capsule. Note the normal pear shape of the glenoid, which can be observed from this perspective. Loss of the normal inferior glenoid widening signifies bone loss in the anteroinferior glenoid and may be present in patients with glenohumeral instability [79].

Step-by-Step Procedure [78] (Figs. 16.10 and 16.11, Box 16.1)

- Examination of shoulder under anesthesia.
- Position patient (beach chair vs. lateral); we prefer lateral decubitus to allow ease of access to the entire 360° of the labrum.
- Mark bony landmarks (acromion, distal clavicle, and coracoid) on the skin.
- Place the posterior portal in line with the glenoid, which is nearly parallel to the lateral aspect of the acromion. Make this portal 2 cm distal and directly in line with the lateral edge of the acromion.
- Perform thorough diagnostic arthroscopy. Identify all pathology and formulate a plan.
- Place the anterosuperior portal high in the rotator interval, immediately posterior or just anterior to the biceps tendon first using an 18-gauge needle. Make the portal incision just anterior to the anterior edge of the acromion. Insert a switching stick.
- Create the anterior mid-glenoid portal, which is just above the subscapularis tendon. An 18-gauge needle is inserted from outside-in starting next to the coracoid and entering the joint immediately superior to the subscapularis tendon. The two anterior portals should be placed as widely apart as possible. An 8.25 mm cannula is inserted into the mid-glenoid portal and will serve as the primary working portal.
  - Ensure that the angle of approach coming into the joint will allow the appropriate angle for drilling and placement of anchors.
  - Evaluate ability to manipulate instrumentation and shuttle sutures inferiorly.
- The arthroscope is placed in the anterosuperior portal and will remain there for the duration of the case (switched over the switching stick).
- Adequately mobilize the anterior-inferior capsulolabral complex. Visualization of the subscapularis muscle fibers medial to the capsule-labral complex indicates adequate release of the capsulolabrum.
  - A bump can be placed in the axilla to lateralize the humeral head and improve visualization, versus a lateral translation strap that is well padded.
- After the labrum and capsular attachments to the glenoid are adequately released, roughen the glenoid with a burr or bone rasp to encourage soft tissue healing.
- Place the first anchor.
  - This may be done either from anterior or from a posterolateral percutaneous portal (7 o'clock portal – see manuscript for description).
  - The first anchor is placed near 6 o'clock position from either the posterolateral portal (percutaneous) or from the anterior mid-glenoid portal (via 8.25 mm cannula).
  - Ensure that the drill guide is well seated on the glenoid with a gentle mallet tap just prior to drilling to prevent slipping.
  - Insert the anchor per manufacturer recommendations and then begin capsulolabral repair from inferior to superior.
- Anterior-inferior capsulolabral repair.
  - Place a shuttling suture or passing device instrument at the most inferior location. This will be used to shuttle the non-absorbable suture from the most inferior anchor.
  - Place the initial suture anchor at the 6 o'clock position, 1–2 mm onto the articular surface of the glenoid and 5–10 mm cephalad to the shuttle suture to appropriately shift the tissue superiorly and retention the IGHL.
  - A combined stitch can be used to tension both the capsule and repair the labrum by passing a curved suture passer through the capsule 5–10 mm lateral to the labrum, exiting the capsule, reentering deep to the labrum, and emerging just lateral to the articular margin.
  - Repeat the process of shuttle/suture anchor placement until normal anatomy has been restored.
- A total of 3 anchors are utilized for a typical anterior instability repair, but more may be required if the tear

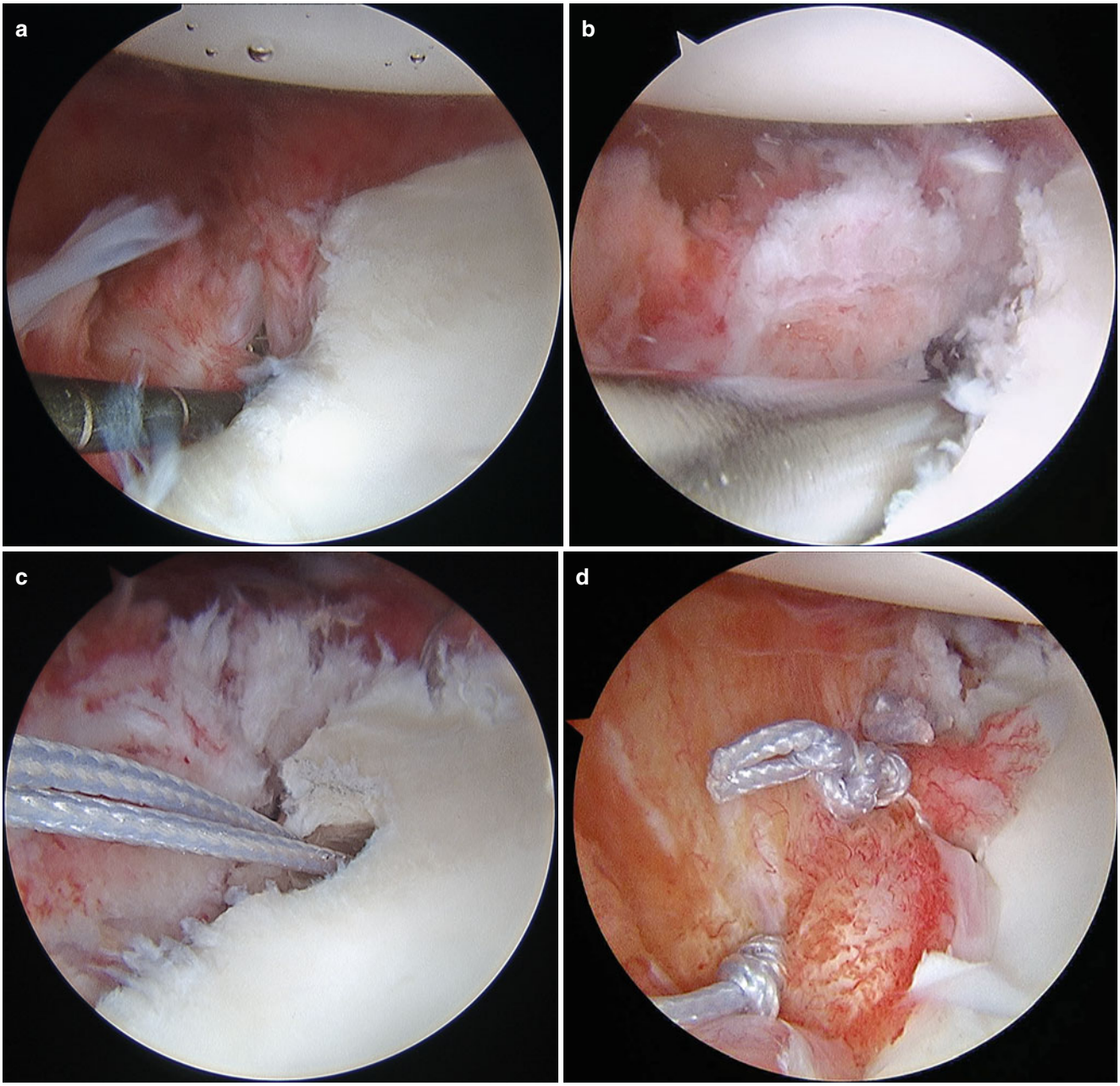

Fig. 16.10 Arthroscopic identification of an ALPSA lesion (**a**), preparation of the glenoid (**b**), and repair of the lesion (**c**, **d**)

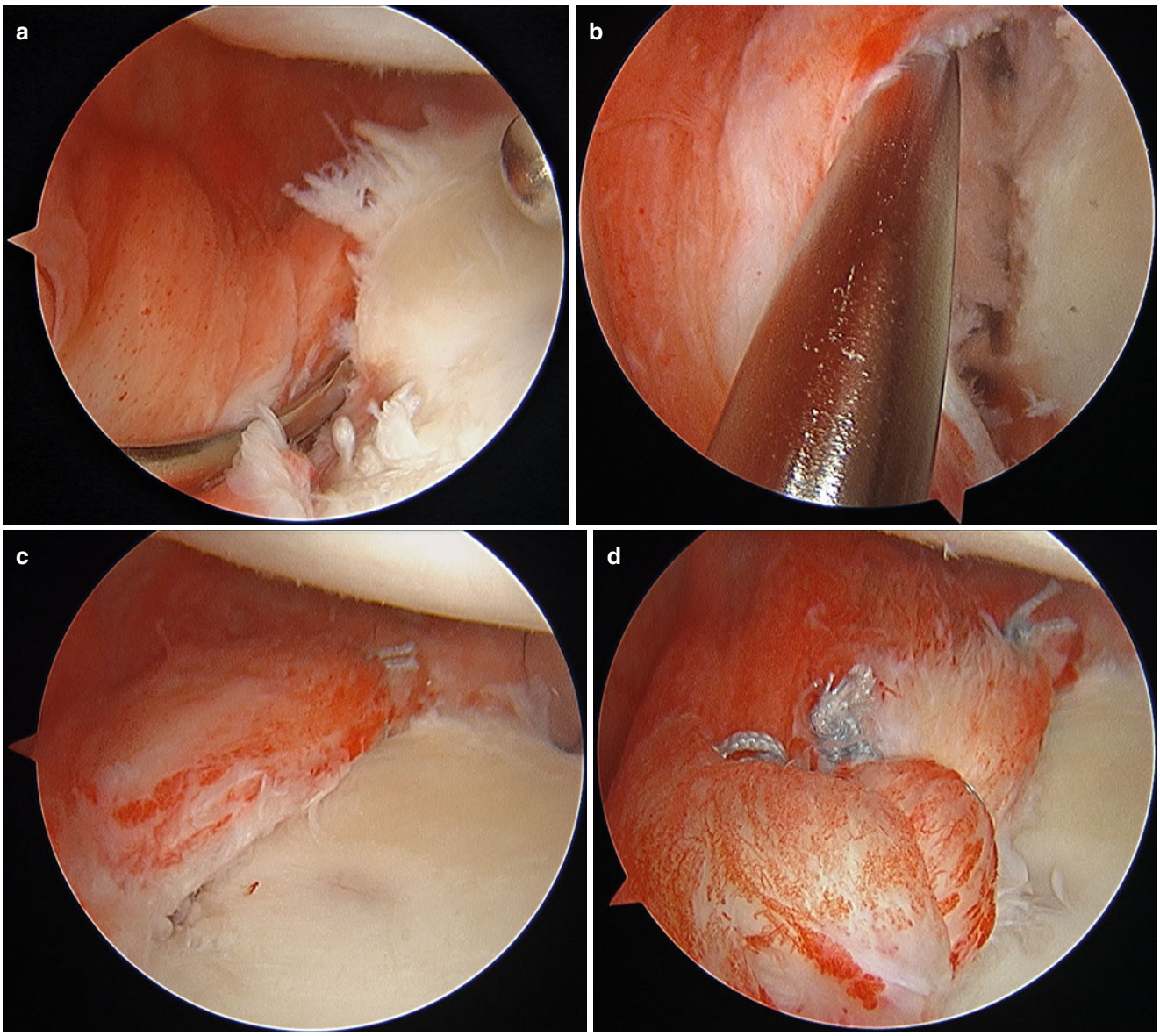

Fig. 16.11 A 26-year-old male with anterior labral tear 2 months out from initial injury with recurrent instability, demonstrating arthroscopic repair steps. Initial tear as viewed from the anterior superior portal (**a**); preparing the anterior glenoid with an elevator and 3.0-mm bone cutting shaver (**b**); first anchor at 6 o'clock inserted from the posterolateral (7 o'clock) portal (**c**); final repair construct with 3 total anchors (**d**)

extends posteriorly. The anchors are typically placed at the 5:30–6:00, 4:30, and 3–3:30 position, with 6 o'clock being the most inferior. Care is taken not to overtighten the labral tissue near the rotator interval as this may constrict the mobility of the capsule and labrum, thereby limiting external rotation at the side.

- The arthroscope may be moved to the posterior portal prior to placement of the last anchor if it is too "crowded" to place the final superior anchor (usually about the 3 o'clock position) with the scope in the anterosuperior portal.
- The arthroscopic wounds are closed in standard fashion, and the wounds are covered with a dry sterile dressing, followed by placement of the affected arm in a padded abduction sling.
- The patients participate in an arthroscopic instability therapy protocol with a sling for approximately 5–6 weeks, starting with strengthening of the scapular stabilizers and passive motion of the shoulder with flexion to 90–120°, abduction to 45, but avoiding abduction and external rotation combined for about 4–5 weeks. Progressive ROM is started at 4–6 weeks, and then a gradual strengthening program including more scapular stabilizing and strengthening exercises. After ROM is restored and the patient has good scapular control, they are allowed to return to sport-specific training and then full activities around 5–6 months.

Box 16.1: Tips and Tricks

- In the lateral decubitus position, the posterior portal is made in line with the lateral edge of the acromion and 1 cm inferior to the posterior tip. This allows for a slightly downward trajectory from the posterior portal, thereby facilitating instrumentation during the case.
- The anterosuperior portal is positioned high in the rotator interval. Following the diagnostic arthroscopy, the arthroscope can be transferred to this portal for excellent visualization of the anterior glenoid.
- The mid-glenoid portal is also created in the rotator interval, slightly proximal to the subscapularis tendon. It is important to provide at least a 2- to 3-cm skin bridge between the anterosuperior and mid-glenoid portals in order to avoid crowding intra-articularly while performing the case.
- A posterolateral portal (7 o'clock position) may be created to allow for percutaneous anchor placement. Additionally, a small cannula may be inserted to facilitate glenoid anchor placement and repair of the labrum inferiorly.
- The axillary nerve is most vulnerable at the 6 o'clock position (12.5–15 mm from the glenoid), which increases with abduction.

Postoperative Care

Preoperative patient counselling in conjunction with intraoperative findings and exact surgical procedure help determine the appropriate postoperative rehabilitation program. Several factors are important to take into account including the type of pathology, direction of instability, quality of the tissue at the time of repair, and any other associated injuries (e.g., rotator cuff tear, biceps tendon tear). The authors recommend an abduction sling for the majority of instability repairs, as it maintains the shoulder in a neutral to slightly externally rotated position.

Physical therapy often begins 7–10 days following most routine instability repairs. Gradual progression with passive and active-assisted range of motion will occur over the first 4 weeks (forward elevation [FE] to 130°; external rotation [ER] to 30°). Between 4 and 6 weeks, these ranges increase to FE 130–180° and ER 30–60°. The subsequent weeks focus on progressing active range of motion with resistive strengthening being incorporated at 8–12 weeks and return to full sports and normal activities at 4–6 months in most cases [60].

Literature Review

Numerous studies have examined arthroscopic versus open repair of recurrent glenohumeral instability. Tables 16.2 and 16.3 summarize the results of the literature over the past 5 years. The rate of recurrent instability following arthroscopic treatment ranges from 2 to 18 % (Table 16.2), while that for open management ranges from 0 to 9 % (Table 16.2). These studies support the dramatic improvement in arthroscopic management of this complex problem and demonstrate that the success rate of arthroscopic treatment essentially equals that of open management, with the added benefit of decreased morbidity. Regardless of the technique, the overall goal of instability surgery is to restore anatomic alignment of the labrum with the glenoid.

Table 16.2 Summary of literature published between 2007 and 2012 on the results of primary arthroscopic treatment of recurrent glenohumeral instability

Author	Year	Study type	Procedure	Sample size	Follow-up (months)	Primary outcome variable	Primary outcome scores	Physical exam	Recurrences no.	Factors associated with recurrence
Ahmed et al. [80]	2012	Retrospective review	Bankart repair and capsular shift	302	60	Recurrent instability	WOSI DASH	Not done	40 (13.2 %)	Severity of glenoid bone loss (≥25 %), engaging Hill-Sachs lesion, age at surgery (<age 20)
Zaffagnini et al. [81]	2012	Retrospective review	Modified Caspari	49	13.7 ± 2.2 years	Redislocation	Rowe UCLA Constant	Not done	6 (12.5 %)	Six patients suffered redislocations; however, the mechanism is not described. Specific factors associated with recurrence were not described
Boileau et al. [82]	2012	Case series	Remplissage and Bankart repair	47	24	Recurrent instability	Rowe Walch-Duplay Constant-Murley Subjective Shoulder Value (SSV)	↓ ER	1 (2.1 %)	Redislocation associated with a fall while playing basketball. Specific factors associated with recurrence were not described
Netto et al. [83]	2012	Randomized controlled	Bankart repair	17	37.5 (20–56)	Recurrent instability	DASH	No ↓ ROM	2 (12 %)	Both patients suffered redislocation and required surgical intervention with an open technique. Specific factors associated with recurrence were not described
Mahirogullari et al. [84]	2010	Prospective randomized	Bankart repair	34	26.6 (12–51)	Recurrent instability	Rowe VAS	↓ ER	2 (5.9 %)	Redislocation following a fall while playing football and while descending stairs. Specific factors associated with recurrence were not described
Voos et al. [20]	2010	Case series	Bankart repair	83	33 (24–49)	Recurrent instability	ASES L'Insalata VAS	No loss of ER	13 (18 %)	<age 25, ligamentous laxity, Hill-Sachs lesion >250 mm^3
Porcellini et al. [85]	2009	Prospective cohort study	Bankart repair	385	36	Redislocation	None	Not done	31 (8.1 %)	Male sex, age at first dislocation (<age 22), time from first dislocation until surgery (>6 months after dislocation)
Robinson et al. [86]	2008	Randomized controlled	Bankart repair	42	24	Recurrent instability	WOSI DASH	No ↓ ROM	3 (7 %)	All three patients suffered a redislocation. The mechanism of injury was not described, nor were specific factors associated with recurrence

Table 16.3 Summary of literature published between 2007 and 2012 on the results of primary open treatment of recurrent glenohumeral instability

Author	Year	Study type	Procedure	Sample size	Follow-up (months)	Primary outcome variable	Primary outcome scores	Physical exam	Recurrences no.	Factors associated with recurrence
Netto et al. [83]	2012	Randomized controlled	Open Bankart repair	25	37.5 (20–56)	Recurrent instability	DASH	No ↓ ROM	0 (0 %)	Not described
Zaffagnini et al. [81]	2012	Retrospective review	Open capsular shift and Bankart repair	33	15.7 ± 2.2 years	Redislocation	Rowe UCLA Constant	Not done	3 (9 %)	Three patients suffered redislocations; however, the specific factors associated with recurrence were not described
Emami et al. [87]	2011	Retrospective review	Bristow-Latarjet	30	2–8 years (mean 5 year)	Recurrent instability	Rowe	↓ ER	0 (0 %)	Not described
Mahirogullari et al. [84]	2010	Prospective randomized	Bankart repair	30	26.1 (12–52)	Recurrent instability	Rowe VAS	↓ ER	1 (3.3 %)	Redislocation following a fall 12 months post-op. The specific factors associated with recurrence were not described
Rahme et al. [88]	2010	Prospective cohort	Bankart repair	68	63 (27–110)	Recurrent instability	Rowe	↓ ER	4 (5.9 %)	One patient experienced a single redislocation during sleep, while the other 3 patients suffered multiple redislocations during sports activities. The specific factors associated with recurrence were not described
Ogawa et al. [89]	2010	Retrospective review	Open Bankart repair	167	8.7 years (mean 5–20 year)	Recurrent instability OA	Rowe	↑ ROM	8 (4.8 %)	One patient suffered a redislocation, while the other 7 experienced subluxation events. The specific factors associated with recurrence were not described
Uchiyama et al. [90]	2009	Case series	Open modified inferior capsular shift	50	61 (24–172)	Recurrent instability	Rowe UCLA	↓ ER	3 (5.8 %)	One patient suffered a redislocation 24 months post-op. The other 2 patients experienced a single subluxation event during judo practice (at 3 and 4 months post-op). The specific factors associated with recurrence were not described
Pagnani [91]	2008	Case series	Open Bankart and capsular shift	103	24 (24–74)	Recurrent instability	ASES	↓ ER	2 (2 %)	One patient suffered a redislocation and another patient experienced a subluxation. The specific factors associated with recurrence were not described

Summary

Successful management of patients with anterior shoulder instability is challenging and depends largely on the accurate diagnosis and treatment of the underlying pathology. Awareness of the pathoanatomy contributing to recurrent anterior glenohumeral instability is paramount in order to appropriately manage this complex problem. A thorough understanding of the principles of anterior instability repair combined with the pearls provided should allow for the comprehensive approach to patients with anterior shoulder instability and ultimately lead to improved patient outcomes.

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Posterior Shoulder Instability

17

Yung Han and Seung-Ho Kim

Introduction

Posterior instability of the shoulder has historically been poorly recognized. This is likely in part due to the rarity of the condition, lack of awareness, and subtle imaging findings. Acute posterior dislocation has a clear clinical presentation, but is reported to be missed between 60 and 78 % on initial presentation [1–3]. Recurrent posterior instability is even more challenging to diagnose because the clinical presentation is often vague and there is generally a poor awareness and understanding of the pathology. As a result, patients, especially those without a discernible inferior component of laxity, are often overlooked or misdiagnosed as having other pathology such as superior labral lesions, internal impingement, rotator cuff disease, or scapulothoracic dysfunction.

However, recent advances in the concept of posterior instability have provided us with reasonable insight into the pathology, pathogenesis, diagnostic examinations, and treatment options. It is important to recognize that most posterior instability is microtraumatic and often presents with bidirectional posterior and inferior instability. Since it has various degrees of inferior components of instability, posterior instability often overlaps with multidirectional instability in its diagnosis, clinical presentation, and management. Therefore, the pathology, pathogenesis, and treatment options described in this chapter are also applicable to multidirectional instability.

Three fundamental questions should be addressed when dealing with patients with posterior or posteroinferior instability. First, what is the principle cause for the patient's instability? Pathology can generally be categorized as "born loose" (atraumatic), "worn loose" (microtraumatic), or "torn loose" (traumatic). It is important to recognize severe bony abnormalities and differentiate between hyperlaxity and instability. Secondly, who needs surgical treatment? By answering this question, we can identify patients who likely will not respond to conservative treatment and will benefit from early surgical treatment. And lastly, once surgery is indicated, what type of surgical procedure should we perform? In this chapter, we will discuss previous biomechanical and clinical studies to answer these 3 questions and detail our surgical approach in treating posterior instability.

Epidemiology

Posterior instability is rare representing only 2–10 % of all shoulder instability cases [4–6]. A recent epidemiological study [7] showed the prevalence of acute posterior dislocation was 1.1 per 100,000 persons per year with a bimodal distribution of males in their 20s–40s and elderly patients over 70 years old. Two-thirds were a result from trauma and one-third were produced during seizures. There was a 17.7 % recurrence of instability and acute traumatic posterior dislocations rarely resulted in recurrent instability. The risk factors for recurrent instability after acute traumatic posterior dislocations were age less than 40, dislocation as a result from seizure, and large reverse Hill-Sachs lesion.

Unlike anterior instability, traumatic posterior dislocations rarely result in recurrent instability and the most common cause of posterior instability is believed to be from repetitive microtrauma. Patients typically are young and athletic or have high physical demands such as military cadets. At the time of surgery, labral lesions have been identified 10–100 % of the time [4, 8–12]. Interestingly, the studies that used arthroscopy found a higher incidence of labral lesions compared with open surgery suggesting that arthroscopy is the gold standard for detecting these subtle lesions. With a thorough arthroscopic examination and appropriate patient selection, labral lesions should be present 100 % of the time at the time of surgery [9, 13, 14].

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Pathophysiology

An optimal functioning shoulder requires a high degree of both mobility and stability. It is fundamentally difficult to optimize both of these components. The ability of the shoulder to find this balance can be attributed to its anatomy. In order to properly treat shoulder instability, it is important to appreciate the anatomy and understand the biomechanical principles governing stability so that these concepts can be used to tailor a surgical approach to correct the pathoanatomy and restore the biomechanics.

The glenohumeral joint is often compared to a golf ball on a tee; however, the joint is more stable than this analogy suggests since this only compares the bony stability and fails to account for the capsulochondrolabral complex and rotator cuff muscles as important stabilizers. The glenoid socket is shallow and thereby sacrifices stability for mobility. This is in contrast to the deep socket of the acetabulum, another ball-and-socket joint, which has more stability but limits motion by impingement. Therefore, it may be reasonable to assume that its ligaments are largely responsible for its stability. However, unlike joints that rely on taught ligaments for its stability, the ligaments in the glenohumeral joint are relaxed through the majority of its range of motion becoming taught only at the extremes. Stability, therefore, is a complex relationship between its static and dynamic stabilizers. The static stabilizers are the bony anatomy (glenoid and humerus), the capsulochondrolabral complex (capsule, labrum, cartilage, glenohumeral ligaments, and rotator interval), and coracohumeral and coracoacromial ligaments, while the dynamic stabilizers are the rotator cuff muscles, deltoid muscle, and possibly the long head of the biceps. The posteroinferior capsule is not as robust as the anterior capsule, and the posterior inferior glenohumeral ligament (PIGHL) is thinner than its anterior counterpart [15]. The PIGHL is the most important stabilizing ligament during the posterior loading position of flexion, adduction, and internal rotation, and the subscapularis is considered to be the most important dynamic posterior stabilizer. The rotator interval is believed by some to be an important posterior stabilizer as it has been shown to resist posterior and inferior translation [16, 17]; however, others believe that its role in stability is not significant [18, 19].

Concavity compression is the most significant mechanism of stability in the glenohumeral joint [20]. The static stabilizers offer the concavity whereby the dynamic stabilizers provide a compressive force on the humeral head into the glenoid cavity to maintain stability. As concavity or compression is increased, a larger displacing force is needed to displace a ball from its socket. The glenoid socket is deepened remarkably by the articular cartilage and labrum. Meanwhile, the rotator cuff muscles predominantly apply a compressive force to center the humeral head into the glenoid cavity.

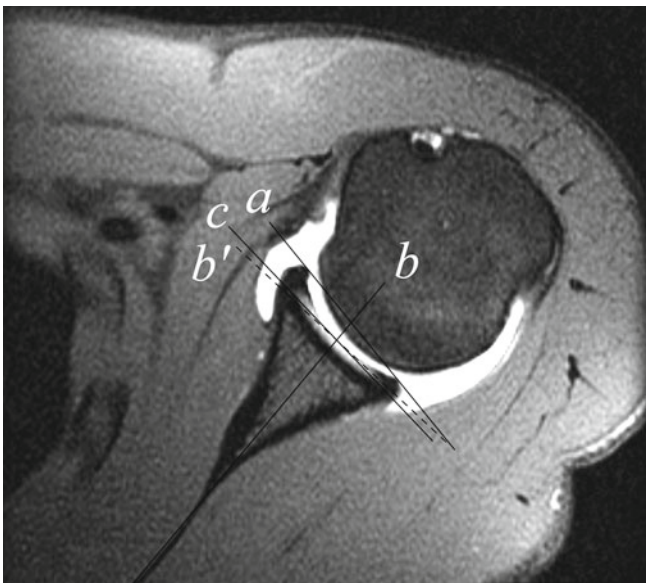

Fig. 17.1 An axial image of an MRA of the right shoulder with atraumatic posteroinferior instability. The retroversion of the (*a*) chondrolabral glenoid is greater than the retroversion of the (*c*) bony glenoid (*b´* signifies neutral glenoid version) and (*b*) signifies reference line representing the plane of the scapular body

Cadaveric studies have shown that an intact labrum resisted tangential forces of up to 60 % of the compressive load [21] and resection of the chondrolabral complex reduced the glenoid height by 80 % and reduced the stability ratio (displacing force divided by compressive force) by 65 % [22]. Ideally, the compressive force should be directed towards the center of the concavity. Stability can be decreased when this is not the case such as in muscular imbalance or when the position of the concavity is changed such as in glenoid retroversion or scapulothoracic dysfunction. Other mechanisms that promote stability, particularly at rest, are negative pressure within the capsule, glenohumeral suction cup effect, and adhesion-cohesion.

Cadaveric studies showed the importance of the chondrolabral complex in stability, and Kim et al. [23] emphasized this clinically. By examining four parameters of glenohumeral containment (bony and chondrolabral version, labral height, and glenoid depth) on T2-weighted axial magnetic resonance arthrogram (MRA) images of patients with atraumatic posteroinferior instability and a control group of people without shoulder pathology, they found that the posteroinferior instability group had increased bony and chondrolabral retroversion in the middle and lower axial images, with the chondrolabral retroversion being more pronounced (Fig. 17.1). The conclusion was that loss of chondrolabral containment posteroinferiorly is a consistent finding in posteroinferior instability and is principally due to loss of posterior labral height. Similar findings were subsequently reported by Bradley et al. [24].

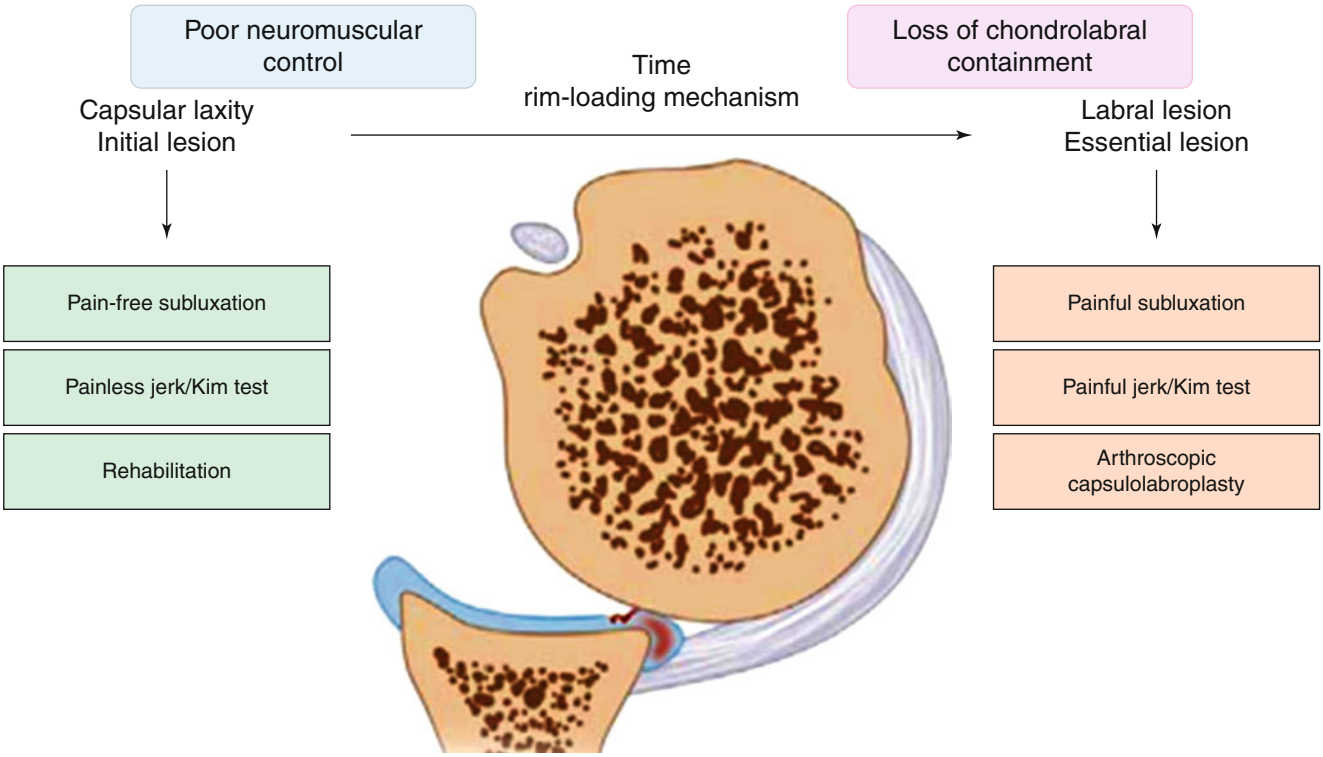

Fig. 17.2 The Rim-loading mechanism. Capsular laxity is the initial lesion in posteroinferior instability. Shoulders with capsular laxity are asymptomatic or minimally symptomatic, and clinical tests present with a painless clunk. However, with poor proprioception and neuromuscular control, rim loading occurs during repetitive subluxations. This leads to a loss of chondrolabral containment and a posteroinferior labral lesion, which is the essential lesion responsible for the shoulder symptoms and painful clunk (Modified from Kim [41]. Copyright: Elsevier 2011. Reproduced with permission)

It was unknown whether the osseous and chondrolabral retroversion is a cause or consequence of posterior instability. However, Kim et al. [13, 23] proposed that the loss of chondrolabral containment is a result of cumulative microtrauma to the posterior or posteroinferior glenoid labrum. A rim-loading mechanism of the humeral head onto the glenoid rim causes plastic deformation of the capsulochondrolabral complex with gradual retroversion and loss of labral height. This theory also suggests that patients with inborn laxity who can subluxate asymptomatically can develop symptomatic instability by repetitive subluxation and damage to the labral rim (Fig. 17.2).

Laxity is often mistaken for instability and it is important to differentiate the two. Laxity can be defined as an increased *painless* translation of the humeral head to or past the glenoid rim, whereas instability is an uncomfortable or *painful* translation of the humeral head to or past the glenoid rim. Studies have shown that there is no difference in laxity between healthy patients and those with instability [25–28]. This suggests that there is a different pathology to instability other than just capsular volume, and this difference may be due to a loss of proprioception. In fact, studies have shown that patients with laxity and instability have poorer muscle coordination and proprioceptive function [29–31]. The capsule and its ligaments have neural structures and mechanoreceptors [32] providing an afferent feedback that mediates joint position sensibility and muscular reflex stabilization. If this mechanism is lost, then the body cannot sense to increase compression by reflex to center the glenohumeral joint during unfavorable forces, and consequently rim loading occurs.

It has previously been reported that a redundant or patulous capsule is the main reason for posterior or multidirectional instability, and the reported incidence of labral lesions at the time of surgery varied among 10 and 100 % [4, 8–12]. Kim et al. [9, 13, 14] reported that labral lesions are found in all instability cases at the time of surgery. While many of these may be obvious on arthroscopy, nearly 40 % are discreet lesions that were likely missed previously [14]. The pathogenesis of the Kim's lesion [13] is repetitive posterior rim loading that gradually creates an avulsion tear at the insertion of the PIGHL. The reason why this lesion is hidden is because the avulsion begins at the deep portion where the PIGHL inserts and the tear propagates laterally to the surface of the cartilage and labrum. These lesions are highlighted by three characteristics: (1) chondrolabral retroversion, (2) a subtle and marginally cracked superficial surface at the

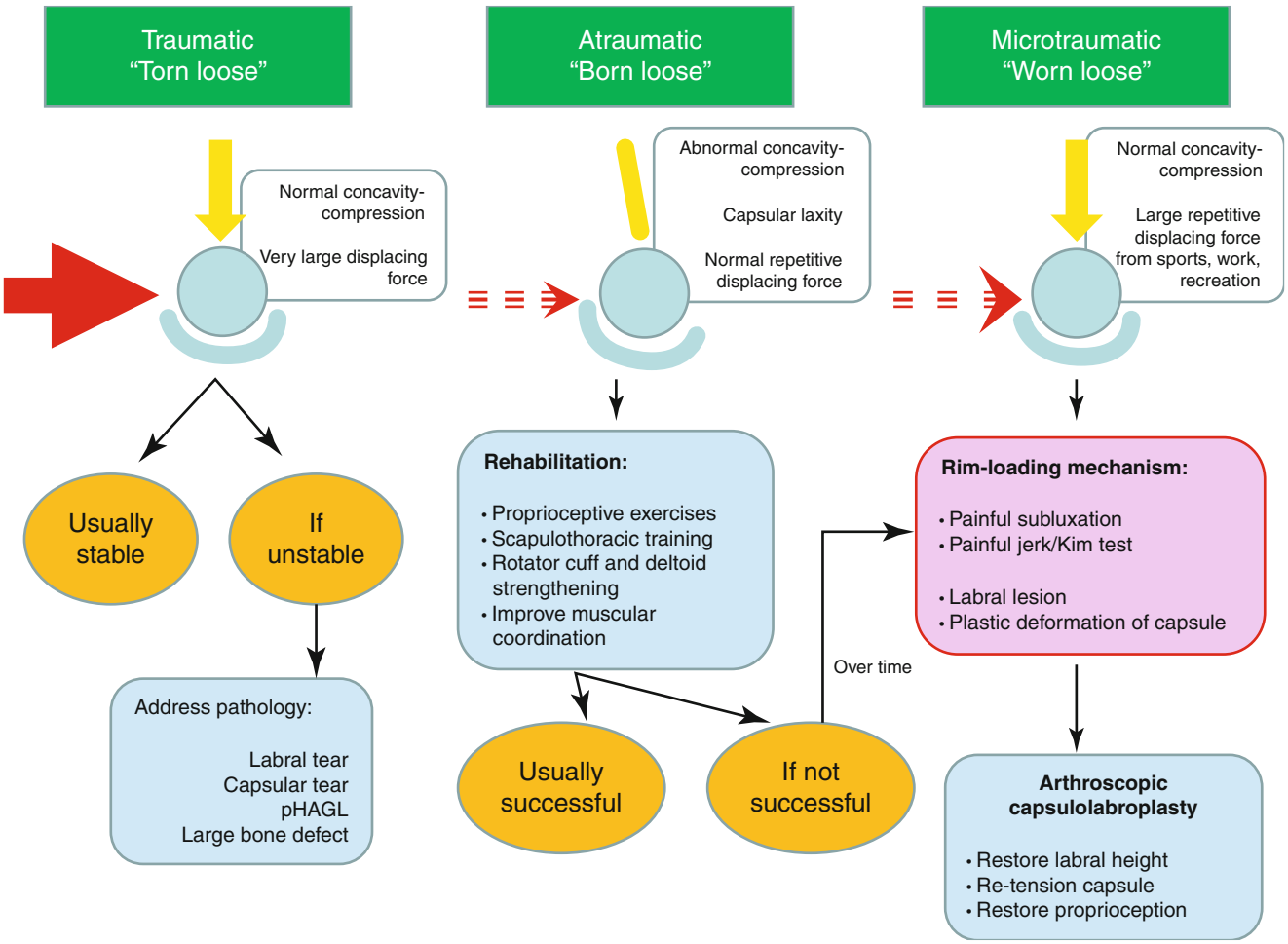

Fig. 17.3 Overview diagram of the pathogenesis and treatment goals of posterior instability using the concavity-compression model. Note the model of the humeral head within the glenoid. The *yellow arrow* represents compression from the rotator cuff muscles of the humeral head into the chondrolabral glenoid concavity. The *red arrow* represents the displacing force. Arthroscopic capsulolabroplasty is indicated when an essential labral lesion develops from the rim-loading mechanism

chondrolabral junction, and (3) a concealed deep and incomplete labral avulsion tear. Whereas previous surgical treatment involved a posterior capsular plication and shift, Kim et al. [13] suggested to probe the cracked surface, complete the tear, and perform a capsulolabroplasty to rebuild the labral height and retention of the capsulolabral complex.

The concept that all instability patients have labral lesions during surgery is important in guiding treatment principles and is only correct with appropriate patient selection and a thorough arthroscopic examination. The author (SHK) found that patients with a painless clunk on the jerk test responded well with rehabilitation [33]. Whereas those patients with a sharp pain and click or clunk did not respond with conservative treatment, but did well after capsulolabroplasty. Therefore, patients were selected for operative treatment based on the suggestion of a labral tear on physical examination. The authors believe that patients with laxity or mild symptomatic instability do not have a labral lesion and will benefit from physical therapy aimed at improving proprioception, muscular strengthening, and coordination. However, instability patients with a positive jerk and Kim's test [34] have a 97 % sensitivity for a labral tear and are thus recommended for surgical treatment after MRA confirmation. Furthermore, patients with laxity or mild discomfort with instability who do not regain proprioceptive and neuromuscular control can develop labral tears over time by the rim-loading mechanism and become more symptomatic as illustrated in Fig. 17.2. Surgery not only restores chondrolabral containment but also has been shown to restore proprioceptive function [30].

In summary, the pathogenesis and treatment goals of shoulder instability can be explained by using the concavity-compression model (Fig. 17.3). In traumatic posterior instability, there is a very large displacing force (e.g., fall or seizure) that overwhelms the concavity-compression mechanism. Recurrent dislocation rarely occurs. In atraumatic

posterior instability, the least common presentation, there may be glenoid hypoplasia, excessive glenoid retroversion, or increased humeral retroversion. Laxity and proprioceptive deficits are very common and there may be a defect in the connective tissue. Therefore, the concavity-compression system may not work as efficiently thereby allowing normal displacing forces to cause subluxation or dislocation. Treatment is typically rehabilitation aimed at maximizing the concavity-compression system with rotator cuff and deltoid strengthening, proprioceptive exercises, and scapulothoracic training. Lastly, in microtraumatic posterior instability, the most common form, there is repetitive microtrauma in the form of rim loading that eventually creates plastic deformation of the capsule and a labral tear, thus compromising the concavity. These patients typically do not respond to a rehabilitation program alone, and surgery is aimed at restoring the concavity. It should be noted that if the concavity-compression system is not restored in atraumatic instability, recurrent subluxations and dislocations can damage the chondrolabral complex similarly to the microtraumatic group, and therefore require surgery. The goals of surgery should be to restore the labral height and retention of the capsulolabral complex. This additionally can restore proprioceptive function and a rehabilitation program should be instituted to further optimize concavity compression.

History

The key to diagnosing posterior instability starts with the history followed by physical examination and imaging. Since the clinical presentation of posterior dislocation is often vague, a high index of suspicion is needed to make the correct diagnosis. An understanding of the pathogenesis and who the high-risk patients are is essential.

The first step, like any medical history, is to understand the chief complaint. Patients will rarely complain specifically of posterior instability. Frank posterior dislocations with a formal reduction are very uncommon, and patients may not have the insight to know that their glenohumeral joint is subluxating posteriorly. More commonly patients will complain about pain and/or shoulder fatigue or weakness with sports or activities. Seldom, patients will be brought in by family because they can pop their shoulder in and out.

The onset of symptoms should be ascertained including a trauma history which is essential in understanding the patient's pathogenesis. This should not easily be disregarded because patients rarely recall a single, major traumatic event. Rather the traumatic event is usually discreet and sometimes remembered if carefully asked. The course is usually insidious and identifying high-risk patients for repetitive posteriorly directed trauma (e.g., blocking in football, bench pressing, throwing, racquet activities, and swimming) is a major clue. If there is no traumatic history, then a possible connective tissue or bone disorder should be investigated. A general instability history should also be obtained with hyperlaxity being assessed on physical examination.

Symptoms range from pain, fatigue, weakness, or instability and may be associated with mechanical symptoms such as clicking or catching. These symptoms typically occur in activities involving a provocative position of flexion, adduction, and internal rotation such as in bench press or blocking in football. They may also occur in specific phases of activities such as the backhand stroke in racquet sports, the pull-through phase of swimming, and the follow-through phase in throwing and golf. It is important to gauge the severity, course, and any previous treatment as this should help guide a treatment plan.

If voluntary dislocation is present, it is important to differentiate between positional and habitual dislocation. Patients with positional dislocation are able to dislocate or sublux their shoulder in the provocative position of flexion, adduction, and internal rotation. These patients should not be excluded from surgical management and should not be confused with habitual dislocators who are able to willfully dislocate their shoulder due to muscle imbalances. There is often a secondary gain or other psychological problems and these patients should receive appropriate referral for a psychiatric assessment. After prolonged habitual dislocation, these patients may progress to unwanted dislocation and subluxation, but generally will improve with rehabilitation.

Clinical Examination

The physical examination for posterior instability starts with a proper orthopaedic examination of both shoulders including inspection, palpation, range of motion, examination of the joint above (cervical) and below (scapulothoracic), neurologic exam, and special tests. The special tests for posterior instability that are most useful are the jerk and Kim test. Additionally, the patient's general and glenohumeral laxity should be assessed.

Hyperlaxity is typically a congenital phenomenon that may be part of a connective tissue disorder (e.g., Marfan's syndrome, Ehlers-Danlos syndrome, osteogenesis imperfecta, or benign joint hypermobility syndrome) or may manifest alone. The latter is thought to be due to incomplete genetic penetrance. Acquired joint laxity may be acquired through training and competition via stretching the capsuloligamentous restraints. General hypermobility can be assessed with the *Beighton score* where a score greater than 4 on a 9-point scale is diagnostic. A point is given for the ability to passively bend the thumb back to the flexor aspect of the forearm per side, dorsiflex the 5th metacarpophalangeal

joint past 90° per side, hyperextend the knee greater than 10° per side, hyperextend the elbow greater than 10° per side, and touch the floor with both palms by forward flexion of the trunk from a standing position with the knees extended.

To test for glenohumeral laxity, the asymptomatic shoulder should be tested first to appreciate the patient's baseline laxity. Inferior laxity can be evaluated with the *sulcus sign*. A downward traction force is applied to the neutral shoulder in the upright position. The downward movement of the humeral head relative to the lateral acromion is measured in centimeters, and the grading scale is according to the change in movement: 1+ is less than 1 cm, 2+ is 1–2 cm, and 3+ is greater than 2 cm. Anterior and posterior translation can be measured using the *load and shift test*. An axial load is applied to the humerus in-line with the scapular plane to center the humeral head within the glenoid and then an anterior- or posterior-directed force is applied on the humeral head observing the amount of translation. Grade 0 has little to no movement; grade I is when the humeral head reaches the glenoid rim; grade II is when the humeral head dislocates but spontaneously reduces; and grade III is when the humeral head dislocates and does not spontaneously reduce.

Tests for instability have recently evolved. The posterior apprehension sign and the load and shift test are not believed to be diagnostically useful tests for posterior instability. The *jerk test*, also known as the posterior stress test, has been considered to be highly sensitive, but some authors have focused on the subluxation and/or relocation "jerk" sound while neglecting the pain component. Kim et al. [33] studied the importance of the pain component in the jerk test and its predictive therapeutic value. Patients with posteroinferior instability were divided into a painless jerk group and painful jerk group at the time of presentation and were prospectively followed during a 6-month rehabilitation program. They found that 93 % of the painless jerk group responded favorably to rehabilitation at a mean of 4 months while only 16 % of the painful jerk group responded favorably. They concluded that a painful jerk test has predictive value in the success of nonoperative treatment. Additionally, Kim et al. [34] in a separate study presented a new test, the *Kim test*, which is a modification of the jerk test to diagnose posteroinferior instability. They found that the jerk test was more sensitive in diagnosing predominantly posteriorly located labral lesions while the Kim test was more sensitive in diagnosing predominantly inferiorly located labral lesions. However, when both tests were combined, the sensitivity of diagnosing posteroinferior labral lesions was 97 %.

The jerk and Kim tests are both used as provocative tests attempting to reproduce painful posterior subluxation. The basis for both tests is to load the glenoid rim with the humeral head provoking a painful response from an essential labral lesion and assessing the degree of instability. The presence of pain, a click, or a clunk should be noted, and a painful click or painful clunk is considered to be a positive test. The jerk test can be performed as previously described by using one hand to stabilize the scapula and the other hand to move the patient's arm to try to sublux the humeral head posteriorly (Fig. 17.4). The jerk and Kim test can be performed

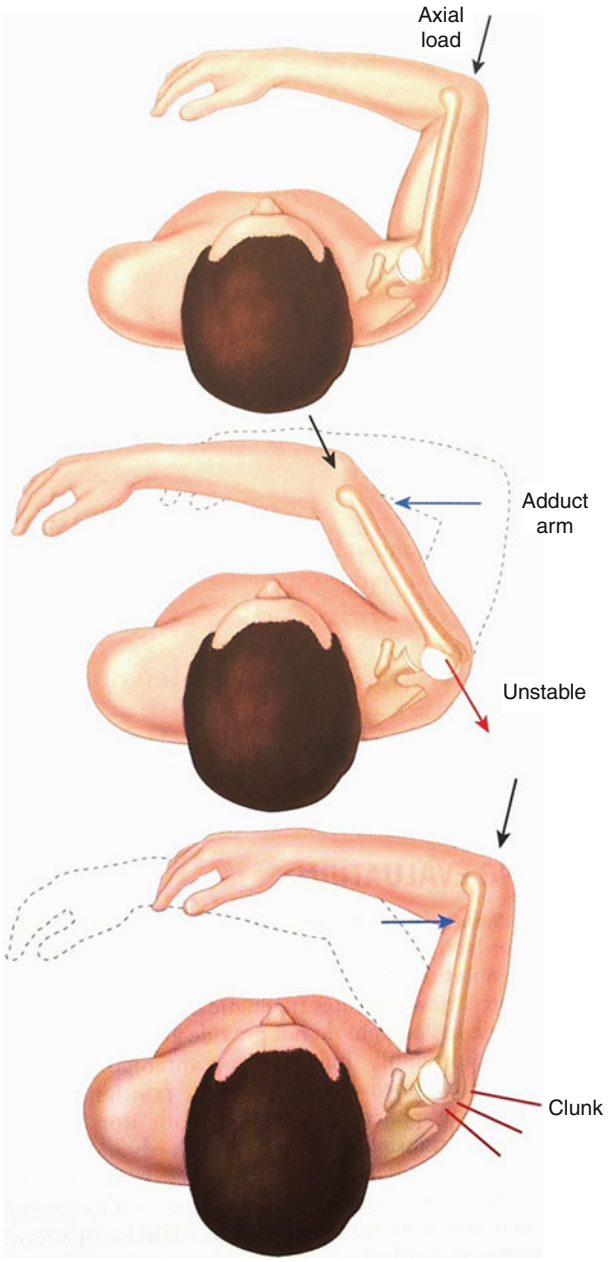

Fig. 17.4 The jerk test. Stabilize the scapula with one hand, while the other hand holds the elbow with the arm in 90° abduction and internal rotation. Firm axial compression force is applied on the glenohumeral joint. The arm is horizontally adducted while maintaining the firm axial load (From Matsen et al. [42])

similarly with the patient in a seated position while an assistant stabilizes the patient's torso. In evaluating the patient's right arm, the examiner holds the lateral aspect of the patient's right arm with his left hand and the patient's right elbow with his right hand. The patient's arm is flexed 90° and abducted so that it is in-line with the scapular plane. The arm is internally rotated 90° and the elbow is flexed to 90°. With the patient relaxed, the examiner loads the humeral head into the glenoid, and then directs the force of the humeral head to the posterior glenoid by adducting the patient's arm while continuing to apply an axial load. The examiner's right arm primarily controls the adduction while the examiner's left arm helps guide the humeral head posteriorly. This maneuver should effectively rim-load the posterior aspect of the glenoid and cause subluxation in a lax or unstable shoulder. An audible click or clunk can be heard at the time of subluxation and also upon relocation. An associated sharp pain is considered a positive finding. The Kim test is performed similarly, but the goal is to load the posteroinferior glenoid. This is done by simultaneously flexing and adducting the patient's arm during the adduction maneuver (Fig. 17.5). The examiner's right hand primarily controls the adduction and simultaneous 45° diagonal elevation while the examiner's left hand guides the humeral head posteroinferiorly. Positive findings are strongly suggestive of a labral tear and have been confirmed arthroscopically [33, 34].

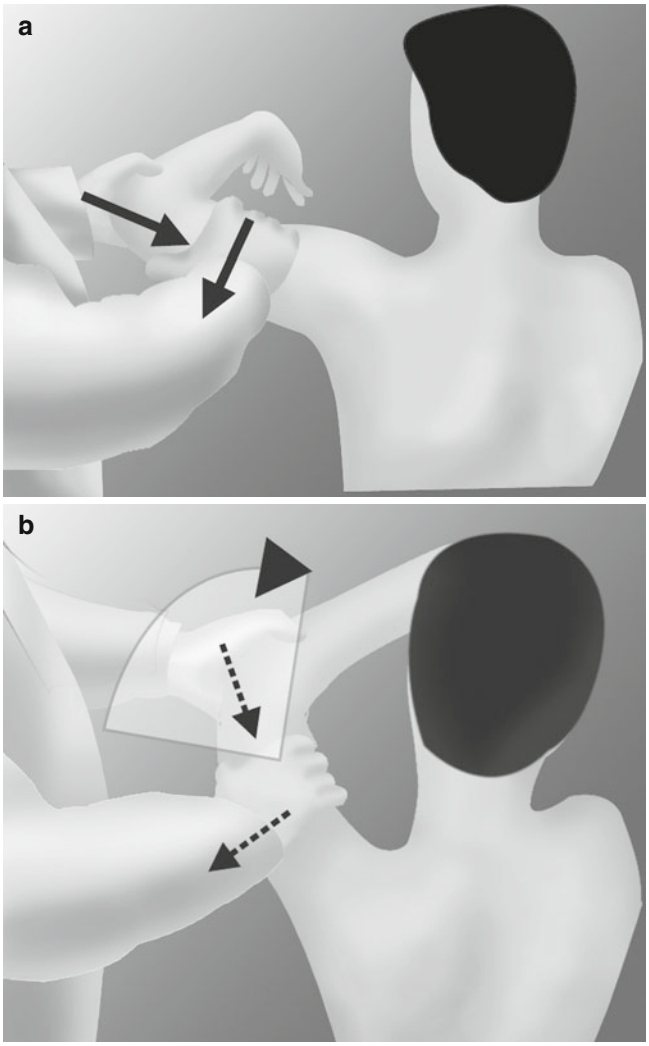

Fig. 17.5 The Kim test. (**a**) With the patient in a sitting position and the arm in 90° of abduction, the examiner holds the elbow and lateral aspect of the proximal arm and applies a strong axial load in-line with the scapula. (**b**) While maintaining an axial load, the patient's arm is elevated 45° diagonally upward and a posteroinferior force is simultaneously applied to the proximal arm. A sudden onset of posterior shoulder pain indicates a positive test result regardless of an accompanying posterior clunk of the humeral head. During the test, it is important to stabilize the patient's torso to counter support the axial load

Imaging

A standard set of shoulder radiographs should be obtained to evaluate for bony lesions. Any abnormality can be further followed with a computed tomography (CT) scan. However, the majority of posterior instability cases will have normal x-rays.

The imaging modality of choice to assess the labrum and capsule is MRA. The posterior and posteroinferior labrum should be assessed for a tear that may be non-displaced or incomplete. A loss of labral height and increased chondrolabral retroversion strongly suggests loss of chondrolabral containment and resultant posterior instability. Posteroinferior chondral erosion may also be seen. Posteroinferior labral lesions can be classified according to the classification system by Kim et al. (Table 17.1). The MR type I lesion is a separation without displacement, type II is an incomplete avulsion, and type III is a loss of contour (Fig. 17.6). Posteroinferior labral tears are very subtle and a recent preliminary study [35] showed better delineation of posteroinferior labral tears when the arm is placed in flexion, adduction, and internal rotation (FADIR). This provocative position puts the posteroinferior capsule and PIGHL on tension, thus potentially separating the tear by traction.

A redundant or patulous posterior and inferior capsule can also be assessed on MRA. However, it is important to consider the amount of injected contrast, gravity-dependent filling, and the position of the arm when assessing capsular redundancy. Dewing et al. [36] showed an increase in posteroinferior capsular cross-sectional area in patients with posterior instability versus a control group. The capsule should also be examined for any tears or a posterior humeral avulsion glenohumeral ligament (PHAGL).

Lastly, the MRA should be assessed for any other pathologies that may be concomitant or may actually be the primary

Table 17.1 Kim classification of the posterolabral lesion based on arthroscopic findings and MRA

Arthroscopic classification		MRA classification	
Type	Finding	Type	Finding
I	Incomplete stripping	I	Separation without displacement
II	Marginal crack	II	Incomplete avulsion
III	Chondrolabral erosion	III	Loss of contour
IV	Flap tear		

pathology. The utility of the MRA is not to make the diagnosis of posterior instability, but rather to confirm the diagnosis of posterior instability made by history and physical examination and gain further understanding of the patient's pathology. Alternatively, the MRA may provide additional information that may cause the surgeon to change the working diagnosis. An example would be a significant PASTA with no posterior labral pathology found on MRA in a patient with a positive jerk test. In this situation, the jerk test is a false-positive test

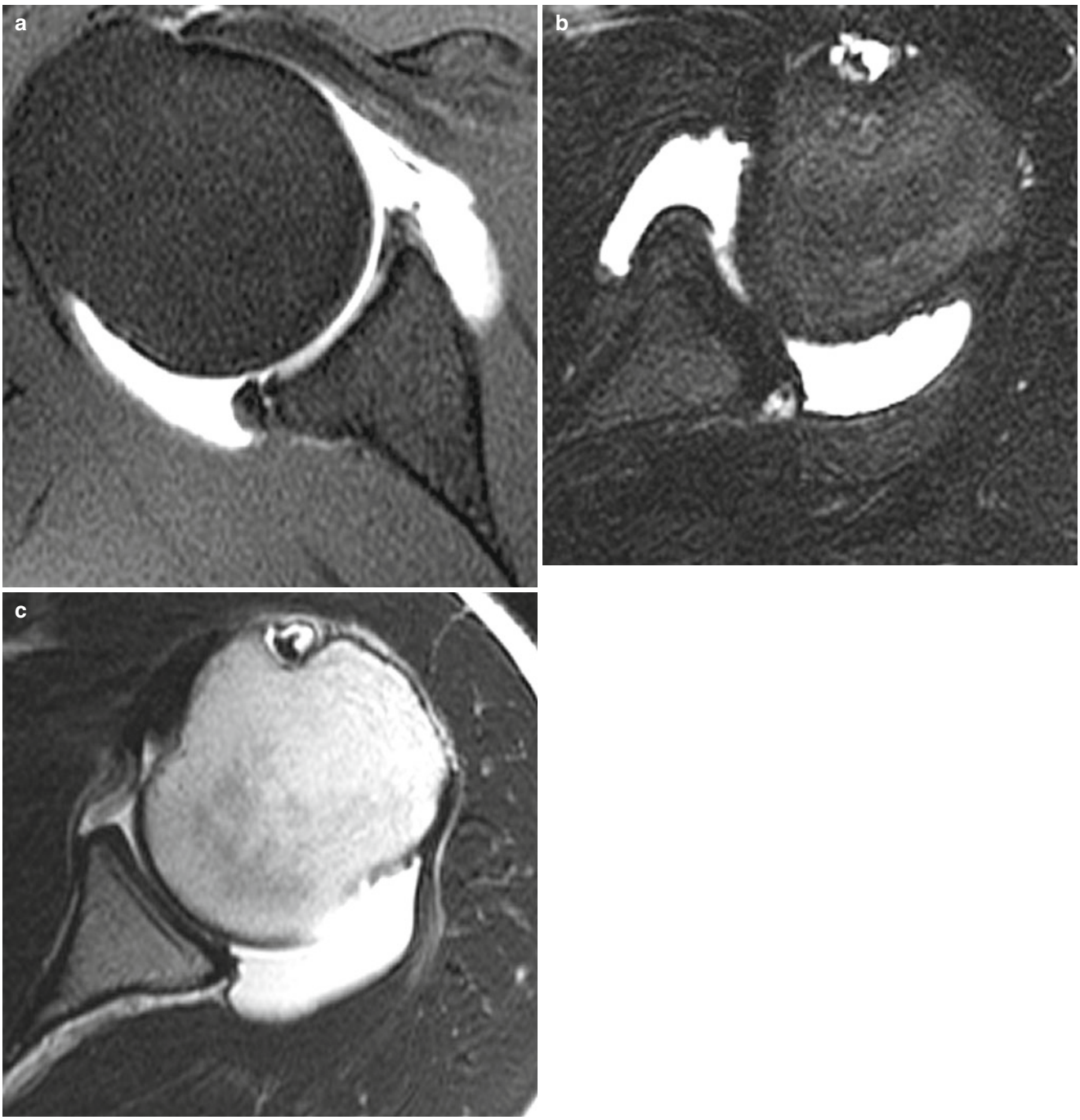

Fig. 17.6 MRA classification of posterior and inferior labral lesions. (**a**) Type I, separation without displacement. (**b**) Type II, incomplete avulsion (Kim's lesion). (**c**) Type III, loss of contour

due to the significant rotator cuff tear in a patient with hyperlaxity. Based on the author's experience (SHK) posterior instability patients have a relatively high incidence of rotator cuff tendinopathy or partial tears, and MRA underestimates this compared to MRI. This should be considered to make the proper diagnosis when assessing the patient.

Treatment: Indications and Contraindications

The general indications for surgical management of posterior instability have been reported to be continual pain and instability after a formal rehabilitation program of 6 months. Our indications have been more selective based on previous research. We try to identify which patients are likely to succeed or fail from conservative treatment, so that patients can be appropriately counseled and involved in the decision-making process. A patient with a painless jerk or Kim test has over a 90 % chance of improving with physical therapy. Whereas, a patient with a painful jerk or Kim test has approximately an 85 % chance of not improving with rehabilitation, and so we offer arthroscopic capsulolabroplasty to these patients. We also try to appropriately select patients for the appropriate surgery. In the case of posterior instability, we try to operate on patients with a labral lesion as evident on history, physical exam, and MRA so that the reason and goals of surgery are very clear. If a labral lesion is not suggestive, then we look to other diagnosis such as rotator cuff pathology with hyperlaxity. While this may often present similarly to posterior instability, the treatment is different and should focus on rehabilitation with improved neuromuscular control and treatment of rotator cuff symptoms. Patients who do not improve with conservative treatment but continue to have a painless jerk test, may benefit from surgical management of their rotator cuff pathology including a bursectomy of their painful bursitis, which may have occurred secondarily, and followed by a proper rehabilitation program.

Thus, our indications for arthroscopic capsulolabroplasty are those patients with a painful jerk or Kim test. Patients, however, may elect to try conservative treatment first with an understanding of a low chance of success. Patients with a painless jerk test are offered a formal rehabilitation program with the understanding that if proper neuromuscular control is not obtained, they can progress to have an essential labral lesion that would likely need surgery, and are thus reevaluated periodically.

Decision-Making Algorithm

Based on previous research, the jerk and Kim test are useful in predicting the success of nonoperative treatment for posterior instability and, therefore, have become hallmark in deciding between operative and nonoperative treatment. A painful jerk or Kim test is strongly suggestive of a labral lesion and upon MRA confirmation arthroscopic capsulolabroplasty is recommended. If the jerk or Kim test is painless, then a formal rehabilitation program is instituted and patients are periodically reevaluated. Persistence of symptoms with a negative jerk or Kim test should alert the clinician to seek out other pathology, whereas evolution to a positive test can occur and require arthroscopic capsulolabroplasty (Fig. 17.7).

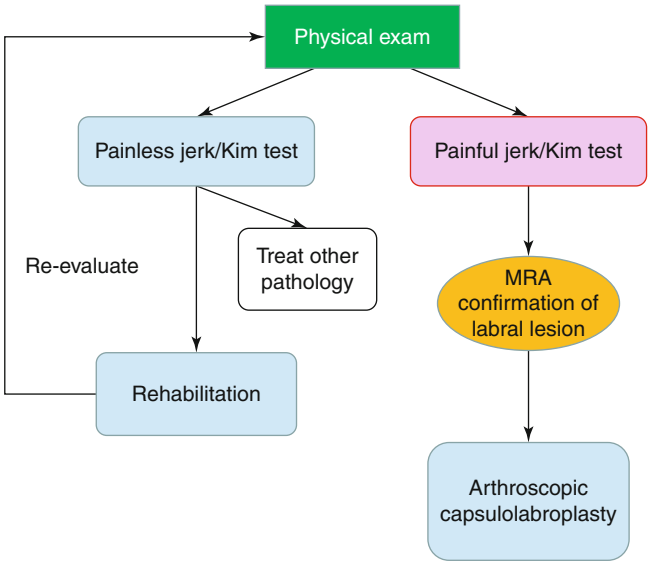

Fig. 17.7 Decision-making algorithm for posterior instability of the shoulder

Clinical Case/Example

A 40-year-old right-hand-dominant baseball player at the recreational level presented to our clinic for a second opinion regarding right shoulder pain for approximately 20 years with a 2-month aggravation. He explained that he first noticed shoulder discomfort and feeling of instability while playing baseball particularly during the follow-through phase of throwing. However, the pain was not severe; therefore he did not seek treatment. Recently, he started weight lifting and has developed severe pain particularly when doing bench press or pushing heavy objects at work. He also notes pain and less velocity when throwing a baseball. He went to a local shoulder clinic and was informed he had a SLAP tear that needed surgical repair.

On gross inspection, there is no obvious deformity or atrophy. There is tenderness to palpation along the greater tuberosity and posterior joint line, but the long head of the biceps tendon was not tender. Active range of motion was

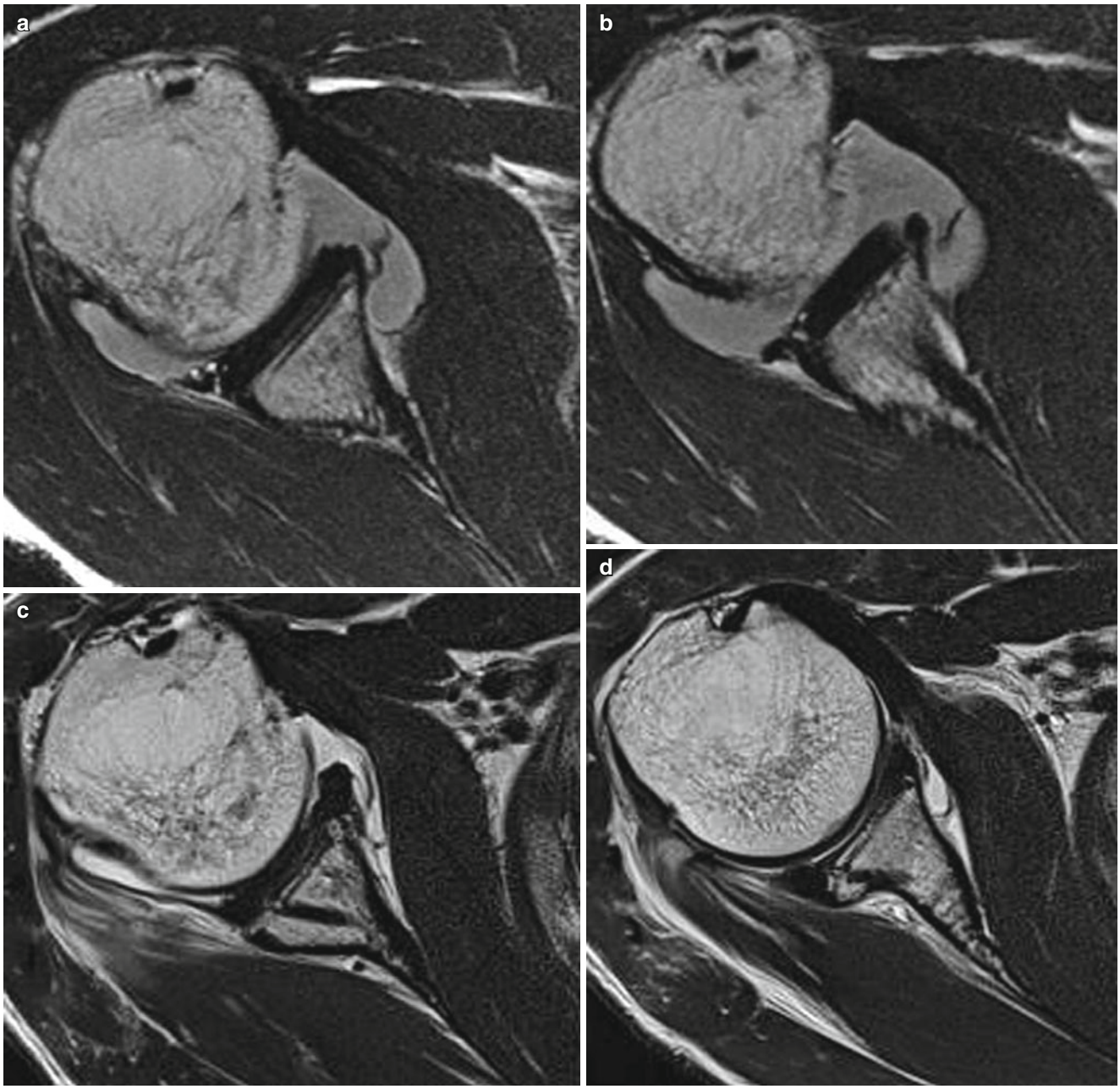

Fig. 17.8 MRA showing posterior labral tear (**a**) with loss of labral height posteroinferiorly (**b**). Postoperative MRI after chondrolabroplasty and restoration of labral height posteroinferiorly in the same patient (**c**, **d**)

forward elevation to 150°, internal rotation to T10, and external rotation at the side to 70° bilaterally. Jobe's test and impingement sign were positive on the right. In testing for a SLAP lesion, the O'Brien test was positive, but the resisted supination external rotation test and the Kim biceps load test II were negative. Sulcus sign for inferior laxity was 2+ on the right and 3+ on the left. In testing for posterior instability, the Jerk test on the right revealed pain, click, and clunk and just a painless click on the left. Kim test showed pain, click, and clunk on the right shoulder.

X-rays were unremarkable except for a small subacromial spur and moderate sclerosis at the greater tuberosity and undersurface of the acromion. On MRA, there was high signal intensity between glenoid and superior labrum. The contrast did not extend laterally and had smooth margins suggesting a sublabral recess over a SLAP tear. There was a grade I bursal-sided partial-thickness tear of the supraspinatus. Lastly, there was a large axillary recess and high signal intensity at the posterior labrum with chondrolabral retroversion (Fig. 17.8).

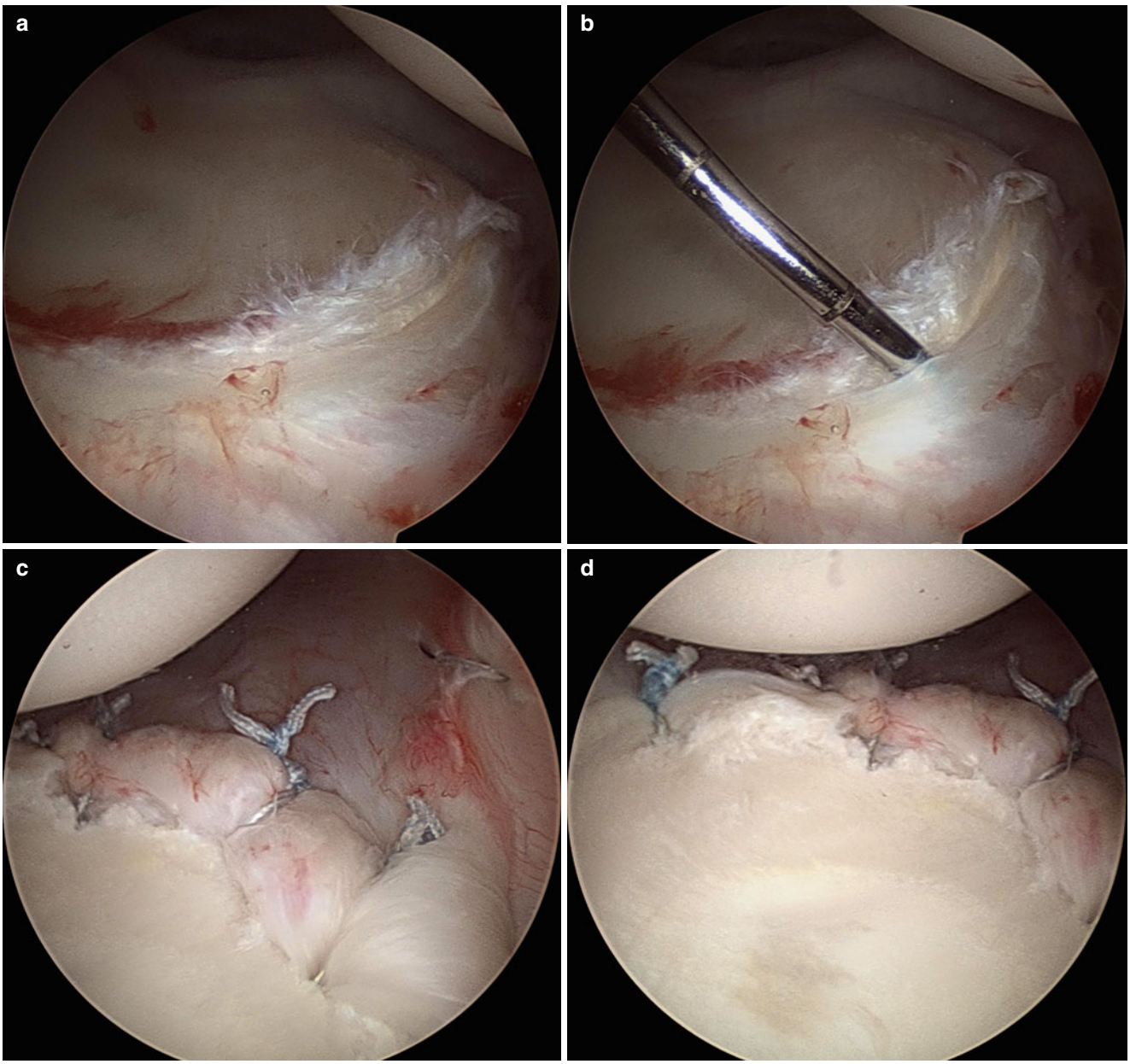

Fig. 17.9 Posteroinferior labral tear with loss of labral height viewing from the Kim posterior portal (**a**). After detaching and mobilizing the labrum (**b**), the labral height was restored, capsule tensioned, and the posterior portal was closed (**c**, **d**) viewing from the transcuff portal

Based on these findings, our assessment of this patient was posterior instability with a low-grade bursal-sided rotator cuff tear with impingement. On arthroscopy, a normal variant of the superior labrum was confirmed. There was a posteroinferior labral tear extending from 6 to 9 o'clock with loss of labral height and a patulous posterior and inferior capsule. Capsulolabroplasty was performed along with a subacromial decompression and debridement of the bursal-sided rotator cuff tear (Fig. 17.9).

The patient underwent a formal rehabilitation program postoperatively and was cleared to play sports at 6 months. On his last follow-up visit 8 months postoperatively, he reported complete and pain-free return to his preinjury activities with no functional limitations.

Arthroscopic Treatment: Surgical Technique

Patient Positioning

Both the beach chair and lateral decubitus position can be used for the procedure; however, the lateral decubitus

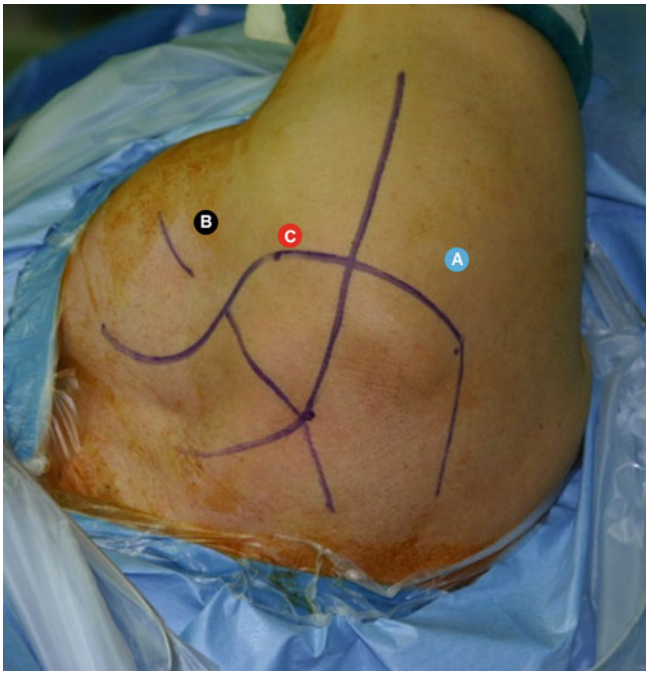

Fig. 17.10 The triple instability portal shown in a right shoulder: Kim posterior portal (*A*), anterior mid–glenoid portal (*B*), and transcuff superior portal (*C*)

position provides superior visualization of the inferior aspect of the glenoid. Our preference is to place the patient in a lazy lateral decubitus position so that the glenoid is parallel to the floor. After administration of regional anesthesia in the form of an interscalene block, the patient is placed in a semisupine position. The affected arm is placed in approximately 3–5 kg of traction with the arm abducted approximately 60° and flexed approximately 20°.

Portals

The conventional portals used for anterior instability (posterior, anterosuperior, and mid-glenoid portals) are often used for posterior instability with the addition of an accessory posterior portal. However, viewing the posterior labrum from this portal is suboptimal and therefore one of the anterior portals becomes the main viewing portal. Additionally, the accessory posterior portal becomes essential because of the improved trajectory for instrumentation and access to the posterior and inferior labrum. This 4-portal setup also creates two tears in the posterior capsule and two tears in the rotator interval. This appears to be counterproductive since the posterior capsule is one of the main structures we are trying to repair, and the rotator interval is believed to contribute to posterior and inferior stability.

Our preferred method is to use the triple instability portal (Fig. 17.10). This setup uses 3 portals: a modified posterior portal (Kim posterior portal), a transcuff superior portal, and a mid-glenoid anterior portal. The Kim posterior portal is established approximately 2 cm lateral from the inferior edge of the posterolateral acromial corner. This portal is approximately 3 cm more lateral from the standard posterior portal and, thus, clears the glenoid rim and provides a "downward" or stadium view of the glenoid face. A mid-glenoid portal is made localizing with a spinal needle just lateral to the coracoid process and through the rotator interval just superior to the leading edge of the subscapularis tendon. A transcuff superior portal is placed just posterior to the anterolateral corner of the acromion. This portal is placed through the supraspinatus muscle just medial to the musculotendinous junction thereby avoids a cuff tear from portal placement.

There are numerous advantages of the triple instability portal setup. First, all the portals are high riding thus providing optimal visualization with a "stadium view" of the glenoid. Second, all the portals allow for vertical access to the glenoid rim, and anchor placement anywhere on the glenoid rim is possible. Third, there is less iatrogenic injury to important structures compared to the 4-portal setup with just one portal through the rotator interval and only one small 5.25 mm portal through the posterior capsule. Lastly, this setup is flexible and is the same setup we use for any directional instability or glenoid work.

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

A standard glenohumeral exam can be carried out from the Kim posterior portal. It should be noted that there is relatively a high incidence of concomitant partial articular-sided rotator cuff tears. The rotator interval should also be carefully inspected; however, in our experience we have rarely found any significant pathology.

The posterior and inferior labrum should be inspected for any labral tears, which can be very subtle. Loss of chondrolabral containment should be recognized. Signs would be a loss of posterior labral height or capsular redundancy. Infrequently, frank capsular tears or a PHAGL can occur. There also may be chondromalacia at the posterior rim ranging from subtle softening to erosion.

Kim et al. [14] classified posteroinferior labral tears according to arthroscopic findings (Fig. 17.11). Type I is an incomplete superficial labral tear. Type II is known as the Kim's lesion and is concealed with marginally cracking with a deep avulsion tear. Type III involves chondrolabral erosion. Type IV has a flap tear. While type II and III may appear to be intact, they are associated with chondrolabral retroversion.

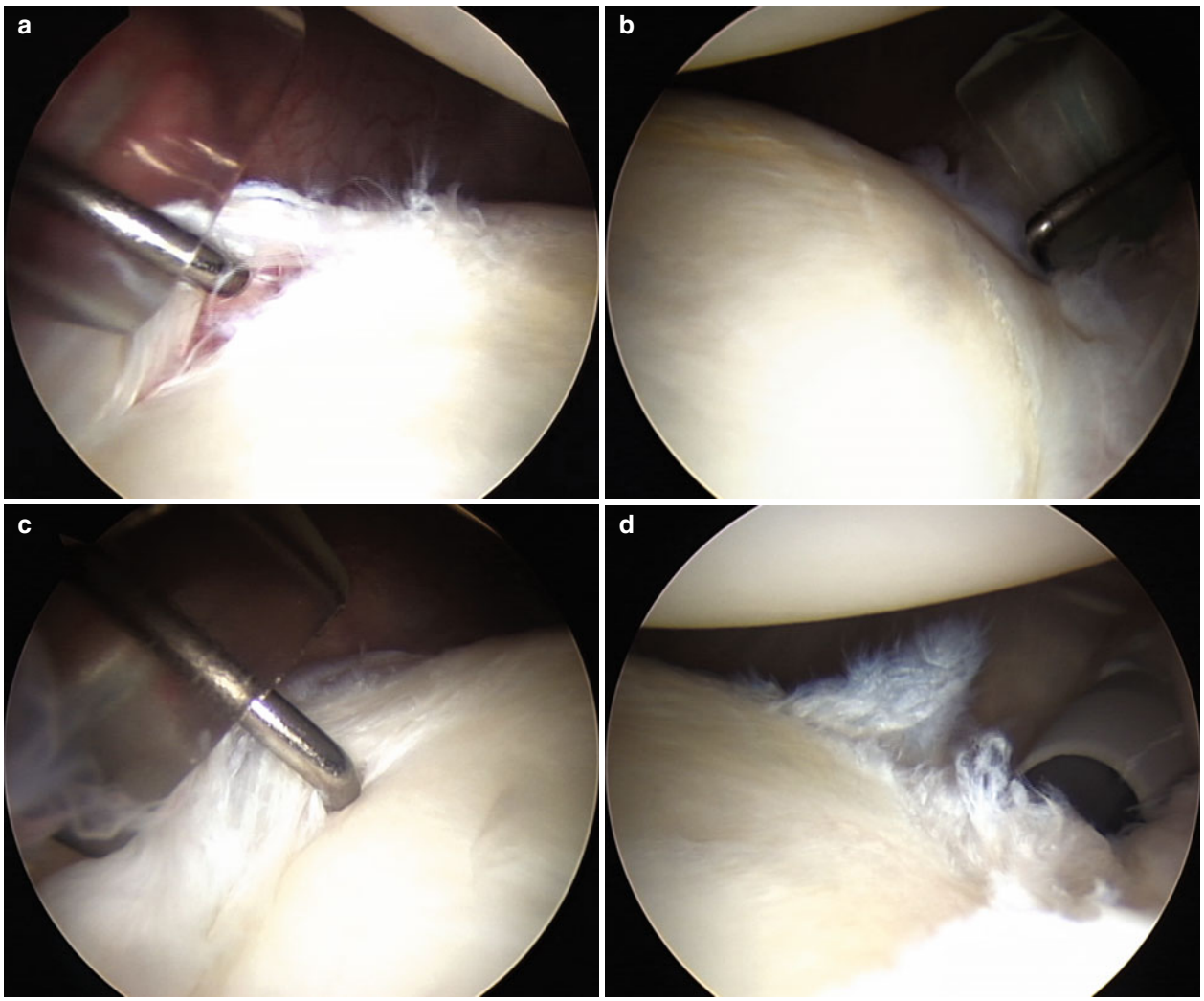

Fig. 17.11 Arthroscopic classification of posterior and inferior labral lesions. (**a**) Type I, incomplete detachment. (**b**) Type II, marginal crack with discreet deep tear (Kim's lesion). (**c**) Type III, chondrolabral erosion. (**d**) Type IV, flap tear

Step-by-Step Procedure (Box 17.1)

Box 17.1: Tips and Tricks

The triple instability portal setup allows for an excellent stadium view of the entire glenoid and vertical access to the entire glenoid rim and minimizes iatrogenic injury compared to the traditional 4-portal setup.

The goal for arthroscopic capsulolabroplasty is to reestablish chondrolabral containment by restoring the labral height and re-tensioning the capsulolabral complex. The general steps are to:

1. Recognize the pathology.
   - Common posteroinferior labral tears are deep, discreet avulsions (Kim lesion) that are easily missed.
2. Completely mobilize the torn labrum.
   - The labral height can be rebuilt and the capsule can be appropriately tensioned and shifted.
3. Prepare the glenoid neck and rim for optimal healing.
   - Rasp the glenoid neck to provide for a bleeding bone bed.
   - Remove the cartilage at the rim for proper anchor fixation and so the labrum can heal at a heightened position to rebuild concavity.
   - Remove interposing tissue and debris that can impede healing.
4. Re-tension the capsule and restore the labral height.
   - Inferior laxity requires a slight superior shift.
   - Tensioning is subjective and appropriate moderation is needed for stability without stiffness.

- Vertically orient anchors so that the labrum heals on top of the glenoid rim to rebuild the labral height.
- Space anchors ~1 cm apart for adequate fixation.
- Do not rely on knot tying to reduce the tissue appropriately, but use the tissue grasper and knot tying techniques to ensure proper reduction and secure fixation.

After diagnostic glenohumeral arthroscopy from the posterior Kim portal, the transcuff and mid-glenoid portals are established. The mid-glenoid portal utilizes a large enough cannula (e.g., 8.25 mm, Twist-In Cannula, Arthrex Inc., Naples, FL, USA) to accommodate a suture hook instrument whereas a small cannula in the transcuff portal is preferred. To avoid cuff damage when making the transcuff portal, care must be made to direct the trocar just medial to the musculotendinous junction, "hook" the edge of the cuff, and then direct laterally before puncturing for proper orientation. The arthroscope is then placed in the transcuff portal so that the posterior labrum can be probed from the posterior Kim portal. A small smooth cannula (e.g., 5.25 mm, Crystal Cannula Smooth, Arthrex Inc., Naples, FL, USA) is placed in the posterior portal at this time.

The posterior labrum should be probed for softening and marginal cracking. If this is present, the tear should be completed with the probe. An arthroscopic liberator knife is then used to completely mobilize the torn labrum from the glenoid neck. Next, the glenoid surface should be prepared to optimize healing. A rasp is used to slight decorticate the glenoid neck and provide for a bleeding bone bed for the labrum to heal onto. Additionally, a ring curette is used to remove cartilage at the glenoid rim to ensure secure anchor fixation at the glenoid rim and a solid healing area so that the reattached labrum heals onto the rim to rebuild the labral height. When working on the posteroinferior aspect of the glenoid, it is easier to view from the transcuff portal and liberate or rasp from the mid-glenoid portal. And when working on the posterior aspect of the glenoid, it is easier to view from the mid-glenoid portal and liberate or rasp from the transcuff portal. The assistant should stabilize the glenoid by providing countertraction on the scapula when working on the glenoid particularly with rasping and when placing the anchor. Debris should be removed with an arthroscopic shaver so that this does not impede the healing process and tissue forceps should be used to assess if the labrum was adequately mobilized.

Next, while viewing from the transcuff portal a double-loaded anchor is introduced from the posterior Kim portal at the 5:45 position in the left shoulder or 6:15 position in the right shoulder. An assistant should provide lateral and anterior traction on the arm until the anchor guide is placed on the glenoid rim. The assistant should then release traction and the surgeon should torque the humeral head with the anchor guide so that optimal anchor position into the glenoid is achieved. The small cannula is then removed from the posterior Kim portal so that a suture hook (e.g., Spectrum Suture Hook, Linvatec Corp., Largo, FL, USA) can be introduced from the posterior portal. A suture grasper is introduced from the mid-glenoid portal and can be used to reduce the mobilized labrum to facilitate puncture with the suture hook and ensure that an appropriate capsular shift will occur. How much capsular tissue to incorporate is based on the surgeon's experience, but it is important not to take too much tissue and create a stiff shoulder. Each paired high strength suture from the double-loaded anchor should be passed through the labral tissue approximately a 0.5–1 cm from each other. All suture limbs are retrieved from the mid-glenoid portal. The small cannula is then repositioned in the posterior portal where the suture limbs are retrieved and then tied using the surgeon's preferred knot and cut. We use the SMC sliding locking knot or the Madi loop non-sliding locking knot. It is important to not assume that the labral tissue will adequately be reduced by the sliding knot. Optimal reduction of the labral tissue and perpendicular positioning of the knot may require assistance with the suture grasper or techniques using the knot pusher. If the knot is tied in the vector of the cannula and knot pusher, the knot will be tied oblique to the glenoid rim and may result in insecure fixation over time. The knot should be positioned and tied on the tissue side to avoid chondromalacia from abrasion with the high tensile suture knot. Anchor placement and labral tissue fixation are repeated using a single-loaded anchor approximately 1 cm apart until the entire torn labrum is reattached.

The anterior capsule should be assessed for redundancy or labral tears. If the labral tear extends anteriorly, it is easier to place the anteroinferior anchor (6:15 position for left shoulder) and shuttle sutures through the anterior labrum before tying the posteroinferior sutures, since tying would effectively close the volume making visualization and instrumentation potentially cumbersome. Anterior anchors are introduced from the mid-glenoid portal and the respective sutures are tied after the posterior labrum has been repaired. In patients with associated severe anterior capsular redundancy, we perform an anterior capsular plication with a suture anchor. While this can also be done with a free suture, the author (SHK) has experienced less capsular tearing utilizing an anchor when tying. The anterior capsule is roughened with a rasp to stimulate healing and a plication and shift is done using a nip and tuck technique.

After capsular repair, the overall capsular ligamentous balance can be observed from the superior transcuff portal by assessing the position of the humeral head relative to the

Table 17.2 Surgical algorithm in arthroscopic capsulolabroplasty

Finding				
Jerk/Kim test	Sulcus	Anterior translation	Predominant instability	Procedure
+	1+	–	Posterior	Labroplasty and capsular shift (posterior)
+	2+	+	Posteroinferior	Labroplasty and capsular shift (posterior and inferior)
+	3+	+	Inferior	Labroplasty, capsular shift (posterior, inferior, and anterior), and rotator interval closure

centrum of the glenoid. Closure of the rotator interval is an option; however, in the author's experience (SHK) this is rarely needed after an appropriate anterior capsular plication. Our general algorithm for rotator interval closure is described in Table 17.2.

Posterior portal closure is performed by viewing from the transcuff portal. A number 2 high tensile suture is loaded onto a penetrating suture retriever (Penetrator Suture Retriever, Arthrex Inc., Naples, FL, USA) and introduced from the posterior portal. The smooth cannula is pulled just posterior to the posterior capsule to avoid entrapment of soft tissue and the capsule is then penetrated on one side of the portal hole. A suture limb is retrieved from the mid-glenoid portal and the penetrating suture retriever is used to penetrate the posterior capsule on the other side of the portal hole and retrieve the suture limb docked at the mid-glenoid portal. Knot is tied from the Kim posterior portal in blind fashion and cut.

Postoperative Care

The arm is supported in neutral rotation with a sling and pillow spacer for 3–6 weeks. During this time, active elbow and wrist motion is encouraged. Gentle range-of-motion exercises are permissible after 3–6 weeks and progressively increased with proprioceptive exercises and strengthening. Return to sports is typically allowed at 6 months.

Literature Review

A review of the literature shows arthroscopic surgery is generally successful in treating posterior instability. But to further interpret and make clinical conclusions from the clinical trials are difficult because: (1) surgical methods differ (mainly involving a capsular plication or repair with or without suture anchors); (2) distinction is not always clear between posterior instability, posteroinferior instability, and multidirectional instability; (3) there are no randomized clinical control trials; and (4) most studies involve small cohorts. Nonetheless, arthroscopic capsulolabral repair with suture anchors has had overall good results.

In 1998, Wolf and Eakin [12] reported on their results of arthroscopic capsular plication with or without suture anchors depending on the severity of the labral tear in 14 patients with posterior instability. Twelve patients had excellent results and two had fair results. There was a 90 % return to preinjury level in recreational or competitive athletics. There was one recurrence after a traumatic episode which resolved after a second arthroscopic surgery with a capsular reconstruction. Kim et al. [9] in 2003 reported on 27 patients with arthroscopic capsulolabroplasty with suture anchors for traumatic unidirectional recurrent posterior instability. All patients were involved in sports and were able to return to their prior sports activity with little or no limitation except one person who had recurrent instability. There were 21 excellent UCLA scores, 5 good scores, and 1 fair score. In 2004, Kim et al. [14] reported their results of arthroscopic capsulolabroplasty with suture anchors for posteroinferior multidirectional instability in 31 patients. Thirty-one patients had stable shoulders after surgery, and one had recurrent instability. All patients had improved function, pain, and shoulder scores with 21 excellent Rowe scores, 9 good scores, and 1 fair score. In 2005, Provencher et al. [37] reported their outcomes for arthroscopic stabilization with or without suture anchors in 33 patients with posterior instability. Seven were considered failures for recurrent instability (4) and pain (3). Mean shoulder scores were: American Shoulder and Elbow Surgeons Rating Scale = 94.6, Subjective Patient Shoulder Evaluation = 20.0, Western Ontario Shoulder Instability Index = 389.4 (81.5 % of normal), and Single Assessment Numeric Evaluation = 87.5. Patients with voluntary instability or prior shoulder surgery had significantly worse outcomes. In 2008, Savoie et al. [38] reported on their treatment of arthroscopic capsulolabral repair with suture anchors, arthroscopic capsulorrhaphy, and mini-open and arthroscopic tendon/capsule plication for posterior instability in 136 shoulders. There were two cases of recurrent instability: one in the suture anchor group ($n = 53$) and one in the Caspari technique ($n = 31$). Also, in 2008, Radkowski et al. [39] compared throwers and non-throwers after arthroscopic capsulolabral repair or plication with or without suture anchors in 107 shoulders with unidirectional posterior instability. There were a total of 11 failures (11 % in the thrower group; 10 % in the non-thrower group) as determined by a score of 5 or less out of 10 on the subjective instability rating. There were no differences in stability, range of motion, strength, pain, function, and ASES scores

between the two groups; however, throwing athletes were less likely to return to their preinjury level of sports compared to non-throwing athletes, 55 % compared to 71 %. In 2012, Lenart et al. [40] reported two out of 34 cases of recurrent instability after arthroscopic capsulolabral repair with or without suture anchors for posterior instability. Mean ASES and Simple Shoulder Test scores improved and mean VAS (Visual Analog Score) pain score decreased from 3.5 to 0.8.

In looking at the studies collectively, there was a 94 % overall success rate (14 failures due to pain or recurrent instability out of 247 cases) and no complications were reported. Some studies included other arthroscopic repair techniques in their results in addition to arthroscopic capsulolabral repair with suture anchors. While it was not possible to extract just these results, it is reasonable to conclude that arthroscopic capsulolabral repair with suture anchors is an effective and safe surgery for recurrent posterior instability.

Summary

Posterior instability of the shoulder has historically been poorly diagnosed and mistreated. Accurate diagnosis requires understanding of the pathogenesis and a careful history and physical examination. MRA is used to confirm the diagnosis, better understand the extent of injury, and examine for concomitant pathology. Patients with a painless jerk and Kim test are usually successfully treated with physical therapy and rehabilitation, whereas those with a painful jerk and Kim test usually respond poorly to conservative treatment. Arthroscopic capsulolabroplasty is offered to the latter patients suggestive of an essential posterior labral lesion. With appropriate patient selection and a thorough arthroscopic examination, surgical results are encouraging and favorable.

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Multidirectional Instability of the Shoulder

18

Anthony A. Romeo and Benjamin Bruce

Epidemiology

Multidirectional instability (MDI) represents a wide spectrum of disease, but is defined as symptomatic, involuntary, and uncontrollable subluxation or dislocation of the glenohumeral joint in two or more directions [1]. Instability should be differentiated from laxity, in which no symptoms are present. Due to its inconsistent definition in the literature, it is difficult to reliably determine its actual incidence. MDI likely represents a less common clinical entity than anterior traumatic instability, representing 7–10 % of all instability cases [2, 3].

Pathophysiology

The essential lesion in MDI is an enlarged, lax glenohumeral joint capsule [4]. This "patulous" capsule fails to provide adequate stabilization to the glenohumeral joint [5, 6]. A host of other pathologic lesions may be found in conjunction with a patulous capsule. Both anterior and posterior labral lesions are commonly identified [5–7]. The biceps tendon has been found to be more anterior compared with normal controls [8]. Some patients demonstrate Hill-Sachs lesions or articular-sided partial-thickness rotator cuff tears. Some authors suggest the rotator interval is widened in MDI, although this finding remains controversial.

The etiology of multidirectional instability is likely due to a combination of factors including anatomic, biologic, and neuromuscular pathologies. Repetitive episodes of microtrauma to the capsule and other stabilizers may be a significant contributor to the progression of this clinical entity [9, 10].

Clinical as well as histopathologic data have suggested that patients with MDI may have an underlying connective tissue disorder that predisposes them towards instability [8]. Biologically, patients with MDI demonstrate an increase in collagen cross-linking, collagen fiber diameter, cysteine content, as well as elastin content when compared to normal shoulder. These likely represent adaptive changes [4, 11]. Although patients often have bilateral laxity, most are only symptomatic on one side. This suggests that factors beyond biology play a role in the development of instability.

Anatomically, static stabilizers of the joint include the glenoid concavity, the labrum, as well as the glenohumeral ligaments. The glenolabral complex is relatively shallow and provides little inherent stability. Nonetheless, some studies have demonstrated that patients with MDI demonstrate shallower glenoid cavities relative to normal controls [11]. The glenohumeral ligaments provide the majority of their effect at extremes of motion. Therefore, the dynamic stabilizers of the shoulder, which include the rotator cuff, biceps, deltoid, and scapula rotators, are critical for midrange stability. They increase the contact pressure of the glenohumeral joint to provide concavity compression [12].

Impaired coordination of the dynamic stabilizers of the shoulder girdle has been suggested as a possible contributor to MDI. Electromyography performed in patients with MDI demonstrated altered activation of the anterior and posterior deltoid muscles compared to normal subjects [13, 14]. Scapulothoracic dysfunction is also considered a contributor to MDI. Failure of the scapula to rotate through shoulder range of motion may force abnormal translation of the humeral head and contribute to the progression of instability [15].

Diminished proprioception has been suggested as a contributor to MDI. Barden et al. showed that patients with MDI demonstrated significantly higher error rates with hand positioning in space compared with normal controls. They

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suggest that the failure of proprioceptive feedback may play a role in the humeral head instability [15].

History

MDI can be challenging to diagnose as it represents a wide spectrum of pathology, and the reported symptoms may be vague. MDI is often found in young adults who present with primary complaints of pain, rather than frank instability [16]. Nonetheless, over 25 % of patients with MDI present with radiographic evidence of previous dislocation. Patients often complain of constant diffuse, background pain that is exacerbated with lifting or sleeping. Many complain of "loose" shoulders that may have associated clicking or snapping. Athletes may complain of decreased strength or performance. The threshold for inducing symptoms may be lower than that associated with traumatic dislocations. Consequently, MDI patients may be more limited in their daily activities. Many patients report neurological complaints such as numbness or tingling. Patients with a history of repetitive overhead activity may be predisposed to MDI through microtrauma.

If frank instability is suggested by the history, it is important to delineate the frequency and mechanisms by which instability occurs. Arm position at time of dislocation may suggest the direction of instability. Many patients with MDI cite a history of low-demand activities inciting dislocations, which require minimal effort to reduce. Some authors have suggested that patients who dislocate during sleep represent a subset of difficult-to-treat "decompensated shoulders" [9].

A detailed history may suggest the direction of instability. Pain and numbness while carrying heavy objects suggests inferior instability. Bench presses or discomfort with pushing may indicate posterior pathology. Pain with the arm in abduction and external rotation, such as a throwing motion, implies anterior pathology [10]. This information is critical in guiding therapy or operative intervention.

Patients should be questioned about previous joint sprains or patellar instability. As noted above, patients with MDI have been noted to demonstrate connective tissue disorders, and workup for these diseases may be warranted.

All patients with a history of voluntary dislocation should be evaluated closely for underlying psychiatric disorders. Patients with ongoing mental health issues respond poorly to operative and nonoperative management until those pathologies have been resolved. A subset of voluntary dislocaters without underlying emotional problems, however, may respond to operative treatment.

It is critical to elicit previous history of physical therapy or operative intervention. MDI is often misdiagnosed as unilateral instability, impingement, brachial plexitis, cervical neck pathology, or thoracic outlet syndrome. A history of failed previous treatments may suggest MDI.

Clinical Examination

A careful clinical examination is essential in differentiating MDI from other causes of shoulder pain. Furthermore, identification of incompetent structures on exam will elucidate appropriate, directed treatment.

The key diagnostic finding for MDI is the reproduction of symptoms with provocative maneuvers. The load-and-shift test assesses the degree of humeral instability. With the patient supine, the clinician stabilizes the elbow while attempting to translate the humeral head anteriorly or posteriorly. Subluxation of the head over the glenoid rim can be palpated, and the magnitude and direction of instability assessed. While patients may have multi-planar laxity, they may be primarily symptomatic in just one direction. Patients with painful posterior jerk test have a higher rate of failure with nonoperative management [17]. Internal rotation strength may be decreased by up to 30 % in patients with MDI [17]. An abnormal sulcus test, which is inferior traction on the adducted arm, suggests laxity. This same test with the arm in 30° of external rotation is specific for the rotator interval (Fig. 18.1). Traction may occasionally provoke neurologic symptoms. An isolated sulcus sign in the absence of symptoms does not suggest MDI. The Gagey test evaluates the competency of the inferior capsule. The test is considered positive when the shoulder can be passively abducted more than 105° with concomitant stabilization of the scapula.

Visual inspection of the shoulder may reveal a protracted scapula or muscle atrophy. The shoulder may appear squared secondary to inferior humeral head subluxation. Active range of motion may reveal scapular dyskinesia. Often patients with MDI will have altered scapular mechanics including medial scapular winging, inferior tip rotation, and poor scapular protraction against the chest wall [18]. The pectoralis minor muscle may become contracted secondary to prolonged scapular malposition, and the patient may exhibit point tenderness over

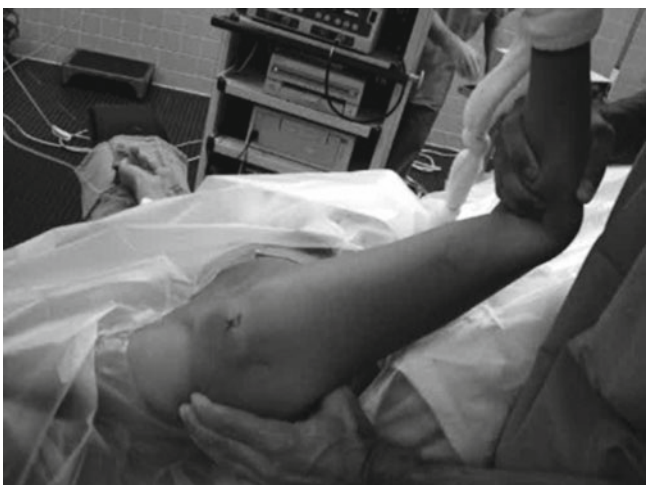

Fig. 18.1 Sulcus sign with arm in 30° of external rotation indicating rotator interval laxity

the coracoid. MDI patients may exhibit pain with manual strength testing as they can develop rotator cuff tendonitis.

In addition to the standard bilateral shoulder strength, range of motion, and instability tests, the exam must include a thorough cervical and neurologic exam. All patients should be evaluated for hyperlaxity including patellar laxity, elbow hyperextension, thumb-for-forearm test, or genu recurvatum. Clinical suspicion for a connective tissue disorder such as Marfan's or Ehlers-Danlos merits further workup as outcomes for surgical outcome are very poor in this patient population [19].

Imaging

Although patients with MDI may have normal radiographs, a complete instability series of x-rays should always be obtained at initial visit as patients can have concomitant shoulder pathology or a history of traumatic dislocation, which can alter the treatment plan. This series consists of glenohumeral AP views in neutral, internal, and external rotation, axillary lateral, West Point axillary, and Stryker notch view. These views allow for evaluation of humeral head displacement on the glenoid, Hill-Sachs lesions, bony Bankart pathology, fractures of the lesser and greater tuberosities, glenoid hypoplasia, or rim defects and fractures. In addition, the AP radiograph can reveal inferior subluxation of the humerus on the glenoid. In patients with suspected glenoid hypoplasia, bone loss, and retroversion, a CT scan with 3 mm cuts with and without humeral head subtraction should be ordered. In addition to radiographs for patients with suspected MDI, the authors' preferred choice of advanced imaging is an MRI and MR arthrogram to further delineate soft tissue pathology involving the rotator interval, biceps, capsulolabral structures, and the rotator cuff (Fig. 18.2). Some authors have suggested a widened rotator interval is present in most cases of MDI. MRI arthrography comparing normal and instability patients, however, failed to demonstrate a difference [18].

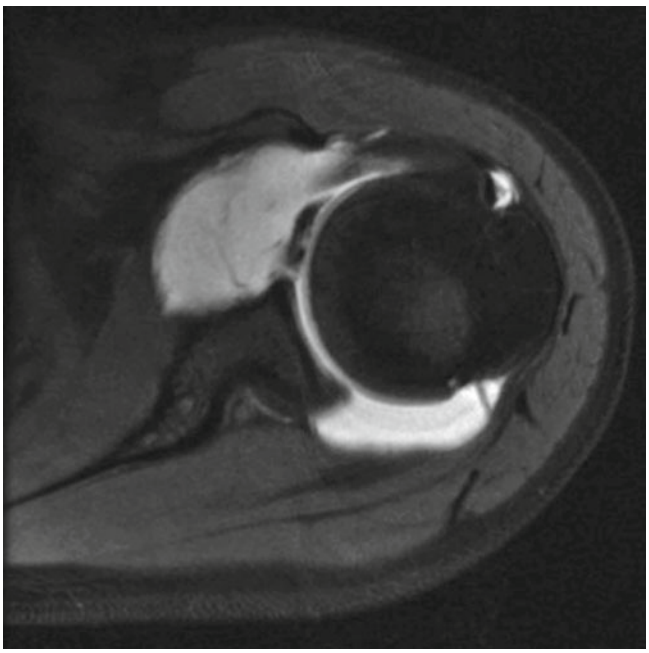

Fig. 18.2 Axial cut of MDI patient demonstrating anterior labral pathology and patulous pouch

Treatment: Indications and Contraindications

Surgical stabilization of patients with MDI should be considered in patients with symptomatic, involuntary shoulder instability that have failed a 6-month trial of physical therapy directed at scapular stabilization, rotator cuff, and deltoid strengthening exercises. Most patients with true MDI will have decreased pain and improved stability with a rigorous shoulder strengthening and rehabilitation program. It is crucial to evaluate progress with a focused scapular retraining program prior to embarking on surgical intervention. Young, athletic patients with instability or generalized ligamentous laxity due to a traumatic event, however, are less likely to respond to therapy compared to patients whose pathology stems from repetitive microtrauma [8]. Although patients with a traumatic etiology to shoulder instability are likely to have concomitant glenohumeral pathology such as a Bankart lesion, this does not preclude a trial of conservative treatment.

An increased awareness and understanding of clinical symptoms and exam findings in patients with voluntary versus involuntary MDI has led to more appropriate patient selection criteria for surgical stabilization. In particular, patients with a history of voluntary shoulder dislocation and psychiatric issues should not be treated surgically in lieu of higher failure rates [4].

Decision-Making Algorithm

Improvements in arthroscopic techniques have led to a shift from open to arthroscopic stabilization, which allows for an outpatient procedure with potentially decreased risk of complications such as subscapularis rupture or postoperative subscapularis insufficiency. With improvements in current implants and arthroscopic techniques, a successful stabilization procedure for patients with MDI can be attained.

The actual repair technique and implants used will be dictated by findings from clinical exam and diagnostic arthroscopy. Treatment of lesions that do not correspond to any physical exam findings may result in overtightened shoulders. Failure to recognize all directions of instability on clinical exam may lead to persistent symptoms postoperatively.

The decision to perform a rotator interval closure is controversial. Successful clinical outcomes can be achieved with and without rotator interval closure [5–7]. Patients who demonstrate a sulcus sign with the arm in external rotation should be considered for rotator interval closure.

Clinical Case/Example

C.V., an 18-year-old right-hand-dominant female, had a 5-year history of bilateral shoulder instability. The initial dislocations were atraumatic but painful in nature. The patient has described multiple instability episodes without the need for closed reduction in the emergency room.

On exam, the patient has a positive sulcus sign and exhibits a positive apprehension sign when the right arm is positioned in abduction and external rotation. Her shoulder could be moved through a full range of motion with no neurovascular deficits. The patient did have evidence of hypermobility bilaterally with hyperextension of the elbows and metacarpophalangeal joints of the fingers (Fig. 18.3).

Radiographic examination revealed a small Hill-Sachs lesion on Stryker notch and AP views of the glenohumeral joint. The axillary lateral radiograph demonstrated a minimal anterior glenoid bone loss. MRI revealed anterior and posterior labral lesions.

After evaluation in clinic, the patient was placed in a therapy program with focus on scapular, cuff, and deltoid strengthening. After approximately 6 months of conservative treatment, the patient continued to complain of shoulder instability and noted that these symptoms occurred with greater frequency. Given the history of recurrent instability, physical exam findings consistent with MDI, and failure of conservative treatment, the patient opted to proceed with arthroscopic stabilization.

Arthroscopic Treatment: Surgical Technique (Video 18.1)

Patient Positioning

We prefer interscalene block for patients undergoing surgical treatment for MDI as it enhances postoperative pain control.

Careful examination of the patient under anesthesia prior to surgical skin preparation will confirm the diagnosis as well as guide treatment. A load-and-shift test as well as sulcus sign should be performed. The magnitude of instability should be evaluated with the patient's preoperative symptomatology kept in mind. A patient with preoperative symptoms and a sulcus sign that remains 2+ or greater in external rotation is pathognomic for MDI.

Both the beach chair and lateral positions can be used for the arthroscopic treatment of MDI. However, the lateral decubitus position has been shown to offer improved visualization of the posterior and inferior aspects of the shoulder, where the significant pathologic lesions may be located [20].

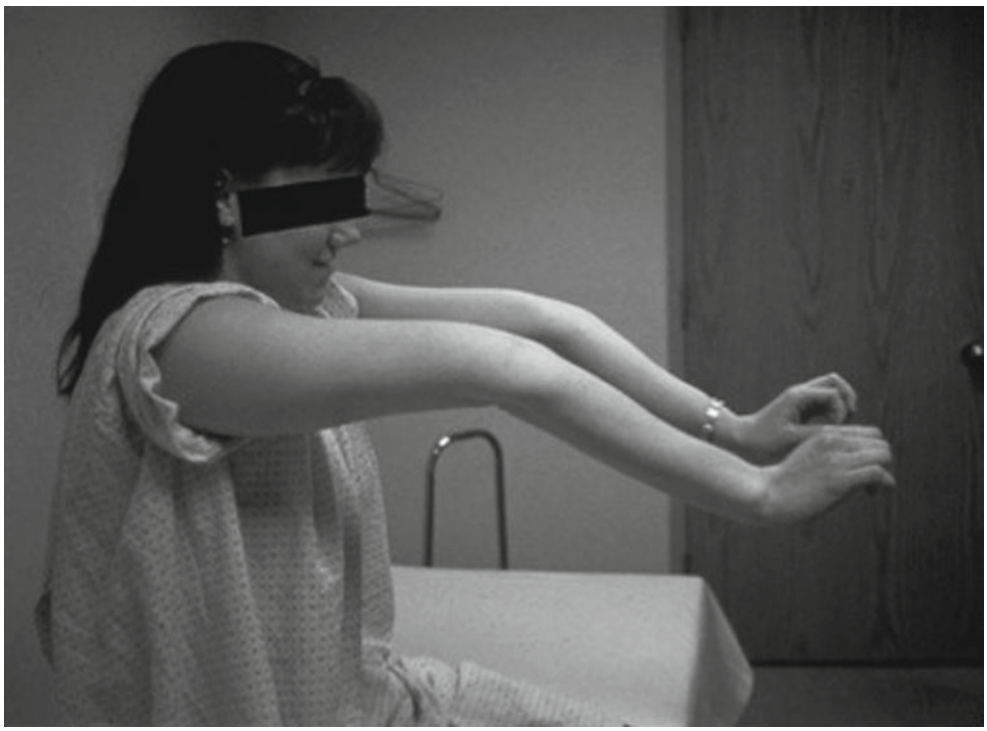

Fig. 18.3 Clinical evidence of hypermobility including elbow hyperextension

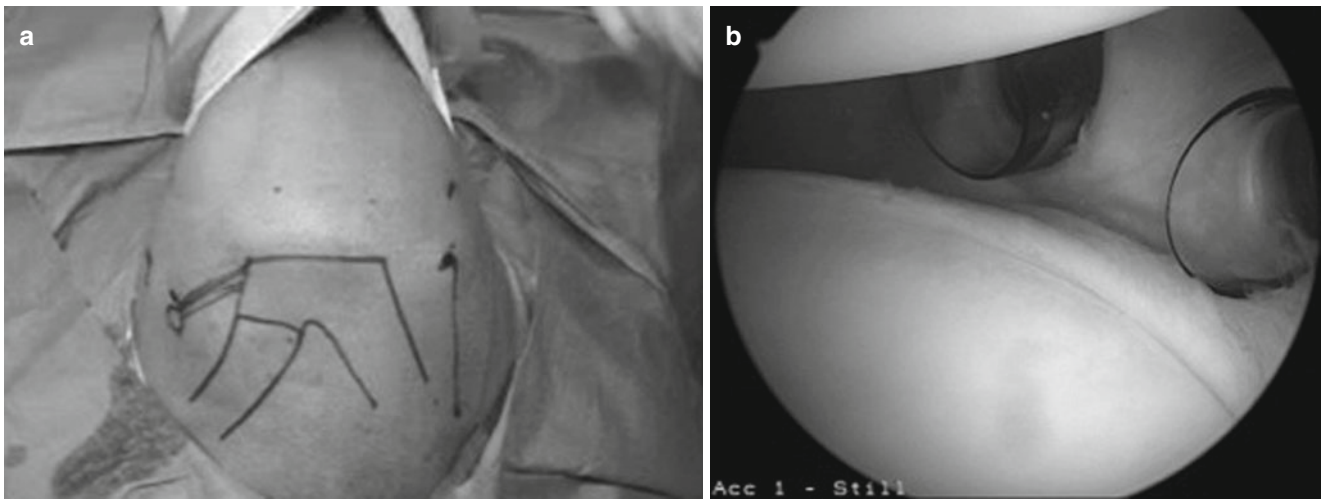

Fig. 18.4 (**a**) Portal placement for multidirectional instability. Standard posterior and anterior portals are established. 5 o'clock and 7 o'clock portals used to achieve a satisfactory trajectory for inferior anchors. (**b**) View from anterior portal demonstrating posterior-superior and posterior-inferior portals

The lateral decubitus position is maintained with a bean bag and careful padding of the peroneal and axillary nerves. The arm is placed in 5–10 lb of longitudinal traction with a traction sleeve at 15° of forward flexion and 65° of abduction. This position maximizes access to the posterior and inferior shoulder joint. We prefer to rotate the bed approximately 180° to provide unobstructed access to the anterior and posterior portals. A bump may be used in the axilla to create more space in the posterior-inferior region. It may be helpful to place the arm in 90° of external rotation to prevent overtightening of the anterior shoulder structures.

Portal Placement

Exact portal placement should be tailored to address the pathologic findings identified on physical exam and diagnostic arthroscopy. Many surgeries can be performed through four portals, although more may be required. All surgeries begin with a posterior portal placed approximately 1 cm inferior and just medial to the lateral border of the acromion. Compared to a standard posterior portal, a more lateral position for this portal permits improved access to the posterior glenoid. An anterior-superior portal is made with the aid of an 18-guage spinal needle, placed percutaneously through the rotator interval to ensure satisfactory trajectory for glenoid anchors. A percutaneous accessory posterior-lateral portal at the 7 o'clock position should be made approximately 1 cm lateral and 2 cm distal to the posterior portal. Placement of this portal is critical to allow access to the posterior-inferior glenoid (Fig. 18.4). After skin incision, a switching stick is used to penetrate the capsule. 8.25 mm clear cannulas are placed in through these portals to accommodate repair, instrumentation, and anchor placement. For the anterior pathology, small percutaneous incisions through the subscapularis may be made to allow satisfactory trajectory for the anterior anchors.

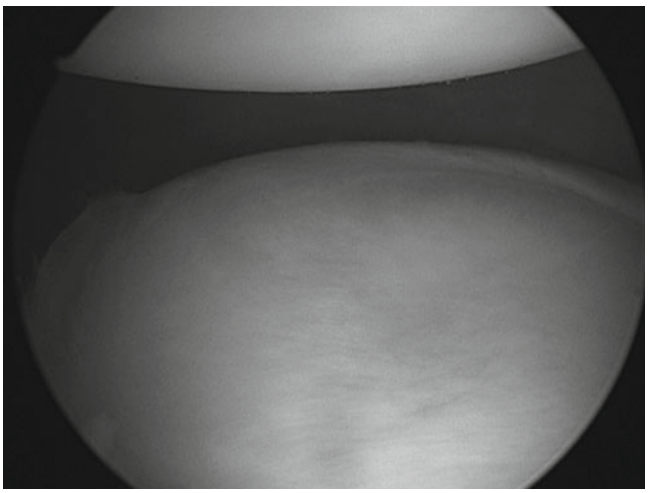

Fig. 18.5 Skybox view

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

Diagnostic arthroscopy should be performed to evaluate the labrum in its entirety, rotator cuff, capsule, articular surfaces, as well as rotator interval. Often there is a "drive-through" sign and "skybox" view with posterior-inferior laxity (Fig. 18.5). Careful examination may reveal a patulous capsule, labral splitting, 360° labral pathology, under-

surface partial tears of the rotator cuff, and widening of the rotator interval. Careful examination of the posterior structures should be performed from the anterior-superior portal. The fibers of the infraspinatus muscle may be visible through the thin posterior capsular tissue. The posterior labrum should be meticulously probed to identify any crack or tear. If such pathology is visualized, the labrum should be taken down and prepared for anchor repair. Three hundred and sixty-degree labral pathology may be identified in MDI patients. In areas where no labral pathology can be identified, the labrum can be used to anchor the capsule plication.

Step-by-Step Procedure (Box 18.1)

Box 18.1: Tips and Tricks

- Portal placement is critical. Ideal portal placement should be localized with an18-guage spinal needle. This needle should be used to touch all areas for planned repair, ensuring appropriate position and trajectory.
- Keep the fluid pressure as low as possible to minimize progressive distention, which can make the latter parts of the case difficult.
- The axillary nerve is at greatest risk anteriorly between the 5:30 and 6 o'clock position. The capsulolabral device should penetrate a maximum of 3 mm into the tissue to minimize risk to the nerve in this area.
- As knots tend to migrate towards the articular surface, it is critical to push the knot away from the glenoid during tightening. Patients may complain of clicking if knots are left too centrally.
- In patients with extremely lax capsules, a double-loaded anchor may be used. The first suture may be used to secure the capsulolabral tissue to the glenoid. The second suture is available if additional capsular plication is required.
- In MDI, if the posterior capsule is thin, the infraspinatus muscle may be incorporated into the plication near its insertion on the humerus.
- Do not incorporate the coracoacromial ligament into the rotator interval closure. Satisfactory rotator interval closure has been obtained when a small amount of internal rotator of the arm is visualized.
- If rotator interval closure is performed, the arm should be immobilized in 30–45° of external rotation to prevent postoperative internal rotation contracture.

After diagnostic arthroscopy, the posterior and inferior structures are addressed first. The posterior capsule should be gently abraded with a rasp to stimulate a healing response (Fig. 18.6). If disrupted, the posterior labrum should be fully elevated from the glenoid rim. Preparation of the posterior chondrolabral junction should be performed from an anterior portal, which allows the appropriate trajectory to minimize the chance of traumatic injury to the labrum or articular surface. A bleeding bony surface is created at the glenoid rim with an arthroscopic burr or rasp.

The essential lesion to address is the patulous capsule. Optimal repair strategy includes shifting the capsular tissue from an inferior to superior position. Each plication reduces the available working area. Therefore, anchors should be sequentially placed and fixed from inferior to superior along the rim of the glenoid. Up to five anchors may be required to adequately address pathology from the 6 o'clock to approximately 10 o'clock position posteriorly.

With the camera in the anterior portal, the first anchor should be placed at the 6 o'clock position through the posterior-lateral 7 o'clock portal. The anchor should be placed 2 mm onto the glenoid rim to create a stable bumper (Fig. 18.7). We prefer a 3 mm double-loaded suture anchor. All sutures should be brought through the posterior-superior portal. A curved capsulolabral device should be used through the posterior-lateral portal to penetrate the capsule approximately 1 cm inferior to the labrum. The capsule should become taut when it is brought to the level of the glenoid. It is important that the entry point for the suture hook is inferior to the placement of each anchor in order to shift the tissue superiorly. The device can then be used to penetrate the labrum. A PDS suture is passed through the capsulolabral device into the joint. An arthroscopic grasper can be used to bring one limb of the suture as well as the PDS shuttle through the posterior portal. The sutures are tied outside the cannula, and the suture limb shuttled through the

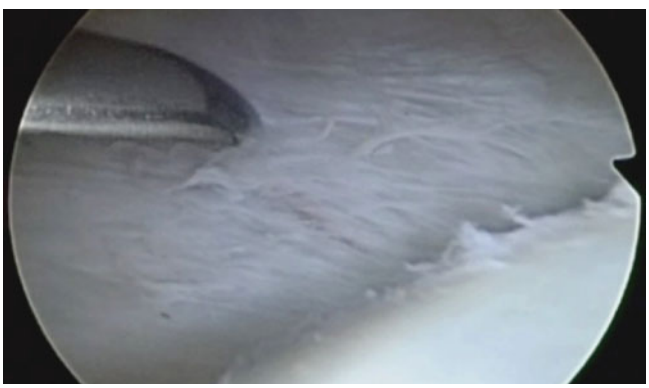

Fig. 18.6 Abrasion of the capsule with a rasp to stimulate the healing response

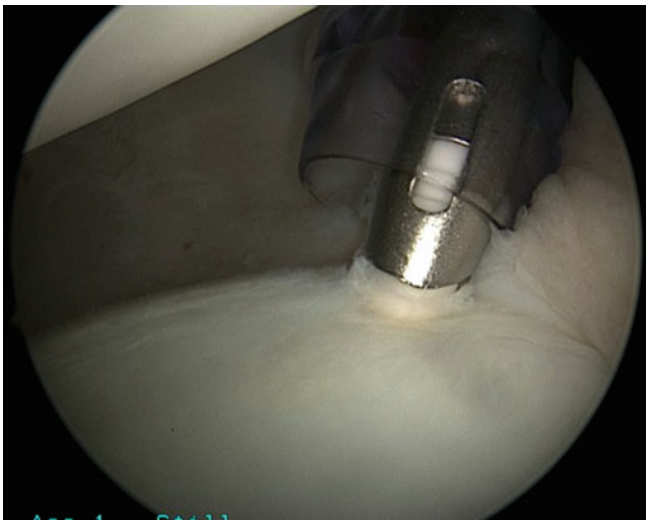

Fig. 18.7 View from the anterior portal. Initial anchor placement in the inferior glenoid at the 6 o'clock position, through the 7 o'clock portal, approximately 2 mm central to the glenoid rim

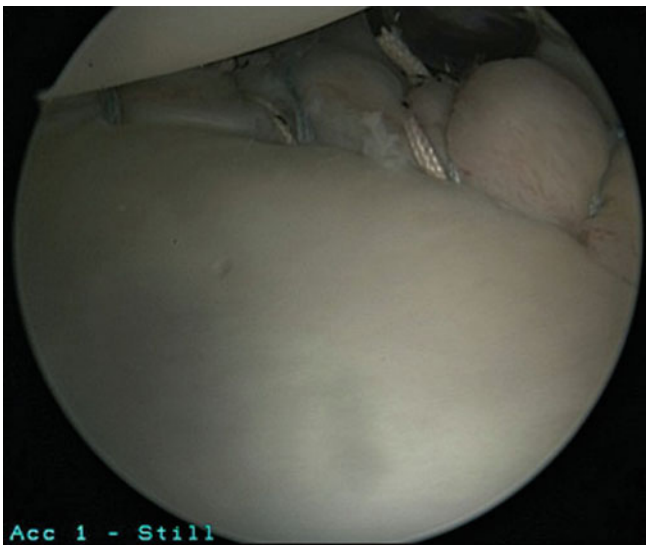

Fig. 18.9 View from the anterior portal. Multiple double-loaded anchors are placed approximately 8 mm apart posteriorly from the 6 o'clock to 9 o'clock position

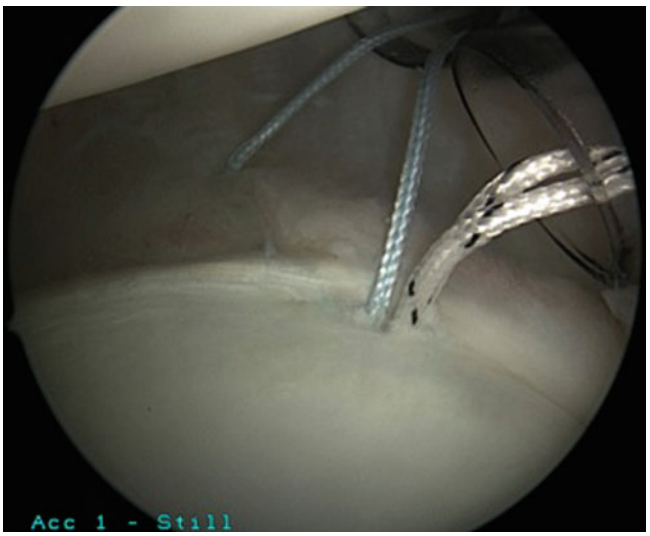

Fig. 18.8 View from the anterior portal. The suture is shuttled through the capsule and labrum with a capsulolabral device and tied. The entry point for the capsulolabral device should be approximately 1 cm interior to the anchor on the glenoid

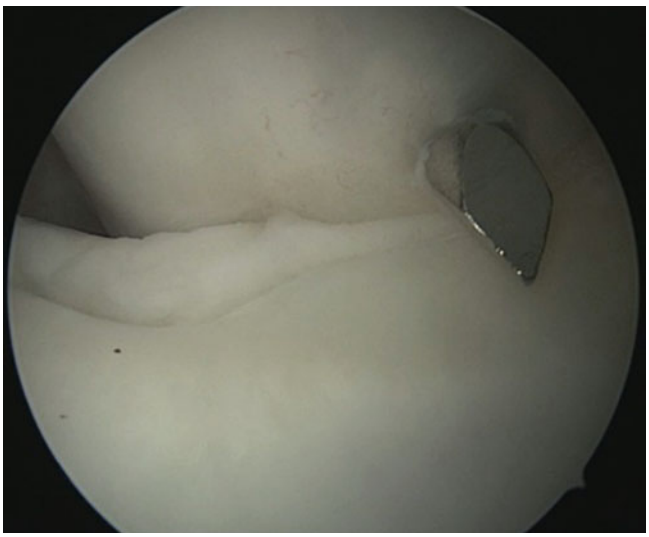

Fig. 18.10 View from the posterior portal. In order to place an anterior inferior anchor, a small percutaneous incision through the subscapularis is made through which an anchor trocar can be passed

capsulolabral tissue (Fig. 18.8). Both ends of the suture are brought through the posterior-lateral portal. The suture is tied with a non-sliding knot backed up with three alternating half hitches, ensuring that the post-limb remains away from the articular surface. The other suture is passed and tied in a similar fashion to provide additional plication. Subsequent anchors can be placed along the posterior glenoid up to the 9 o'clock position using the 7 o'clock and posterior portals for proper anchor trajectory (Fig. 18.9). For areas without frank labral tears, simple plication sutures through the capsule and labrum are passed and tied.

Once the posterior and inferior structures have been repaired, the anterior capsulolabral structures should be addressed. With the arthroscope in the posterior portal, the anterior glenoid can be prepared with a burr after the labrum has been liberated from the glenoid. The anterior capsule should be abraded with a rasp and can be safely performed from the posterior-lateral portal. An 18-guage spinal needle is used to identify a satisfactory trajectory for a 5 o'clock anchor, which is placed through a small percutaneous incision through the subscapularis tendon (Fig. 18.10). A curved capsulolabral device is used through

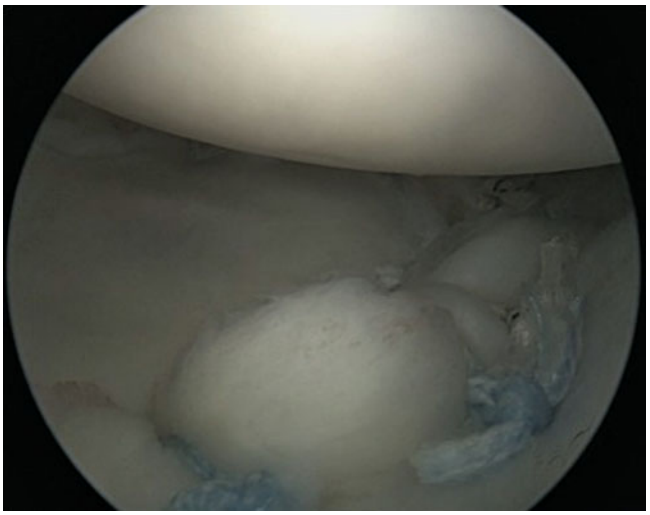

Fig. 18.11 View from the posterior portal demonstrating satisfactory glenoid bumper without capsular redundancy

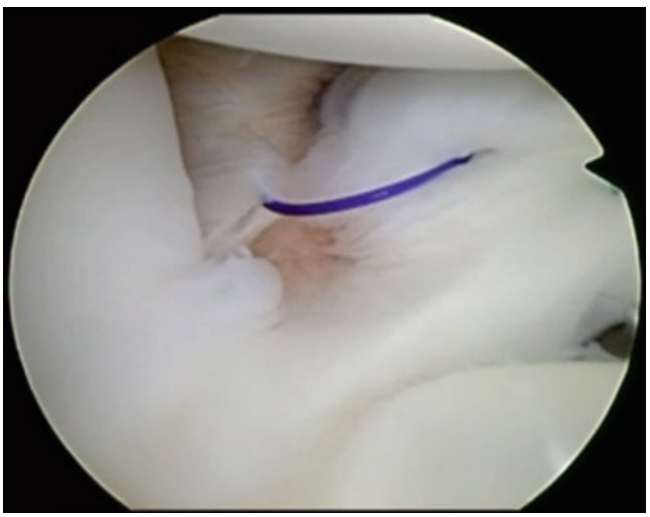

Fig. 18.12 View from the posterior portal. PDS suture used to close the rotator interval

the posterior-lateral portal to capture approximately 1 cm of tissue inferior to this anchor. The capsule should become taut when brought to the level of the anchor. The PDS shuttle and one limb of the suture are brought through of the posterior-lateral portal where they are tied outside the body. The suture limb is shuttled through the capsulolabral tissue and tied away from the glenoid surface. Anchors should be progressively placed superiorly along the glenoid. Additional sequential percutaneous incisions may be required through the subscapularis. In general, we place no fewer than three anchors from 6 o'clock to 3 o'clock positions. The final repair construct should produce a glenoid bumper providing sufficient tension along the tissue without capsular redundancy (Fig. 18.11).

The rotator interval should then be assessed clinically. If persistent laxity is identified with the sulcus sign in 30° of external rotation, the surgeon should consider rotator interval closure. Arthroscopic determination of rotator interval closure is difficult, but displacement of the biceps tendon may suggest pathology. An 18-guage spinal needle is passed through the supraspinatus tendon 1 cm medial to the humeral head. A PDS suture is threaded through this needle into the joint and retrieved though the superior aspect of the subscapularis (Fig. 18.12). The PDS limbs are retrieved in the subacromial space where they are tied. This technique may need to be repeated with more medial stitches to adequately close the rotator interval.

Postoperative Care

In general, the postoperative rehabilitation protocol for patients undergoing arthroscopic treatment of multidirectional instability should be individualized for each patient, depending on the location of intra-articular pathology, the direction of primary instability, and the type of surgical treatment used. Immediately after surgery, patients are immobilized in a sling with a 30° abduction pillow. The arm should be in neutral rotation. Patients are instructed to begin passive pendulums and gentle passive scapular retraction exercises. In addition, they are allowed to perform active range of motion of their wrist, hand, and elbow. The average immobilization period is 4–6 weeks, with timing of progression dictated largely by the magnitude of the injury and the extent of the repair.

Active-assisted glenohumeral range of motion is generally instituted at the 4–6-week mark. In cases that involve repair of posterior instability, internal rotation and cross body (horizontal) adduction are restricted for a full 6 weeks postoperatively. Along with progression from active-assisted to active ROM protocol, the patient may begin rotator cuff strengthening and scapular stabilization program at this time. Patients progress to pulleys and weights to improve deltoid and rotator cuff strength and endurance. Sport-specific training programs are then initiated prior to return to play. The majority of patients may return to play or return to manual labor approximately 6 months postoperatively. Range of motion and strength should be at least 80–90 % of their opposite extremity prior to being cleared for full activities. After completion of formal rehabilitation, it is usually necessary for a patient to maintain shoulder stability and strength through a home exercise program approximately two to three times per week.

Literature Review

Many patients diagnosed with MDI can be treated successfully with nonoperative management. Burkhead and Rockwood reported 88 % good or excellent results at 2-year follow-up after conservative treatment [21]. However, 70 % of patients treated conservatively had opted for surgical treatment or had fair or poor ratings for their shoulders at 7- to 10-year follow-up [22].

Arthroscopic treatment of MDI successfully improves pain and function in most patients [5, 23–25]. Even patients with large labral lesions of greater than 270° can be successfully treated, although they may have some mild persistent instability [5]. Although most patients improve in terms of pain and function, Baker et al. reported that only 65 % of athletes were able to return to the same level of sport after arthroscopic treatment [23]. The most common complication of multidirectional instability treatment is persistent or recurrent instability. The incidence is between 2 and 12 % in current arthroscopic series [6, 26–28]. Aggressive capsular plication may result in postoperative stiffness, especially with external rotation [29]. Postoperative axillary nerve palsy has not been reported, but its proximity to the operative field puts this structure at risk.

Summary

Multidirectional instability presents a difficult clinical entity to diagnose and treat as it represents a wide spectrum of disease. Conservative management with physical therapy remains the mainstay of initial treatment. If surgical intervention is warranted, satisfactory patient outcomes can be achieved when pathologic lesions that correlate with preoperative findings are addressed during surgery. Successful surgery is dependent upon successful volume reduction and restoration of balanced capsulolabral attachments.

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The Overhead Athlete

19

W. Ben Kibler, John E. Kuhn, Aaron D. Sciascia, and Tim L. Uhl

Introduction

The overhead athlete with a painful shoulder may have many causative factors contributing to the symptoms. This chapter will present an overview of the mechanisms through which shoulder symptoms develop and provide guidelines for evaluation and treatment.

The term "disabled throwing shoulder" (DTS) [1] is a general term that describes the limitations of function that exist in symptomatic overhead athletes – from baseball players to tennis players – in that they cannot optimally perform the task of throwing or hitting the ball. In the large percentage of cases, the DTS is the result of a "cascade to injury," [1] a process in which the body's response to the inherent demands of throwing or hitting result in a series of alterations throughout the kinetic chain can affect the optimal function of all segments in the chain. These alterations of function, termed dysfunctions, can have anatomical, physiological, and/or biomechanical causative factors (Table 19.1).

Very rarely will one specific anatomic injury or physiologic alteration be present by itself or be responsible for all of the disabling symptoms. More commonly, several will be found and act together to create the total dysfunction. The athlete with the DTS must be evaluated for all of the possible factors to arrive at a complete and accurate diagnosis that will allow treatment and serve as a baseline for functional outcomes assessment.

Physiological Factors in DTS

Muscle Strength Imbalance

Muscle strength balance is key in dynamically stabilizing the glenohumeral joint throughout the entire range of arm motion, accurately positioning the glenoid and humerus to confer ball and socket kinematics, and stabilizing the scapula on the trunk as a stable base for arm action. Important force couples include anterior/posterior rotator cuff activation to compress the humeral head into the glenoid fossa, rotator cuff/deltoid to stabilize the moving arm into the socket, and upper trapezius/lower trapezius: serratus anterior to position and stabilize the scapula. Multiple studies have demonstrated

Table 19.1 Causative factors for DTS

Anatomical
Superior labral (SLAP) injuries
Biceps injuries
Rotator cuff injuries
Internal Impingement
Instability (Bankart/capsular laxity)
Physiological
Muscle strength imbalance
Muscle inflexibility
GH internal rotation deficit (GIRD)
Total arc of motion deficit (TAMD)
Biomechanical
Kinetic chain deficits
Scapular dyskinesis
Altered mechanics

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imbalances of muscle strength in both asymptomatic and symptomatic overhead athletes [2–5], all of which demonstrate a relative or absolute strength increase in the anterior rotator cuff muscles and a relative or absolute strength decrease in the posterior rotator cuff muscles. This mismatch alters the anterior/posterior force couples that stabilize the glenohumeral (GH) joint and increase concavity/compression [6, 7] and decreases the deceleration capability for the shoulder in the follow-through phase of throwing or serving. Weakness in external rotation has been determined to be a risk factor for shoulder injury [8]. Weakness and alteration in activation sequencing of the serratus anterior and lower trapezius are frequently seen in the DTS athlete. These alterations contribute to scapular dyskinesis, which has been associated with impingement [9], rotator cuff injury [10], and labral injury [1].

Muscle Inflexibility

Multiple muscles around the shoulder have been found to develop tightness as a result of throwing. The most commonly affected muscles are the pectoralis minor, subscapularis, and latissimus dorsi. The pathophysiology is believed to result from chronic tensile overload and resulting scar or from a muscle adaptive response [11]. The tight pectoralis minor creates a tendency for scapular anterior tilt and acromial downward tilt decreasing the arm's ability to cock or reach maximal abduction [12–14]. The tight subscapularis decreases arm external rotation limiting arm cocking. The tight latissimus dorsi limits overhead positioning and cocking.

Glenohumeral Internal Rotation Deficit (GIRD) and Total Range of Motion Deficit (TROMD)

These alterations are seen in virtually every athlete with DTS. They create multiple problems in and around the throwing shoulder, including scapular dyskinesis due to a windup of the tight posterior structures [15], external impingement due to anterior superior humeral head translation in follow-through [16, 17], and posterior-superior humeral head translation in cocking and anterior superior translation in flexion which increase labral shear [1, 18].

Biomechanical Factors in DTS

Kinetic Chain Deficits

The kinetic chain is the mechanism by which force is generated from the large muscles of the core, transferred through the funnel of the shoulder, and passed to the delivery point, the hand [19–22]. The optimal coordination of the body segments to generate and disseminate the forces results in the minimization of the degrees of freedom in the entire chain which facilitates the efficient completion of the task [23]. Finally, the interactive moments produced through this system protect the joints from excessive loads [20, 23]. Kinetic chain deficits in the legs, hips, trunk, and scapula have been found in 50–67 % of athletes with shoulder injuries [24–27].

A screening exam for the kinetic chain deficits includes observation of standing posture, the one-leg stability series (stance and single leg squat) [26], and measurement of trunk flexibility and strength.

Scapular Dyskinesis

The scapula plays key roles in linking the force generating areas of the core to the force delivery site (the hand) and in providing a dynamically stable base for the moving arm. Studies have accurately documented the composite three-dimensional motions of the scapula in association with arm movement and throwing [28–31]. They show a pattern of progressive retraction, upward rotation, posterior tilt, and controlled internal/external rotation to maximize glenohumeral stability during arm motion. Throwers have specific compensations in position but display the same direction of motions during arm motion [31]. Deviations from these patterns are considered to have implications for injury [27, 32–34].

Scapular dyskinesis (dys – alteration of, kinesis – motion) represents altered dynamic motion and static position of the scapula [35] which alters the efficiency of the throwing motion.

External shoulder impingement, internal shoulder impingement [9], anterior capsular laxity [36], labral injury [1], and rotator cuff weakness [10], all of which can create symptoms and/or exacerbate the dysfunction in the DTS, are found in association with scapular dyskinesis in a large percentage of DTS patients [1, 37]. Addressing the dyskinesis has been shown to decrease impingement symptoms [38], improve rotator cuff strength [39], and decrease symptoms in labral injury [40].

Scapular resting position and dynamic motion can be evaluated by observing the scapula as the arms move into forward flexion and descent [41]. Medial border prominence is characterized "yes" – seen, dyskinesis present, or "no" – not seen, dyskinesis not present. Finally, the effect of dyskinesis on symptoms can be estimated by corrective maneuvers. The scapular assistance test creates posterior tilt and can decrease external impingement symptoms [15, 42]. The scapular retraction test [15] and/or scapular reposition test [5] creates external rotation and posterior tilt and can increase demonstrated rotator cuff strength and decrease internal impingement symptoms in labral injury [4, 5].

Altered Mechanics of the Throwing or Serving Motion

The throwing or serving motion creates such high forces and loads, requires such a wide range of motion, and is done so frequently that efficient mechanics of the motion is one of the most basic points to be achieved in order to develop proficiency in throwing or serving. The ability to detect alterations in mechanics can help the clinician in the evaluation of DTS.

While the athlete is in the "cascade to injury" process, he or she will continue to try to optimally complete the task of throwing or serving the ball. If one body segment or area is injured or altered, other areas will try to compensate by changing the position or motion or by trying to generate more force or go through a different motion. These alterations can be observed either visually or through video tape.

The baseball throwing motion was studied by videotaping, and specific deficits in key trunk and arm motions that correlated with efficient or inefficient mechanics were identified [43]. The efficient motions were associated with lower shoulder torque and lower elbow valgus load. The five effective motions identified are the pelvis leading the trunk towards home plate during the early cocking phase, the throwing hand on top of the ball (pronated forearm) during in early cocking, the elbow reaching maximum height by stride foot contact, the lead shoulder closed and pointing towards home plate at stride foot contact, and the stride foot pointed towards home at stride foot contact [43].

Similarly, a system of visual or videotape evaluation of the tennis serve has been developed. Key "nodes" of efficient serve motions that correlate with maximum serve speed and force development include the use and position of the feet on the ground (back foot push up and through), knee flexion to extension, hip counter rotation and posterior tilting, coordinated trunk/hip rotation, and shoulder cocking position in line with the body [10].

This knowledge is important to help understand what the normal mechanics are and how the athlete is attempting to compensate for any anatomic, physiologic, and/or biomechanical deficits. It can identify some of the altered factors, can guide restoration of the altered factors, and suggest preventative measures to proactively correct the altered factors before the full blown symptoms of DTS occur.

Anatomic Factors in DTS

These factors are the most commonly cited as causative and most clinical exam and imaging techniques are developed to identify them. While they are at the heart of the problem and need to be corrected to provide optimum anatomy, they are commonly not the only factors.

Superior Labral (SLAP) Injuries

SLAP lesions are the most common injury associated with the DTS [44, 45]. They are predominantly found in the posterior-superior glenoid, from 10:00 to 12:30 on the right shoulder, although they may extend anterior and posterior from that region. Current theories describe the injury resulting from a pathological tightness of GH internal rotation creating a peel back of the biceps/labrum complex [1, 18], although others suggest a repetitive internal/external rotation ("weed pulling") mechanism [46]. These lesions would result in loss of the roles of the labrum in normal shoulder function.

Labral roles in shoulder function have been traditionally identified as an attachment site for the biceps, a bumper to deepen the glenohumeral socket and to improve stability by minimizing glenohumeral translation [47], and to help increase capsular tension [48]. However, labral roles may actually be more complex. The actual role of the superior labrum as a mechanical bumper is controversial. The amount of increased mechanical GH translation after superior labral resection was only 10 %, meaning that the superior labrum likely has other functional roles other than just a mechanical stability role [49]. Recent biomechanical studies [6, 48] have highlighted three other important functions for the labrum:

1. As a deformable structure with high compliance interposed between two surfaces to more evenly distribute contact pressures between the surfaces, increase boundary lubrication, and maximize concavity/compression characteristics – much like a washer between two surfaces
2. As a pressure sensor maximizing proprioceptive feedback
3. As an attachment site for muscles and ligaments, to optimize their tension

Experimental labral release resulted in significant changes in capsular tension [48]. Another cutting study demonstrated increased glenohumeral translation that was restored to normal by posterior-superior labral repair [40].

The intact labrum would result in optimal glenohumeral kinematics in dynamic shoulder motion, resulting in smooth GH motion in rotation, stable ball and socket kinematics, and maximal force transfer from the engine of the core and legs through a stable linkage at the shoulder to the delivery mechanism, the hand. The labrum should be seen as a key component for functional glenohumeral stability.

Many labral "injuries" diagnosed on imaging are not clinically significant, in that they are not contributory to the symptoms or dysfunction in the DTS. The clinically significant SLAP injury is one in which the anatomic alteration in the labrum results in elements of the clinical history of the dysfunction that can be attributed to the loss of labral roles, and the injury can be highlighted by specific physical exam tests that are clinically useful for detection of the injured labrum. It is a positive diagnosis, not a catch-all term in the presence of shoulder pain of unknown etiology.

Fig. 19.1 Depiction of the modified dynamic labral shear. (**a**) The patient's scapula is stabilized, and the arm is moved into 100-110° of abduction and 90° of external rotation. (**b**) The arm is lowered into 60° of abduction, maintaining external rotation to create a peel-back and shear on the posterior labrum

The history findings suggestive of loss of labral roles include:

- Pain upon external rotation/cocking – indicating increased posterior-superior translation [1, 6, 49]
- Weakness in clinical or functional arm strength – indicating pain and/or increased translation
- Symptoms of internal derangement (clicking, popping, catching, sliding) – indicating loss of the bumper effect or washer effect or decreased capsular tension
- Feeling of a "dead arm" [50] – indicating loss of proprioceptive feedback, decreased capsular tension, and increased translation

These are not exclusively seen in a labral injury, but point towards the loss of labral roles.

Of the labral exam tests, the *modified dynamic labral shear* (*M-DLS*) *test* [51] has been shown to be of high clinical utility in the evaluation of labral injuries when the test is performed in the manner described (Fig. 19.1). It is performed by abducting the arm and flexing the elbow to 90°. The arm is then abducted in the scapular plane to above 120° and externally rotated to tightness. A shear load is applied to the joint by maintaining external rotation and horizontal abduction and lowering the arm from 120° to 60° abduction. A positive test is indicated by reproduction of the pain and/or a painful click or catch in the joint line along the posterior joint line between 120° and 90° abduction. The test has a sensitivity of 0.72, specificity of 0.98, positive predictive value of 0.97, and a positive likelihood ratio of 31.6 [51]. Other labral exam tests advocated include *O'Brien's active compression test* [52], the *relocation test* [53] with pain as the indicator, and an anterior levering maneuver to place a posterior load and shear.

In addition, intra-articular exam tests can provide clues to loss of labral roles. A positive *painful arc of motion test* in the Hawkins-type motion, with no relief by scapular posterior tilt in the scapula assistance test [34] indicates increased translation. A positive O'Brien's maneuver indicates loss of the washer effect, increased biceps tension, and increased translation. The M-DLS test has been shown to have high clinical utility as it specifically replicates a peel-back phenomenon. A positive test indicates loss of biceps stability, loss of washer, and increased translation.

Biceps Injuries

Injuries of the intra-articular biceps tendon are less common in throwers. It is thought they develop from repetitive tensile strain, but no specific mechanisms for injury have been presented. It may be due to excessive tensile loading or damage to the medial and lateral pulley system at the biceps outlet. There is controversy regarding the role of the biceps in shoulder stability and function. Low load or static situations have failed to demonstrate significant alteration in kinematics if the biceps is cut or is absent [54]. However, many studies of biceps contribution at higher loads and large motions show that the biceps plays important roles in these situations [55–57].

The dysfunction that is associated with the DTS results from loss of these higher load biceps roles in the GH joint stability, with decreased joint stability at maximum abduction/external rotation and loss of maximum concavity/compression, both of which result in decreased precision in the arthrokinematics at the high loads and forces seen in the overhead athlete [40, 48, 58]. Pain from biceps tendinopathy can also create a major dysfunctional problem.

Clinical exam for biceps injuries is also imprecise. The most widely used tests include *Speed's test* and *Yergason's test*. A

Fig. 19.2 Depiction of the uppercut maneuver. (**a**) The elbow is placed in 90° of flexion and 40° of supination with a compression load through the arm. (**b**) The arm is forward flexed towards the head, like an upper cut punch in boxing. The resistance load is applied gently through the motion. A positive test reproduces the anterior shoulder pain in the biceps groove, or creates a pop or click indicating biceps subluxation

more recently described test, the *upper cut* [51] has been shown to have higher clinical utility, although it is still too insensitive for precise diagnosis (Fig. 19.2). A combination of clinical symptoms (localized pain, point tenderness to palpation, tendon instability, and weakness of supination) plus the clinical tests can result in acceptable clinical utility for identification.

Rotator Cuff Injuries

These injuries are commonly seen in the DTS and range from tendinopathy/tendinitis to partial undersurface tears to full-thickness tears [1, 44–46]. Controversy exists regarding their pathophysiology. They may be caused by external impingement [59], may be created through the "internal impingement" process [9] or a torsional stress process (hypertwist) [1], or may be a reflection of a chronic tensile process with apoptotic changes [60, 61].

Very frequently, SLAP injury and rotator cuff injury are found to co-exist. This coupling is usually referred to as "internal impingement," thought to be due to compressive, shearing, and twisting loads on the rotator cuff and superior labrum in maximal shoulder abduction/external rotation.

Clinical Presentation

The clinical exam will demonstrate many features of the anatomical, physiological, and biomechanical deficits associated with all throwers with DTS. They include kinetic chain deficits such as hip and trunk weakness and inflexibility, scapular dyskinesis, and glenohumeral internal rotation deficit (GIRD). Specific exam clues for SLAP lesions include a positive dynamic labral shear (DLS) [51] or active compression (O'Brien's) test [52]. Specific exam clues for rotator cuff involvement include external rotation weakness at 90º abduction [62], positive external rotation lag sign [18], and weakness with the arm horizontally adducted in front of the opposite shoulder, a Whipple maneuver.

Imaging

The labral injury can be confirmed by MRI, MRI arthrography, or CT arthrogram, but should not be defined by imaging. Specific criteria have been developed to distinguish a labral alteration, but MRI is best viewed as a static estimation of labral status with inconsistent relation to the dynamic roles. A percentage of patients will demonstrate "labral tears" without symptoms relating to loss of the labral roles.

MRI imaging studies in asymptomatic throwers [63–67] have demonstrated that cuff disease (including surprisingly high levels of partial-thickness rotator cuff tears) and effusions – findings that would normally be considered pathologic – are often seen in an asymptomatic thrower's shoulder and may not be a source of symptoms. The aphorism "treat the patient, not the X-ray" is particularly relevant in the thrower with DTS.

Treatment Guidelines

The Concept of Adaptive Pathology

Throwing requires repetitive high loading of the osseous and soft tissue structures. In order to achieve extremes of external rotation required for throwing with high velocity, these structures undergo adaptive remodeling and possibly failure. The fact that MRI scans on asymptomatic throwers demonstrate significant findings that would normally be considered the source of the athlete's pain suggests that these athletes have developed these anatomic alterations in order to throw at high levels, just as their humerus has remodeled into greater retroversion. Chronic SLAP lesions in throwers may allow for increases in external rotation required for throwing. Articular-side partial-thickness rotator cuff tears may represent failure of the tissue in external rotation, again allowing for extremes of external rotation required for high-level throwing. It is conceivable that in some throwers, the anatomic repair of these structures will lead to an inability to achieve the extremes of external rotation required to throw at high velocity and may end a thrower's career. This may explain the relatively poor results obtained with the surgical treatment of the pathology. The throwing athlete can be considered on the edge of a cliff in terms of the demands of the throwing motion on the anatomic structures. Treatment of the DTS should be to put the athlete back on the edge of the cliff and not restore his shoulder to "normal" anatomy.

Nonoperative Treatment

Nonoperative rehabilitation has often been advocated for DTS patients. Protocol content is directed towards improving the deficits in shoulder rotation and scapular muscle flexibility, strength, and strength balance and maximizing kinetic chain function [40, 68–71]. Multiple protocols have been described, but evidence regarding the exact indications, role, and effectiveness of the protocols is not clear. One study reported 49 % of patients had a positive outcome [72]. This study showed that a specific rehabilitation program can be an effective treatment in many DTS patients and should be implemented as the first treatment options.

Surgical Treatment for the DTS Patient

Surgery is to be used only after extensive and appropriate rehabilitation fails. The rehabilitation must address deficits in internal rotation (GIRD) and total ranges of motion (TROMD), scapular dyskinesis, and muscular and kinetic chain deficits. If the athlete improves clinically with regard to GIRD, TROMD, and dyskinesis, but still has pain and cannot throw, surgery may be offered in an attempt to salvage a career. Surgery should be considered a method to improve the anatomy within the shoulder so that rehabilitation can be successful. When performing surgery, a minimalist approach is ideal. In rotator cuff disease, debridement is likely preferable to repair, and if repair is performed, a transtendinous repair of the delaminated rotator cuff is likely to produce better outcomes than repair to bone. With regard to SLAP lesions, repairing the "peel-back" posterior labrum is performed surgically, but it is important to avoid over-constraining the biceps, which serves as an important restraint to external rotation of the abducted arm [58].

Arthroscopic Treatment: Surgical Technique

SLAP Lesions

The results for current methods of surgical treatment of superior labral tears in the thrower's shoulder have been reported [73–92]. If return to play at the same or higher level is used as the outcome measure of interest, only 4 of 21 series reported success rates at or above 85 % [85, 86, 91, 92]. As can be seen there is a great degree of variation with successful outcomes ranging from 22 to 94 %. This likely relates to the low level of evidence of these series, as there is no controlling for acuity of the SLAP lesion, location of the SLAP lesion [73], presence of additional pathology, indications for surgery, surgical technique, postoperative rehabilitation protocols, and willingness of the athlete to return all of which may affect the outcome. These results have led many throwing athletes to have great concern if their surgeon recommends labral surgery [93]. As described above athletes with SLAP lesions and rotator cuff tears seemed to fare worse than those with isolated SLAP lesions [73, 74]. Interestingly, one study demonstrated that a history of an acute

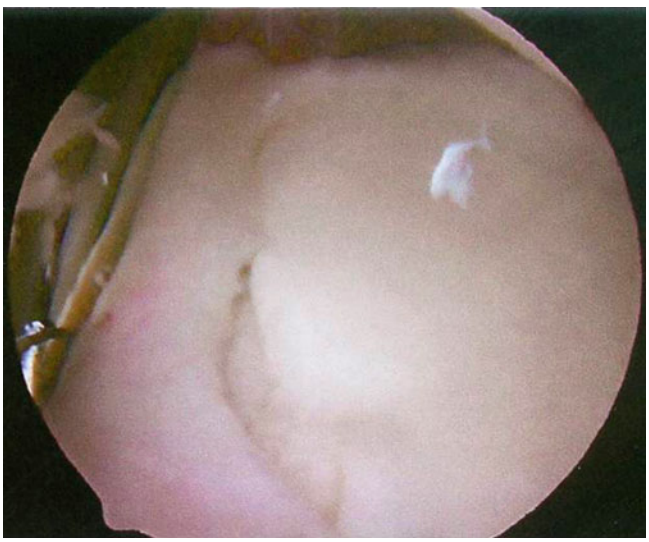

Fig. 19.3 Example of a type II superior labral lesion

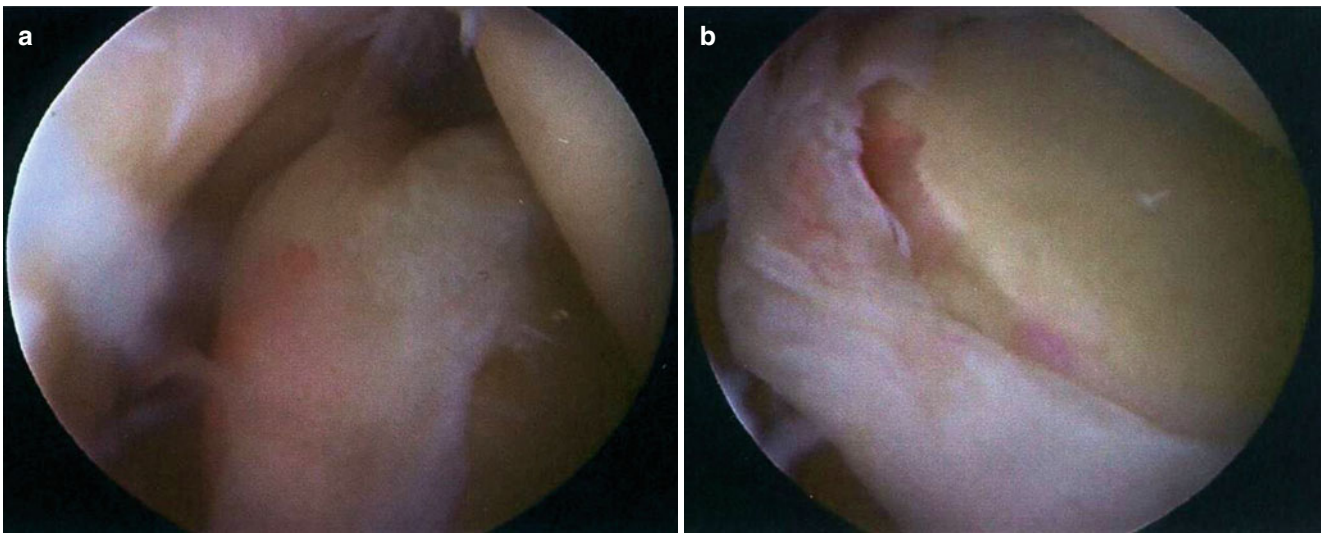

Fig. 19.4 (**a**) Example of static labral injury. (**b**) Example of the peel-back phenomenon when external rotation stress is applied to static injury

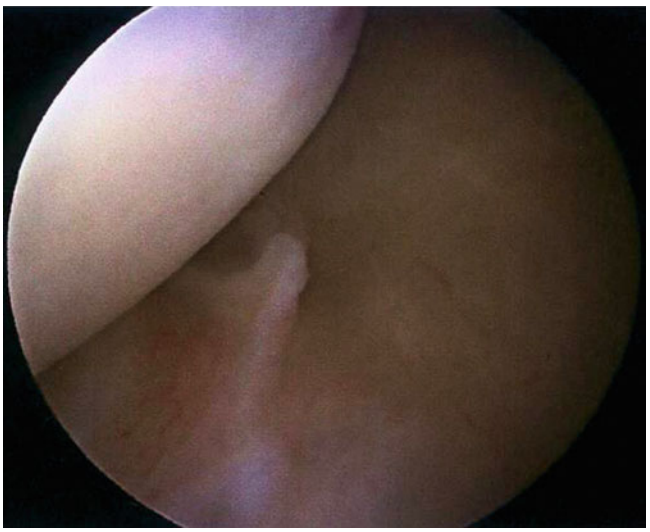

Fig. 19.5 Some labral tears will extend into the substance of the labrum. Frequently the posterior band of the inferior glenohumeral ligament (P-IGHL) will be lax, showing no distinct band

injury had better return to play rates than having chronic symptoms [75]. These overall poor results point to the need for a more detailed understanding of what intra-articular pathology needs to be treated and how it should be treated.

If arthroscopy is recommended, the arthroscopic evaluation of the suspected labral injury must be specific in order to understand and treat the labral injury properly. The arthroscopic findings most frequently associated with a clinically significant labral injury include:

1. A type II or higher lesion denoting loss of attachment from the glenoid [94] (Fig. 19.3)
2. A peel-back phenomenon indicating labral detachment, increased compliance, loss of washer effect, and loss of bumper effect [1, 6, 49] (Fig. 19.4b)
3. Glenoid articular cartilage damage or chondromalacia indicating increased translation [95]
4. Loss of capsular tension indicated by a drive-through sign or loss of tension in the posterior band of the inferior glenohumeral ligament (P-IGHL) (Fig. 19.5)
5. Continuation of the superior labral tear away from the glenoid into the substance of the labrum, indicating extension of the peeling back into the fibers of the capsule (Fig. 19.5)
6. Increased posterior labral thickness, indicating increased translation and shear with compression on the labrum
7. Excessive posterior inferior capsular thickness and scar indicating end-stage capsular damage that helps create GIRD [44]

Care must be taken to differentiate labral detachment from anatomic variants such as sublabral foramina, a Buford complex attachment of the middle glenohumeral ligament, or a meniscoid-like labral attachment that does not peel back [96].

Based on these principles, arthroscopic treatment guidelines for labral injury include:

1. Evaluation of the peel back, labral injury and mobility, glenoid surface, and capsular tension by direct visualization
2. Preparation of the glenoid to maximize bone to labrum healing (Fig. 19.6)
3. Multiple anchor placement to secure at least 2-point fixation of the labrum on the posterior-superior glenoid (10:30 and 11:30 on the right shoulder) (a double-loaded single anchor is still only one-point fixation)
4. Placement of enough posterior-superior anchors to eliminate the peel back (Fig. 19.7)
5. Evaluation of biceps mobility after anchor and suture placement to make sure there is adequate motion of the biceps in shoulder external rotation
6. Fixation of the insubstance labral injury to improve the posterior capsular tension (Fig. 19.8)

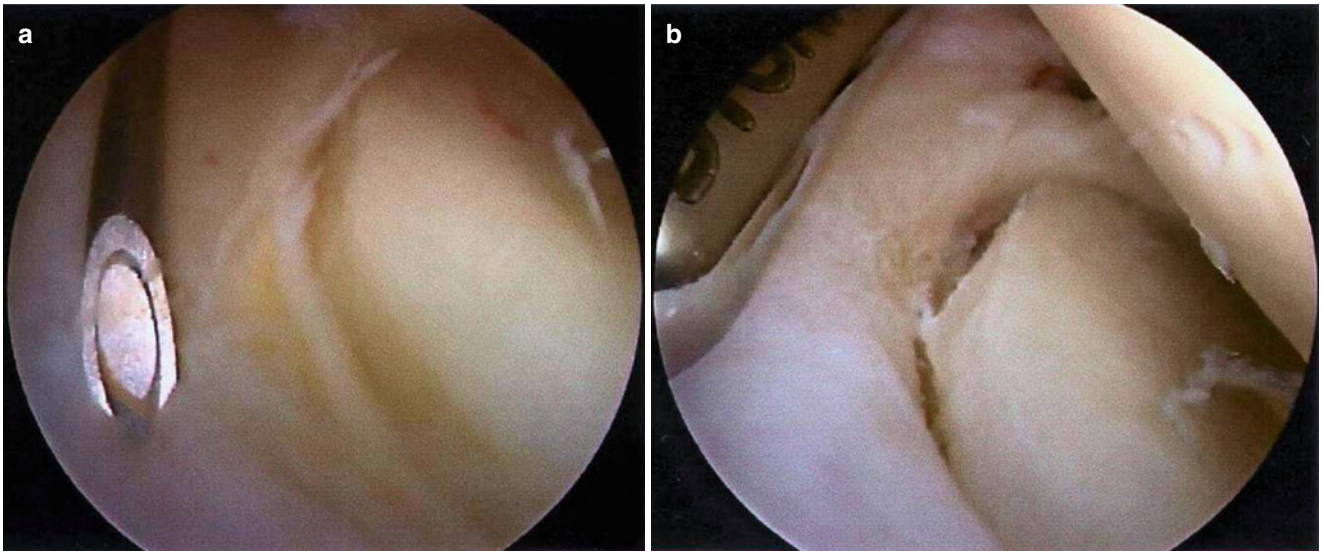

Fig. 19.6 Preparation of the glenoid prior to labral repair. (**a**) The labral injury demonstrates peel back, separation away from the glenoid, and occasionally granulation tissue at the base of the lesion. (**b**) The glenoid must be abraded to a bleeding bed and the labrum should be mobilized so it can be advanced

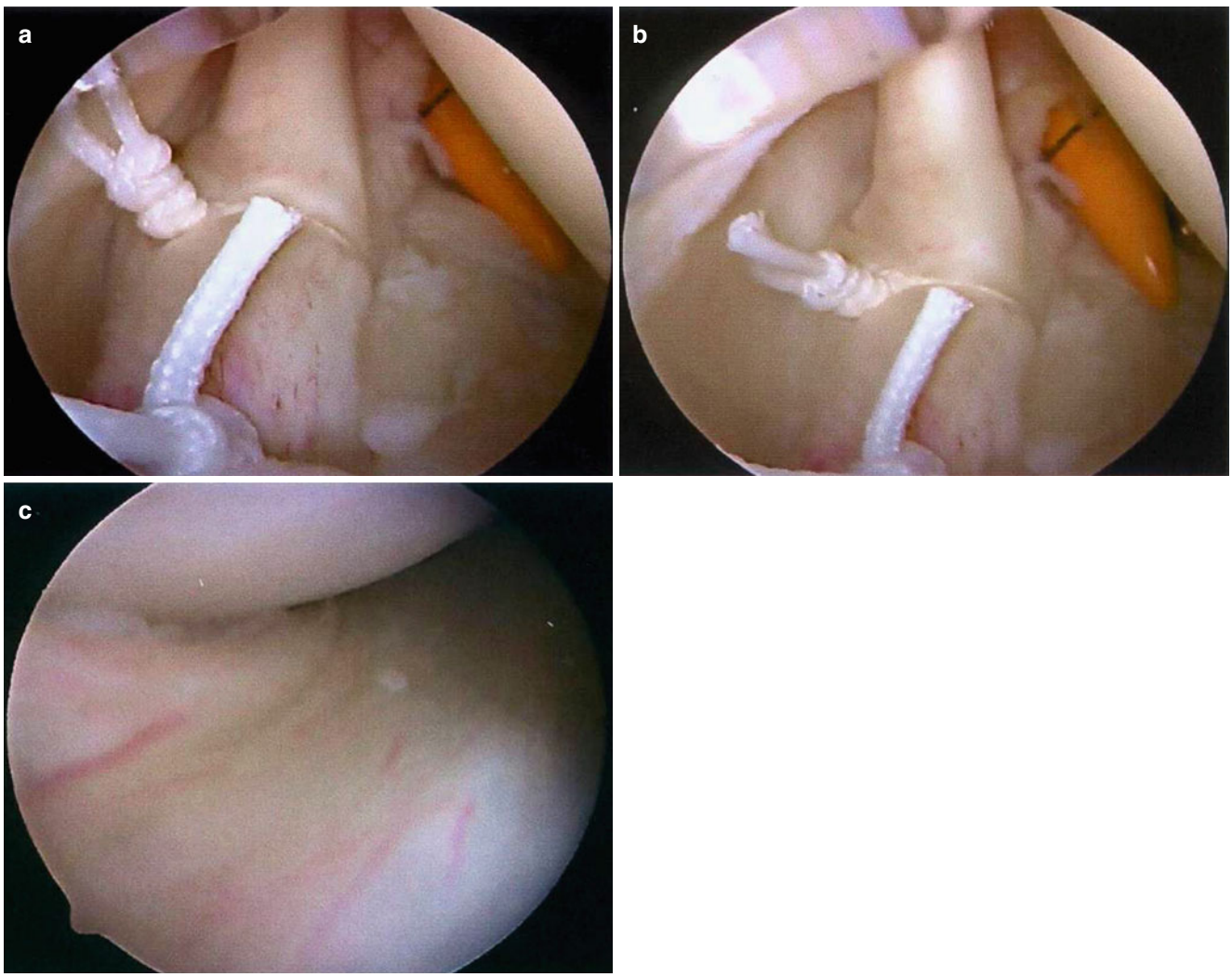

Fig. 19.7 Anchor placement for elimination of peel back. (**a**) Multiple point fixation of the superior labrum to the glenoid. The anchors are placed at approximately the 10:30 and 11:30 positions on the glenoid, and the suture knots are placed posterior to the labrum to reduce suture impingement. (**b**) The arm is moved into abduction and external rotation. Notice that the biceps root is not tethered and can move without impairment. Also note that the peel-back is negative. (**c**) The tension in the posterior band and capsule has been restored (as compared to figure 19.5)

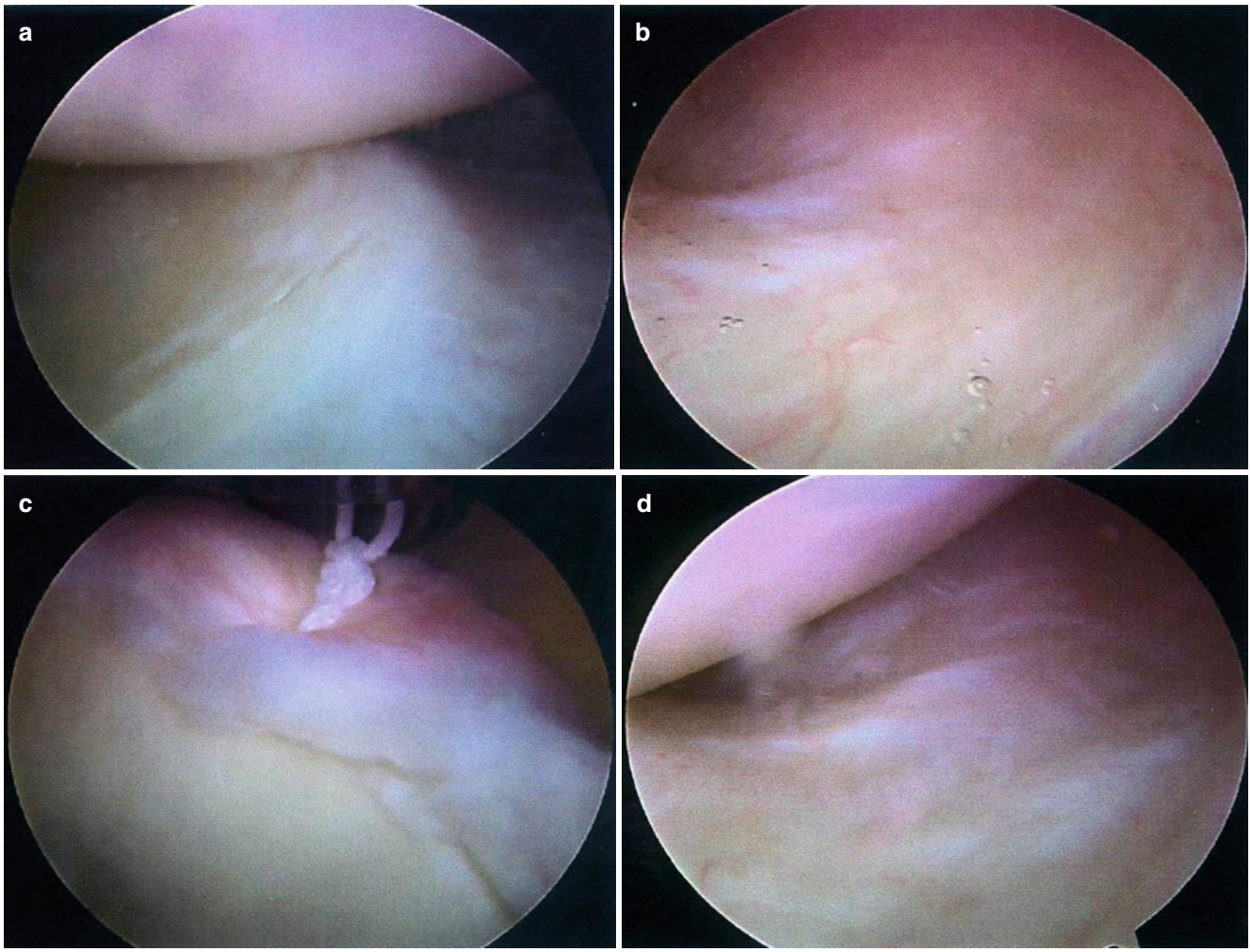

Fig. 19.8 Improving posterior capsular tension through fixation of insubstance of injury. (**a**) The superior labral tear has extended into the substance of the posterior labrum, probably as an extension of the peel-back. (**b**) The P-IGHL and capsule are lax with no evidence of a band (**c**) View from the anterior superior portal. The insubstance lesion has been repaired by a suture anchor and suture (**d**) The P-IGHL shows a definite band and the capsule demonstrates tension

7. Rare placement of anchors and sutures in the anterior superior glenoid (12:00–2:30 on the right shoulder) to reduce the chance of biceps tethering
8. Evaluate the effect of the labral repair on capsular tension by evaluation in the P-IGHL tautness and elimination of the drive-through (Fig. 19.7c)
9. Assess total GH rotation to ensure no external rotation has been lost
10. Treatment of the associated pathology in the joint

Rotator Cuff Tears

The surgical treatment of rotator cuff tears in the throwing athlete has received more attention, but again most studies are low-level evidence [82, 97–103].

The rotator cuff injuries associated with DTS are almost always partial undersurface injuries. The exact etiology is not known, with suggested pathophysiology ranging from a "hypertwist" of the rotator cuff [39] to chronic tensile loads and "microtears" [104] to excessive horizontal abduction and increased contact pressure [104, 105]. The injury is usually not at the actual tendon attachment to the footprint, but starts 2–5 mm off the attachment site (Fig. 19.9). There is usually a good sized amount of tendon still attached to bone. This suggests that the injury may be more frequently associated with the proposed hyper horizontal abduction/compression model of causation. The arthroscopic appearance may include superficial fraying of the tendon, deeper involvement of the tendon, delamination and horizontal extension within the tendon, or complete detachment. There is likewise no consensus on the importance of each of these types of injuries. Some baseball team physicians feel that a certain amount of undersurface injury is an inevitable result of the need to maximize shoulder horizontal abduction and humeral external rotation and is a "normal" finding in high level athletes [106]. These "injuries" would more likely be the superficial fraying seen in the infraspinatus several millimeters off the attachment site [106]. These should be treated with superficial debridement. There is a

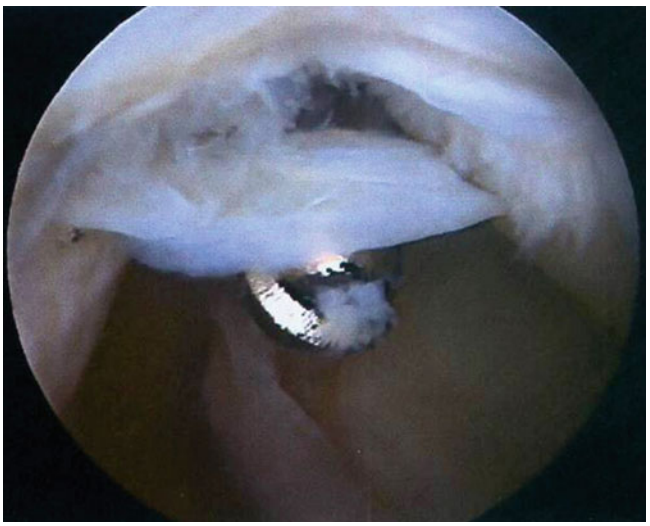

Fig. 19.9 The partial undersurface rotator cuff tear demonstrates full attachment to the humeral head, insubstance tear and delamination to the muscle-tendon junction

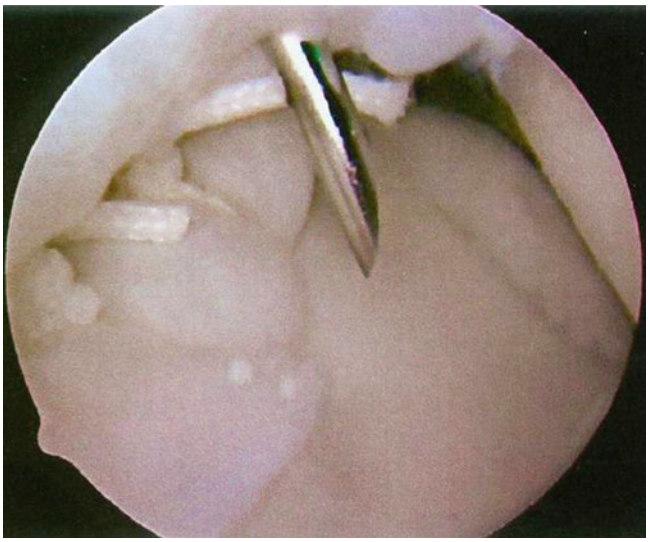

Fig. 19.10 The partial tear can be repaired by multiple sutures that close the delamination. Many different devices can be used to pass and retrieve the sutures

consensus that the partial rotator cuff tears that are delaminated, extend horizontally in the tendon, and involve the supraspinatus and infraspinatus should be considered for repair [46, 98, 107]. The exact method of repair for the partial-thickness injuries is still debatable. Various techniques include insubstance trans-tendon repair [46, 106, 108], trans-tendon reattachment to bone using bone anchors [108, 109], and full-thickness completion of the tear and formal repair [103]. Anecdotal observation and limited clinical studies suggest that cuff repair to bone can be associated with post-op limitation of range of motion [98, 110]. Since any treatment that restricts capability of achieving maximum range of motion is detrimental to optimum overhead throwing or serving performance, most guidelines recommend trans-tendon suture repair rather than tendon takedown and reattachment to bone [46, 106, 108].

Guidelines for the trans-tendon repair include:

1. Establishment of the tear pattern and provisional reduction of the tear
2. Light debridement of the tear surfaces and the bony footprint
3. Establishment of a superior portal to pass the sutures
4. Reduction of the tear
5. Use of a trans-tendon passing device to receive a suture delivered from an anterior portal (Fig. 19.10)
6. Passage of both limbs of a suture to create a mattress stitch (Fig. 19.11)
7. Tying in the subacromial/subdeltoid space

Multiple sutures are usually needed to complete the repair.

Formal rotator cuff repair with anchors back to bone is advocated for high-grade (greater than 75 % of footprint detachment) partial tears or complete full-thickness tears.

The rotator cuff surgery is usually performed after the intra-articular surgery. If a Wilmington type anterolateral portal has been used for the SLAP repair, it can be established through the rotator cuff lesion and can be used for suture passage and repair.

Biceps Injuries

Biceps injuries associated with the DTS do occur, but their incidence is not known. Biceps release with either tenotomy or tenodesis has been advocated in the treatment of DTS, sometimes as a primary treatment [111]. Others have advocated a more specific approach, doing a tenotomy or tenodesis in cases where biceps pathology can be clearly identified, such as biceps generated pain, biceps subluxation, or intra-articular/intra-biceps groove tendinopathy. This relates to the fact that the dysfunction in DTS is not always or completely related to loss of biceps stability or attachment through the labrum, and those other labral roles in the maintenance of functional joint stability should be addressed, even in the face of biceps pathology. It has been demonstrated that a simulated SLAP tear significantly increases GH translation that biceps tenodesis does not restore the normal translation, but posterior labral repair does restore the translation [112, 113].

If biceps surgery is indicated, biceps tenodesis is recommended in younger patients to preserve maximal elbow flexion strength. Multiple techniques appear to produce similar

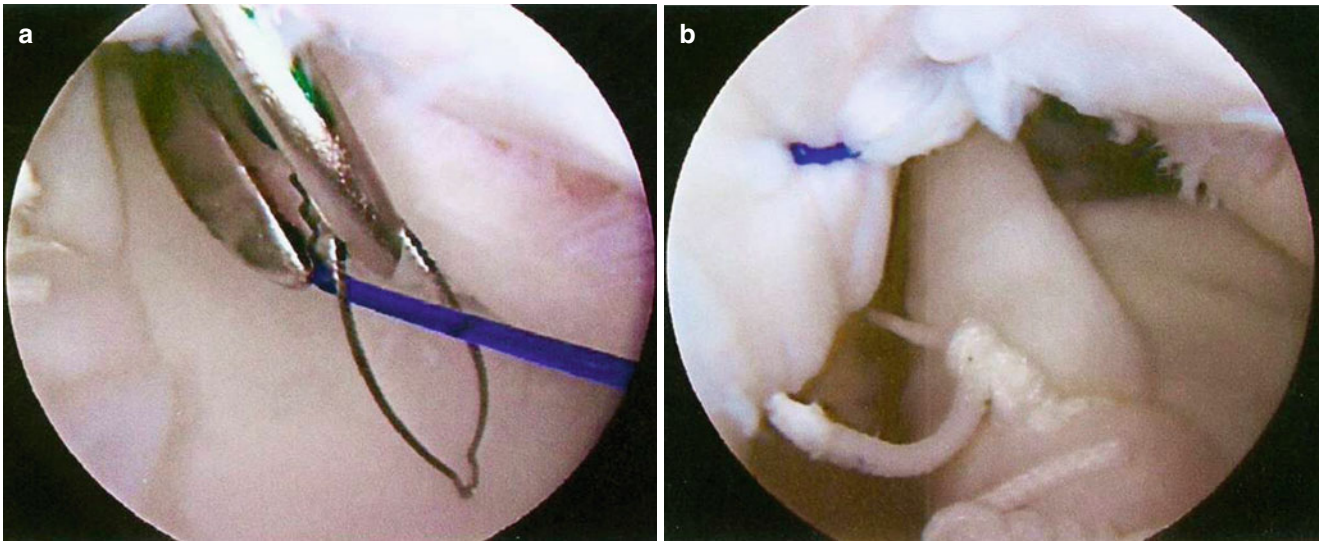

Fig. 19.11 Technique for suture passing. (**a**) Both limbs of an absorbable suture can be passed from the inside of the joint to the subdeltoid space where they can be tied. (**b**) Reduction of the delamination tear by multiple sutures

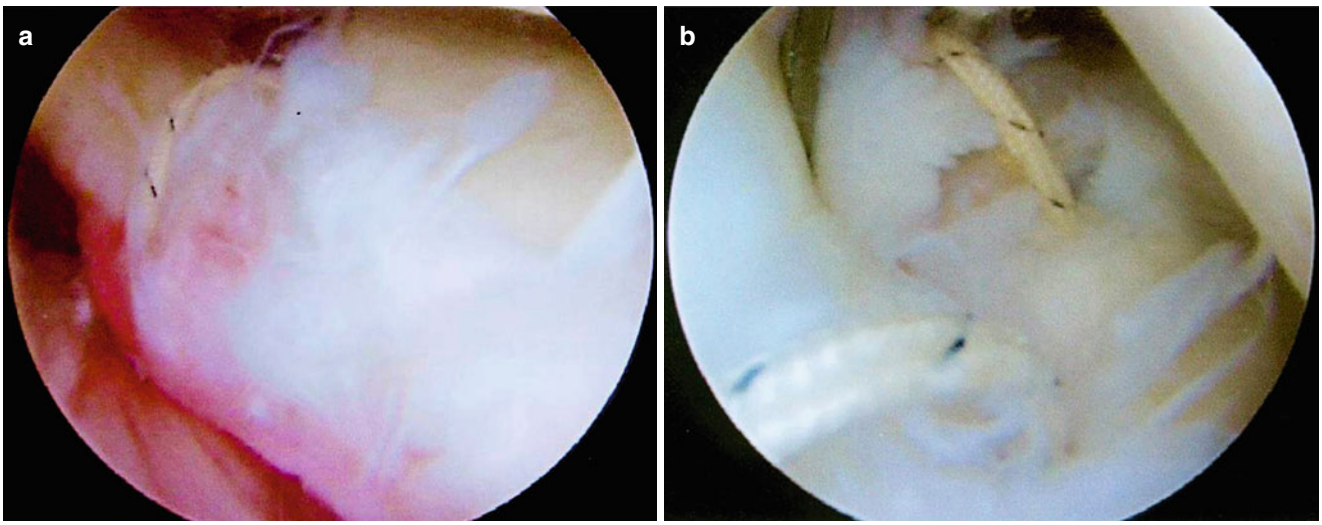

Fig. 19.12 Misplaced anchors creating a tethered biceps tendon. (**a**) A stiff suture passed around the biceps root, tethering the biceps motion and eventually loosening the knot. (**b**) An anteriorly placed knot resulted in tethering of external rotation, eventually resulting in further tissue injury and loosening of the knot

results, including arthroscopic soft tissue tenodesis at the rotator interval [114] and arthroscopic or open suprapectoral or subpectoral tenodesis to bone using anchors or fixation screws [115, 116].

The "Failed" Surgery in DTS Patients

When the surgical treatment fails in DTS patients, multiple areas of possible causative factors can be investigated. The first point is: Was the correct diagnosis made? Was there a true SLAP injury or just an incidental MRI finding? A detailed history and review of clinical exam and arthroscopic findings can be beneficial. Were other diagnoses such as biceps tendinopathy, anterior/inferior instability, or rotator cuff disease not identified and treated? These should be evident on the clinical exam or repeated imaging.

The second point is: Was the correct surgical technique employed? Technical missteps include placement of a 12:00–1:00 anchor with tethering of the biceps (Fig. 19.12), inadequate number of anchors so that the labrum is not stabilized (Fig. 19.13), inadequate preparation of the bony edge to

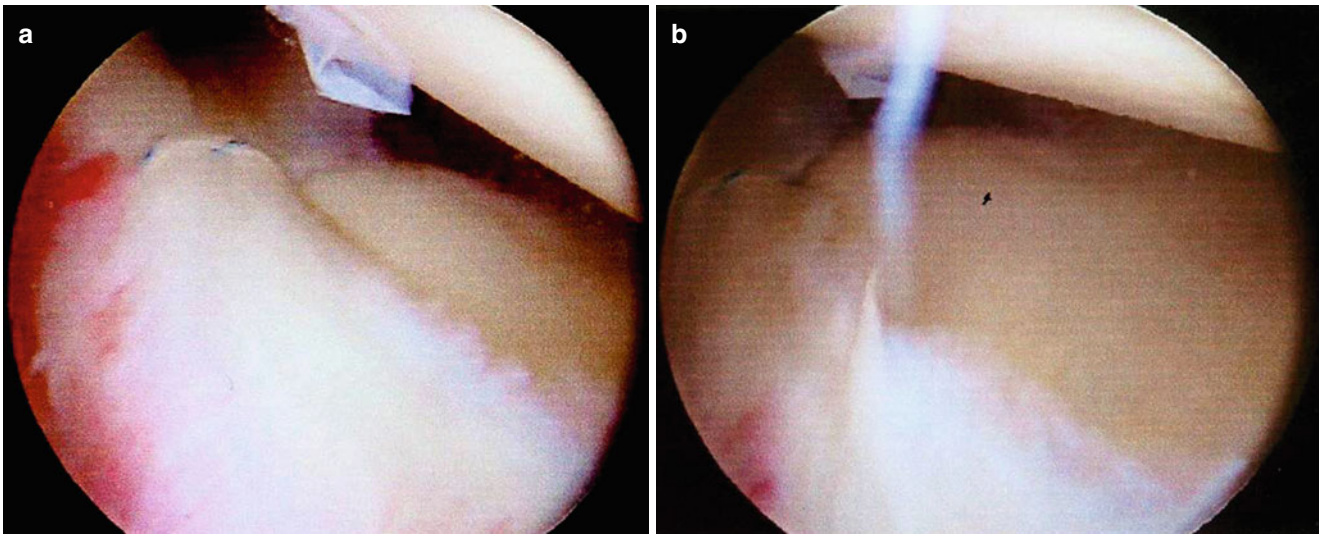

Fig. 19.13 Inadequate number of anchors during labral fixation. (**a**) Single point fixation at 11:45 close to the biceps root. (**b**) The single point fixation did not eliminate the peel back as the arm is moved into abduction and external rotation

allow a good healing response, poor knot-tying technique, failure to address posterior inferior or anterior inferior capsular laxity, failure to stabilize the labrum in the face of a biceps release (Fig. 19.14), and over-constraining the rotator cuff by complete repair to bone.

Revision surgery should reevaluate the joint anatomy, release tight or over-constrained structures (Fig. 19.15), adequately prepare the bony bed, and address the pathology on the principles already suggested. If the biceps has become secondarily injured as a result of the failed surgery, release/tenotomy/tenodesis should be performed as part of the treatment.

The third point is: Was the correct rehabilitation protocol instituted? Rehabilitation must be specific and address deficits in all the areas that may be contributory to the DTS, including shoulder rotation deficits, rotator cuff strength and balance, scapular motion control, and kinetic chain function [45, 69–71]. Review of the protocols the patient used can identify if these areas were addressed in the proper sequence and with the proper exercises. The clinical exam can demonstrate any continuing deficits that may need to be addressed, either as corrective interventions or preparatory for further surgery.

Postoperative Care

Rehabilitation following surgery in the DTS patient is essential to restore all the physiological and biomechanical factors that can be altered to produce the DTS dysfunction. The body works as a unit and frequently fails as a unit. The initial exam and the post operative exams should be comprehensive enough to evaluate all of the factors and should monitor the progression in restoration of the factors.

The rehabilitation program should be progressive through specific stages and should be inclusive enough to address the most common deficits seen.

The exercise program is a progressive and sequential in order divided into three phases based on the level of disability and tissue irritability that exists. Movement through the phases is variable and based on achieving functional capabilities rather than adhering to specific time frames. Phase 1, the acute phase, should minimize loads on the injured tissues, so it should focus on scapular and glenohumeral muscular activation particularly in correcting timing of muscular activation to assure they are working synchronously. Phase 2, the recovery phase, should focus on strengthening and restoring core, kinetic chain, and progressive isotonic strengthening. Phase 3, the functional phase, should focus on sport specific actions, and include endurance and ballistic exercises. Strengthening exercises are oriented towards endurance emphasis with higher repetitions and lower resistance. An athlete should demonstrate the ability to perform 3–4 sets of 15–20 repetitions with correct form prior to progressing to a greater resistance.

Acute Phase

Patient education is the first goal to inform the patient of precautions to protect the surgical repair and to understand the procedure that were performed. Sharing the surgical report with the treating physical therapist or athletic trainer benefits the patient as this helps put all of the rehabilitation

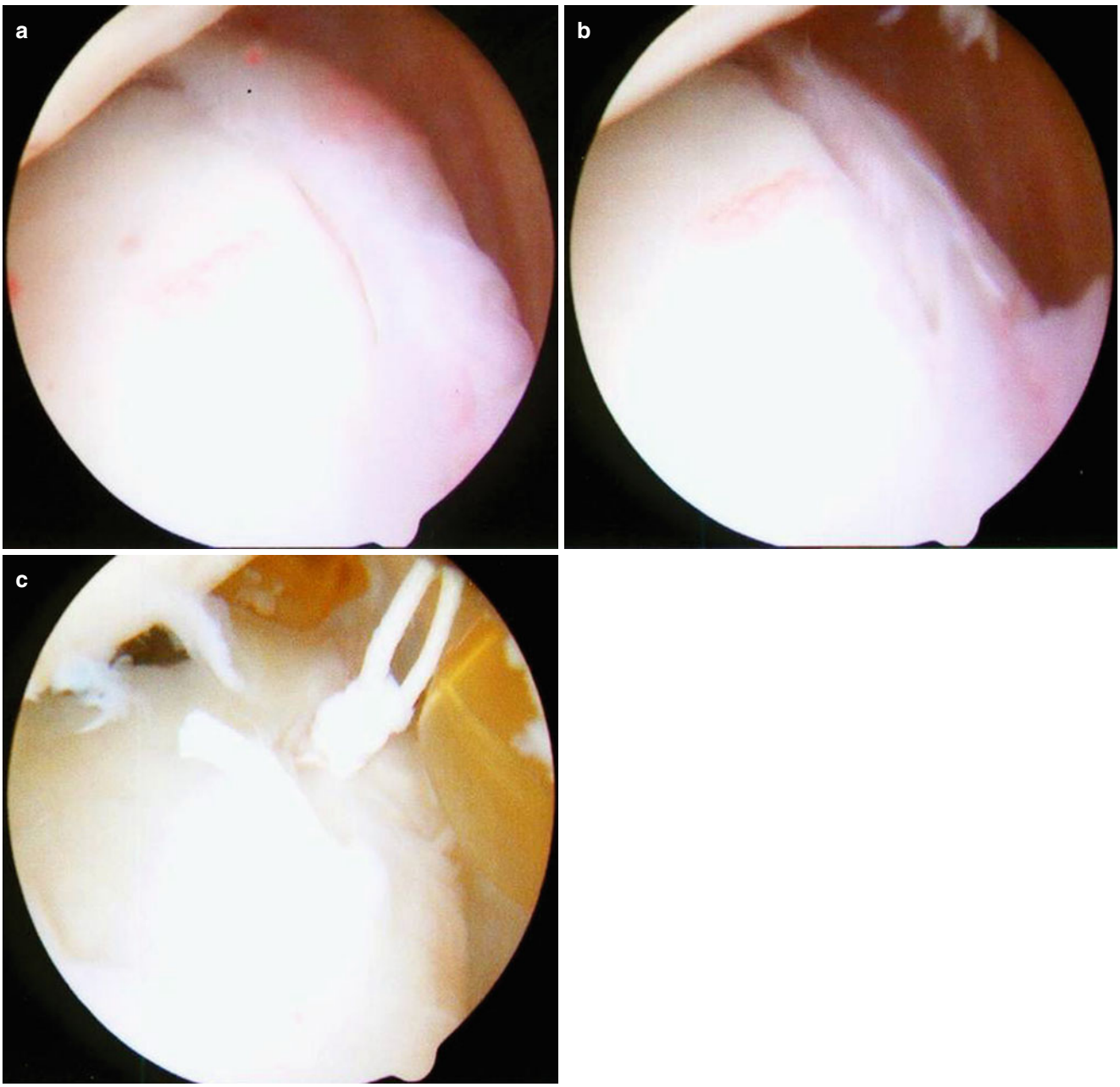

Fig. 19.14 Failure to stabilize the labrum during concurrent biceps release. (**a**) Superior labral tear treated primarily with a biceps release and tenodesis. The patient's symptoms did not improve. (**b**) The peel back is still positive as the arm is moved into abduction and external rotation. (**c**) The labrum is stabilized by 12:00 and 1:00 anchors with a negative peel back

team on the same page. As part of education, integrating key points of protecting the healing repaired tissue is paramount. Proper positioning of the arm and shoulder are important for minimizing pain and protection. It is important that all members of the rehabilitation team are clear and consistent in instructing the patient regarding what activities are allowed and prohibited during this phase. The arm is typically immobilized for a time period; it is often appropriate to start working on the rest of the kinetic chain to address deficits.

Nearly 50 % of ball velocity comes from leg forward step and trunk rotation [117]. Addressing deficits found in the trunk and lower extremity is important as the ability to accelerate the elbow and wrist during throwing is due to torque generated more proximally in the trunk. Throwing performance may also benefit by targeting the core musculature, as increased pelvic velocity during arm cocking phase and upper torso rotation during the acceleration phase of throwing is positively related to ball velocity [118].

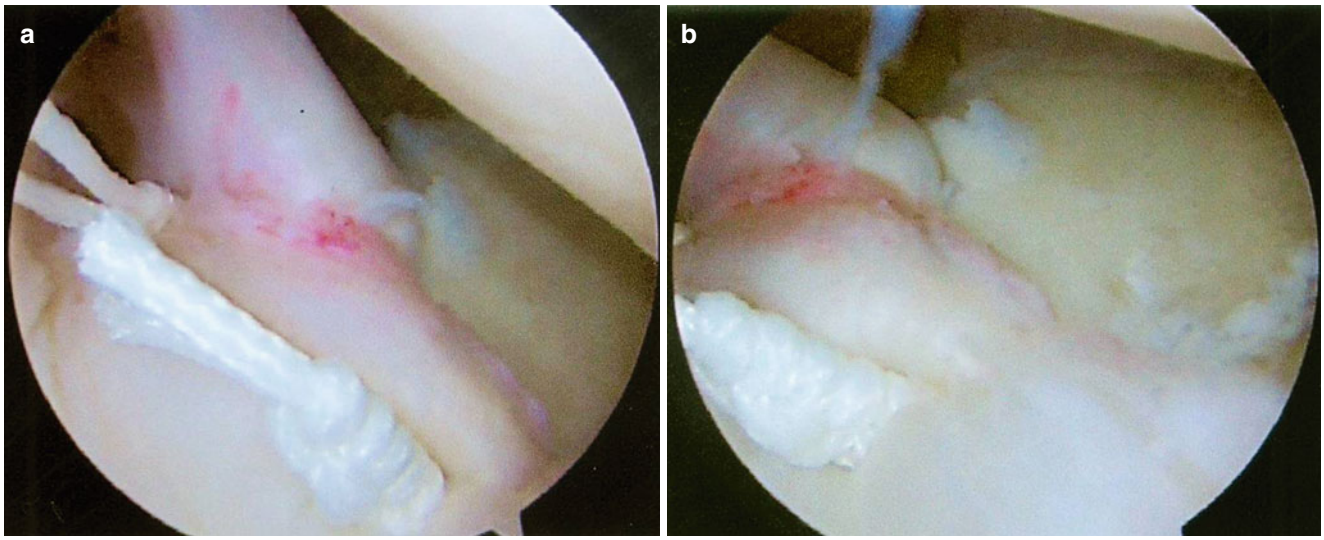

Fig. 19.15 Release of over-constrained structures. (**a**) Same patient as figure 19.12. The constraining sutures have been removed and the superior labrum has been stabilized by 10:30 and 11:30 anchors. (**b**) The peel back is negative and the biceps is not tethered as the arm is moved into abduction and external rotation

Limitations of motion of the spine, hip, and the lower extremity should be addressed early in the rehabilitation protocol. These exercises can be started while the injured area is still being protected.

Reestablishment of range of motion follows a typical pattern of passive to active assistive exercises before starting resistive exercises. The guiding principle is to protect repair so limited motions are often recommended based on the tissue repaired. Gradually increasing the motion in elevation and rotation directions has been previously recommended in the literature and provides good general guidelines, but each physician may have specific parameters on individual patients. The primary principle that drives reestablishment of motion during this phase is regaining scapular control and gradually increases muscular demands of surrounding shoulder girdle musculature. Initial management should focus on scapular musculature control, which facilitates stability in order to prepare the shoulder musculature for more dynamic and stressful exercises. Repeated emphasis on proper position and movement instructions are important as many of the movement strategies learned are only temporary. Scapular retraction and depression exercises, such as the lawn mower exercise, can be performed during the initial protection period (i.e., while wearing a sling) as these maneuvers integrate the kinetic chain and scapular musculature which is safe for most all patients. Passive and active assistive range of motion is facilitated by supporting the arm on a table top or ball which has two benefits: by unloading the arm weight so the muscles are not significantly challenged and the proper movement pattern can be facilitated to minimize the chance of substitutions.

Recovery Phase

The focus of this phase is to completely reestablish shoulder range of motion and begin developing strength and endurance of upper extremity and the entire kinetic chain. Incorporation of leg exercises into the comprehensive rehabilitation program is advantageous at this phase as it gets an early start on common limitations in athletes with DTS. Critical areas are hip inflexibility and weakness in the core.

There is no one exercise that targets all the abdominal muscles; therefore a program that addresses anterior, lateral, and posterior sides of the torso should be undertaken. Mat exercises emphasizing endurance are good starting points, with consideration given to positions that limit stress to the shoulder. However, the athletic demands require engagement of the lower extremity into core exercises. Exercises such as rotational chop and lift activities that simulate the demands of throwing are recommended [119]. The use of unstable surfaces, such as stability balls or foam mats, have demonstrated increased core activation [120, 121] and have the potential to improve core strength.

Individuals with DTS often present with shoulder range of motion (ROM) deficits [122, 123]. Deficits in GH internal rotation (GIR) and total rotational range of motion (TROM) can be predictive of future injury [124, 125]. The goal of the rehabilitation program is restoration of ROM deficits to acceptable values [124, 126–128]. The healthy throwing athlete exhibits increased external rotation and concurrent loss of internal rotation on the throwing side, yet retains a total arc of motion of approximately 180° which is relatively equal to the opposite side [124, 125, 128–130]. Reestablishing internal rotation of the throwing shoulder should approxi-

mate 18° (range 13–20°) of the non-throwing shoulder [124, 125]. TROM in the throwing shoulder should be within 5° of the nondominant shoulder, and TROM should not exceed 186° to avoid an increased risk of injury [125]. The cross-body stretch and sleeper stretch target the posterior shoulder musculature and capsule to effectively improve GIR and glenohumeral horizontal adduction (GHA) and can be introduced as tolerated by the recovering shoulder [130, 131]. Additional beneficial treatment techniques include joint mobilizations [132] muscle energy techniques [133] and soft tissue mobilization to facilitate full recovery is appropriate at this phase if motion is not returning as the expected pace for these patients [134].

Addressing muscular deficits and imbalances is the second goal of this phase. The acute phase initiates scapular muscular control while in this phase exercises can be progressed to dynamic short lever arm activities to progress proximal functional control. Overactivation of the upper trapezius during elevation which indicates a muscular imbalance around the scapular force couple has been found in injured shoulders [32, 135] and often indicates the exercise is placing greater demands on the arm than the patient is ready for. Facilitating scapular and rotator cuff musculature should use short lever arm exercises such as side lying external rotation with arm below shoulder level before progressing to overhead strengthening activities [136]. Establishment of proximal stability should precede longer lever arm activities (i.e., prone horizontal abduction exercises) in order to establish proximal functional control without substitution.

Deficits in shoulder strength are predictive of future injury [8]. Addressing these deficits during the recovery phases is critical to allow athletes to return to full level of function. There is often a rush to return to throwing activities, without establishing full arm, core, and lower extremity strength, flexibility, and endurance; the postsurgical athlete is being set up for a failed intervention. Using established value for range of motion [124, 126, 128] and strength [137–139] should be accomplished before considering sport specific activities. Athletes presenting with shoulder symptoms are typically found to have strength deficits of the rotator cuff [140, 141] and scapular musculature [3] which may present with various movement patterns [142]. Treatment of these deficits have many common themes and treatment protocols [143].

Functional Phase

Progression to dynamic exercises that incorporate all shoulder muscles and simulates function is the next stage of rehabilitation. Exercises in this phase are frequently individualized for specific goals, and direct supervision by a rehabilitation specialist is critical to meet the individuals' needs and anticipated functional levels.

The final stage of shoulder strengthening includes plyometrics. These dynamic exercises develop maximal power capability and should be integrated with kinetic chain plyometric exercises.

Return to Play

The decision for return to play should be based on restoration of all components necessary to achieve function. It includes optimal fixation of the pathologic anatomy; resolution of the flexibility, strength, and strength balance deficits; and demonstration of normal mechanics of kinetic chain and arm motion. Functional progressions such as the return to throwing program [69, 144] can then be instituted. Advancement through the functional progressions should not be rushed as the reprogramming of the motor patterns, the normalization of muscle flexibility and joint motion, the restoration of muscle strength and power, and the reacquisition of the fine motor skills required for each pitch take a long time. Non-pitchers can usually progress more rapidly through these phases.

Summary

The term DTS best describes the dysfunctional reality of the overhead athletes. Because it involves alteration in anatomy, physiology, and mechanics, a thorough knowledge of normal kinetic chain mechanics is required as a baseline. Evaluation of the DTS patient involves a comprehensive exam. Treatment includes a significant rehabilitation component which can result in improvement in a number of patients. Surgical treatment should be based on specific indications, have specific objectives, and involve specific techniques. In the case of failed operative treatment, an in-depth investigation of the multiple causative factors must be done. Specific rehabilitation programs focused on restoration of all of the altered factors is required to produce optimal results.

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SLAP Lesions

20

John M. Tokish and Richard K.N. Ryu

Epidemiology

Lesions of the superior labrum remain a diagnostic and therapeutic challenge for the arthroscopic surgeon. Given the variability of the anatomy in this region, its controversial role in shoulder biomechanics, and the natural history of fraying of the aging glenoid labrum, few diagnoses generate as much controversy in terms of the decision and method of treatment as the "SLAP" tear.

Andrews et al. [1] were the first to describe these tears in conjunction with partial-thickness rotator cuff tears in a population of pitchers in 1985. Later that year, the same authors [2] reported specifically on tears of the superior labrum in 73 pitchers. In 1990, Snyder et al. [3] described a series of superior labral anterior to posterior lesions, as a subset of arthroscopic shoulder surgeries, and coined the term "SLAP tear." Since these publications, lesions of the superior labrum have become increasingly recognized as a source of shoulder pain, and treatment of these lesions has become a common, and some might suggest overtreated, surgical intervention. In Snyder's original description, the authors reported on 27 SLAP tears in over 700 arthroscopic shoulder cases, for an incidence of less than 4 %. Other publications have shown a significant increase since that time. Onyekwelu et al. [4] reported that the incidence of SLAP repairs in New York state rose by a factor of 5.5 between 2002 and 2010. A similar increase has occurred in the US Active Duty Department of Defense population, where some 6000 SLAP repairs were performed between 2004 and 2008, making it the fourth most common shoulder surgery in that population [5]. Finally, Weber et al. [6] recently reported on trends among candidates for part II certification by the American Board of Orthopaedic Surgery. The authors noted a concerning "disproportionate enthusiasm" for SLAP tears, noting that recent candidates performed SLAP repairs at three times the published incidence supported in the current literature, including patients into their 9th decade of life. The reported incidence rates from the literature are included in Table 20.1.

The reasons for this increasing incidence are multifactorial. Vangsness et al. [14] studied the normal anatomy of the biceps origin. The authors noted four different types of insertions and highlighted the difficulty of defining what represented a labral lesion versus a normal variant. Definitive physical examination findings have also proven illusive, with no single test demonstrating simultaneous sensitivity and specificity [15]. While MRI and MRA have become the imaging modality of choice, several authors have questioned its accuracy and noted the potential for a high false-positive rate [16–20]. Even diagnostic arthroscopy, long been considered the gold standard of this diagnosis, has demonstrated substantial interobserver and intraobserver variability in agreeing on the diagnosis and treatment of SLAP lesions [21]. Thus, the definitive diagnosis and proper treatment of the SLAP lesion remains a controversial area of shoulder surgery. This chapter will attempt to guide the reader toward a comprehensive understanding of this condition with the best literature available along with pearls and pitfalls gained along the way in the treatment of over 500 SLAP tears.

Pathophysiology

The anatomy of the biceps-labral complex is variable. The tendon, along with the medial attachment of the superior labrum, forms a subsynovial recess which extends for several millimeters from the edge of the superior glenoid [20, 22]. Habermeyer et al. [23] reported the tendon originated from the posterior labrum 48 % of the time, while 20 % originated from the supraglenoid tubercle and 28 %

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Table 20.1 Reported incidence of surgically treated SLAP lesions in the orthopedic literature

Authors	Incidence/prevalence of surgical SLAPs	Noteworthy
Snyder (1990) [3]	3.3 %	First report of SLAP lesion incidence
Snyder (1995) [79]	4.7 %	F/u of 140 SLAPs
Maffet (1995) [28]	11.8 %	Highest rate in the literature, included significant number of patients with instability in combination with SLAP
Kim (2003) [35]	6.6 %	139 SLAP cases
Onyekwelu (2012) [4]	5.5-fold increase	Numbers reported as percentage of total number of surgeries done
Zhang (2012) [61]	1.65-fold increase	Numbers reported as percentage of total surgeries 2004 vs. 2009
Weber (2012) [6]	9.4 %	Represents a threefold increase compared to literature

Table 20.2 SLAP tear classification

Type	Clock face description	Description	Authors
I	11 to 1	Fraying of superior labrum	Snyder (1990) [3]
II	11 to 1	Detachment of biceps anchor	Snyder (1990) [3]
III	11 to 1	Bucket handle tear with intact biceps	Snyder (1990) [3]
IV	11 to 1	Bucket handle tear with biceps extension	Snyder (1990) [3]
V	11 to 5	Bankart plus SLAP	Maffet (1995) [28]
VI	11 to 1	Flap tear of bucket handle SLAP	Maffet (1995) [28]
VII	11 to 3	SLAP extension into middle glenohumeral ligament	Maffet (1995) [28]
VIII	11 to 7	SLAP with posterior extension	Mohana-Borges (2003) [30], Nord/Ryu (2004) [31]
IX	11 to 11	Circumferential (pan-labral) lesion	Powell (2004) [32], Nord/Ryu (2004) [31]
X	11 to 1+	SLAP with noncontiguous posterior labral tear	Beltran (1997) [29], Nord/Ryu (2004) [31]

from both locations. Vangsness et al. [14] confirmed this variability in a cadaveric study, noting that 50 % of the tendons originated from the superior glenoid labrum with the remaining tendons attached to the supraglenoid tubercle. Other studies have noted that the majority of shoulders that have biceps insertion into the labrum have an entirely posterior or posterior-dominant labral insertion, but the attachment is variable and may be posterior, posterior-dominant, or equally anterior and posterior into the superior labrum [20]. Thus, one must be careful about making the diagnosis of a pathological biceps lesion solely on its position of insertion. Sublabral foramen, posterior attachments, and variable anatomy are more commonly the rule than the exception.

This combination of variable anatomy and the multitude of deforming forces seen in the athletic shoulder may result in a number of different injury patterns to the superior labrum. Snyder et al. [3, 24] originally classified SLAP tears it into four types. Type I lesions have an intact biceps origin with labral fraying. Type II lesions have a detached biceps-superior labrum origin. Type III lesions have an intact biceps anchor with a bucket handle tear of the superior labrum. Type IV lesions have a bucket handle tear of the labrum with the tear extending into the biceps tendon. Over time, other authors have expanded this classification to its current inclusion of 10 different types (Table 20.2) [3, 28–32].

The described mechanisms of injury for the SLAP tear are almost as numerous as the types themselves. In the first description of these tears, Andrews et al. [2] hypothesized that the eccentric load to the biceps during deceleration of the pitch was responsible for a repetitive traction injury to the biceps root. Snyder et al. [3] originally noted the most common mechanism of injury in their patients to be a fall on the outstretched arm and believed a combination of a compression force to the superior joint surface and a proximal subluxation force on the humeral head was the mechanism of action. Burkhart et al. [33] presented a "peel-back" mechanism as a torsional force in the abducted externally rotated arm to be a SLAP generator, and these same authors [34] later published that a primary posterior-inferior capsular contracture leads to a posterior-superior shift of the humeral head, culminating in a shear force at the posterior-superior labral attachment resulting in a posterior-superior type II SLAP injury. Thus, tensile, compressive, torsional, and shear forces have all been implicated as the generator of the SLAP lesion, making a simple or unifying mechanism unlikely for this injury.

History

There is great variability in the presentation of patients with SLAP lesions. Younger athletes often present after sustaining a traumatic injury whereas older ones commonly have more of an insidious onset. Pain is often the reason for initial presentation, but the location and pattern can be variable, mimicking other

types of shoulder pathology [20, 35, 36]. In Snyder's original series [3], the most common patient complaints were pain along with "popping" and "catching" in the shoulder. Patients may complain of a painful, deep click in the shoulder or of a sensation of "giving way" with overhead or other rotational movements of the shoulder. Throwers may complain of loss of velocity and control with pain occurring in the late cocking and early acceleration phases. Older athletes may complain of a sensation of weakness from associated pain which may be caused by associated rotator cuff pathology or secondary subacromial impingement. Complaints of instability more commonly come from younger athletes and may be associated with a history of traumatic shoulder dislocation, potentially obscuring the possible contribution of a concomitant superior labral injury. In a study looking at 30 patients with a mean age of 48 years who presented with labral or SLAP lesions, 24 had the onset of acute symptoms and 6 had insidious onset. Eleven patients' injuries were sports related and 11 occurred from falls. The predominant complaint of all patients was pain [37].

A thorough history should be taken to determine if the pain originated from athletic participation or other traumatic event and if the pain worsened with sports activity. Athletes should be asked whether the pain diminishes their desired performance level. Complaints of popping, clicking, or grinding may be indicative of a symptomatic SLAP lesion. If athletes complain of a sensation of snapping with certain shoulder motions, the problem may be tendon instability at the bicipital groove, an uncommon finding in this population. Patients with biceps tendon pathology often complain of pain in the anterior region of the shoulder over the bicipital groove, but this can be highly variable dependent upon whether associated pathology in the shoulder exists.

Clinical Examination

The physical examination in the patient with the suspected SLAP tear can be very confusing due to the presence of commonly associated injuries. Nevertheless, a thorough evaluation can be a key in understanding which findings are adaptive, versus those that are pathologic. Physical examination should include careful inspection for shoulder asymmetry, muscle atrophy, or for signs of direct trauma. The scapula should be evaluated for rhythm, and any signs of winging should be noted, as correction of such dyskinesis is paramount to an optimal outcome. The shoulder should be palpated for focal tenderness. Active and passive range of motion (ROM) should be measured and compared with the contralateral shoulder. Overhead athletes should be checked for internal and external rotation with the shoulder abducted 90°. Throwers often have increased external and decreased internal rotation of the dominant shoulder, but if the internal rotation side-to-side difference is greater than 25°, the athletes may have glenohumeral internal rotation deficit (GIRD) predisposing them to SLAP tears and internal impingement [38–40]. Total motion arc measurements, comparing the affected to the contralateral shoulder with side-to-side differences of greater than 5° or 10°, can also indicate an internal rotation deficit and the potential for associated rotator cuff and labral abnormalities.

Rotator cuff musculature to include the supraspinatus, infraspinatus, teres minor, and subscapularis should be evaluated and compared to the contralateral side. The mature athlete will commonly show weakness on examination which may indicate rotator cuff pathology either from subacromial or internal impingement.

Multiple special tests exist for evaluation of SLAP lesions and biceps tendon pathology. Yergason and Speed's testing should be performed to check for biceps tendinopathy. O'Brien's test, anterior apprehension test, compression-relocation test, Whipple test, biceps load tests I and II, Jobe relocation, crank test, pain provocation, internal rotation resistance strength test, passive compression test, anterior slide test, and resisted supination-external rotation tests have all been described to detect SLAP lesions [9, 10, 15, 20, 25, 40–49]. Although most authors report good results with their respective tests, independent researchers comparing examination and intraoperative findings have concluded that clinical findings alone are not reliable in diagnosing SLAP lesions [50, 51] (Table 20.3). The multitude of clinical tests reflects the difficulty in making an accurate diagnosis with a comprehensive physical examination. A preponderance of positive tests rather than any one single test may be the strongest indicator of a symptomatic SLAP lesion [55].

Imaging

Standard imaging of the shoulder suspected of a SLAP tear may include anteroposterior, scapular Y, and axillary lateral X-rays. While these studies can be useful in identifying associated pathology, their ability to evaluate the SLAP tear is limited unless a supraglenoid tubercle avulsion is present. The MRI or MRA has long been the imaging modality of choice in this pathology (Fig. 20.1), and several studies have demonstrated its ability to detect the lesion. Differentiating between the normal variable anatomy of the superior labral complex and the presence of a pathologic SLAP can be difficult, however, and is one of the reasons why the accuracy of MRI evaluation of SLAP tears is constantly challenged [16, 17, 19]. Certain techniques including an abducted externally rotated (ABER) view may help distinguish normal from pathologic. The addition of intra-articular contrast for an MRA allows visualization of fluid extending under

Table 20.3 Reported sensitivities and specificities of various physical tests for SLAP tears

Test	Authors	Sensitivity (%)	Specificity (%)
Active compression test	McFarland (2002) [50]	47	55
	Stetson (2002) [45]	54	31
	Guanche (2003) [55]	63	73
	Nakagawa (2005) [40]	54	60
	Myers (2005) [39]	78	11
	Parentis (2006) [54]	63	50
	Michener (2011) [48]	55	38
	Hawkins (2012) [49]	85	10
	Average	*62*	*41*
Speed's	Guanche (2003) [55]	18	87
	Nakagawa (2005) [40]	4	99
	Parentis (2006) [54]	48	67
	Oh (2008) [15]	32	66
	Hawkins (2012) [49]	50	54
	Average	*30*	*75*
Crank	Stetson (2002) [45]	46	56
	Guanche (2003) [55]	40	73
	Nakagawa (2005) [40]	58	72
	Myers (2005) [39]	35	70
	Parentis (2006) [54]	13	83
	Michener (2011) [48]	91	42
	Average	*47*	*66*
Anterior slide	McFarland (2002) [50]	8	84
	Nakagawa (2005) [40]	5	93
	Parentis (2006) [54]	10	82
	Michener (2011) [48]	5	69
	Average	*7*	*82*

the superior labrum and dissection under the bicep anchor on coronal images and can increase the sensitivity for detecting SLAP injuries [19].

Several studies have evaluated MR technology in the detection of superior labral tears. Applegate et al. [56] showed that MR arthrography had a 100 % sensitivity, 88 % specificity, and 92 % accuracy in diagnosing chronic labral tears. Waldt et al. [57] demonstrated that MR arthrography was 82 % sensitive and 98 % specific 75. Other studies, however, have challenged the accuracy of MR technology in the diagnosis of SLAPs. Phillips et al. [26] recently reported on the accuracy of non-contrast MRI in the detection of SLAP lesions and noted that MRI is a poor predictor of a surgically confirmed SLAP tear. Amin et al. [58] noted a specificity of only 50 %, highlighting the limitations of conventional MR imaging for SLAP lesions, and the potential for overdetection. Perhaps the greater challenge occurs when the SLAP tear is present in the setting of concomitant pathology. The MRI cannot discern which findings are pathologic, and the presence of abnormal MR imaging should not be considered diagnostic of a SLAP lesion. MR technology can be a useful adjunct in the detection of SLAP tears but should not be relied upon as the primary means of diagnosing this pathology.

Treatment: Indications and Contraindications

The indication to surgically address superior labral and biceps anchor or tendon pathology can be challenging, as normal anatomy is variable and sometimes difficult to distinguish from true pathology, and furthermore, asymptomatic superior labral lesions can occur in association with shoulder injuries. The incidence of isolated SLAP lesions ranges from 2 to 30 % [3, 15, 35, 54]. The reported incidence of SLAP lesions associated with other pathology is much higher. In one study of 544 shoulders, 25 % were diagnosed with SLAP lesions, and 88 % of these had coexistent pathology [35]. This coexistent pathology increases with age, and one should be reluctant to make the diagnosis of an isolated SLAP tear over the age of 40. In older patients, it appears that successful outcomes are driven more by successfully addressing these concomitant pathologies rather than the SLAP tear itself. Coleman et al. [11] compared a series of isolated SLAPs with a series of SLAP lesions which were also treated for impingement. The authors found that only 65 % of the isolated SLAP group had good or excellent results and that 21 % of this group eventually developed clinical impingement. Enad and Kurtz [59] demonstrated higher ASES scores in patients treated for SLAPs with

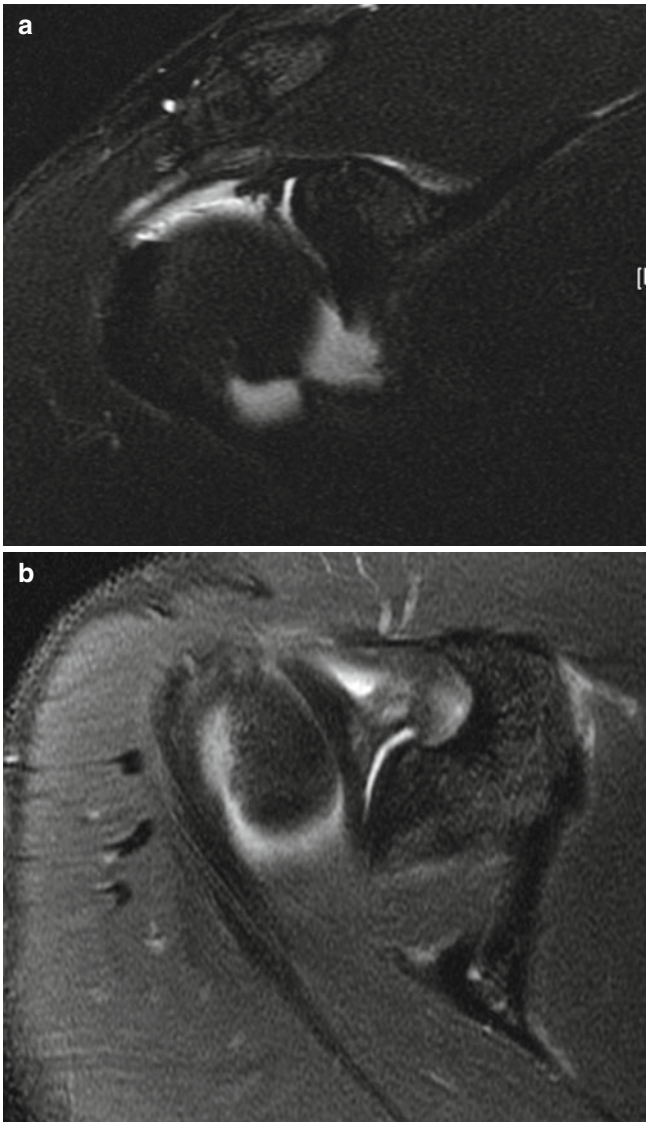

Fig. 20.1 Coronal (**a**) and axial (**b**) T2 MR images of superior labral tear. Note on the axial, the tear extends posterior to the biceps anchor

additional pathology over isolated SLAP repair. There is also controversy in the preferred method of treatment of the SLAP lesion. Repair versus tenotomy or tenodesis is currently a hotly debated topic, and few studies directly compare the various techniques. In the setting of combined pathology, it may be preferable to tenotomize the biceps instead of repairing the SLAP. Franceschi et al. [53] noted higher UCLA scores with tenotomy rather than with SLAP repair, in patients undergoing primary rotator cuff repair. Boileau et al. [60] reported superior outcomes in a group of patients treated with tenodesis or tenotomy compared to those who had a SLAP repair. The groups did demonstrate a significant difference in age, with younger patients undergoing repair, introducing a potential bias to the study. While no level 1 study compares these different approaches, it does appear that the indications for SLAP repair are becoming more narrow and restricted [61].

Decision-Making Algorithm

The final and perhaps most important tool in the decision-making process for a suspected SLAP tear is the diagnostic arthroscopy. There are certainly times when a SLAP tear can be obvious (Fig. 20.2), where displaced labral tissue or raw cancellous bone at the insertion can be visible. Other arthroscopic features consistent with a pathologic SLAP lesion include superior excursion of greater than 5 mm, sublabral granulation tissue, chondral changes, and frayed labral tissue at the base of the biceps attachment. But even experienced arthroscopists can disagree between a normal variant and a pathologic SLAP tear. Gobezie et al. [21] reported on the intraobserver and interobserver variability in SLAP diagnoses among experienced shoulder surgeons. The authors noted that as a whole the group had difficulty distinguishing type II SLAP lesions from normal shoulders as well as in distinguishing type III and IV lesions from one another. Thus, the proper diagnosis of the pathologic tear depends on a proper history, physical examination, appropriate imaging studies, and diagnostic arthroscopic findings consistent with pathologic and confirmatory evidence. Once a solid diagnosis of a SLAP tear is made, decision making must take into account a patient's age, chosen activity, potential for rehabilitation, concern for cosmesis, and associated pathology. The young patient with an isolated type II SLAP is a very different scenario than the 50-year-old laborer with a type II SLAP and a concomitant rotator cuff tear. In the latter case, there may be little or no evidence that the concomitant SLAP lesion is pathologic and contributing to symptoms. Asymptomatic superior labral separation as part of a senescence pattern is well documented [62]. Isolated SLAP lesions are uncommon and, when encountered, may be considered the primary causative factor for shoulder pain and dysfunction, necessitating surgical intervention.

Snyder's original classification carries with it helpful guidelines for most types: Type I tears are not controversial, as there is general agreement that little or no treatment is warranted with a debridement occasionally required. Likewise, there is agreement in the setting of tears with large displaced fragments that do not compromise the biceps insertion that debridement can remove a mechanical flap or nonstructural piece of labrum. Occasionally repair of a type III or IV lesion might be warranted if associated with shoulder instability. Other SLAP tears in the setting of an extended labral instability pattern, such as the types V, VIII, or IX, also engender a consensus to repair, with excellent results in restoring stability as reported in the literature [27, 63, 64].

The type II SLAP, however, remains a controversial topic among arthroscopists. While satisfactory pain relief has been reported in roughly 90 % of patients in many studies [25, 65–68], return to play, and especially throwing, has been more elusive. In systematic reviews done on the topic, return to throwing has been reported between 22 and 64 % [69, 70], reinforcing the concept

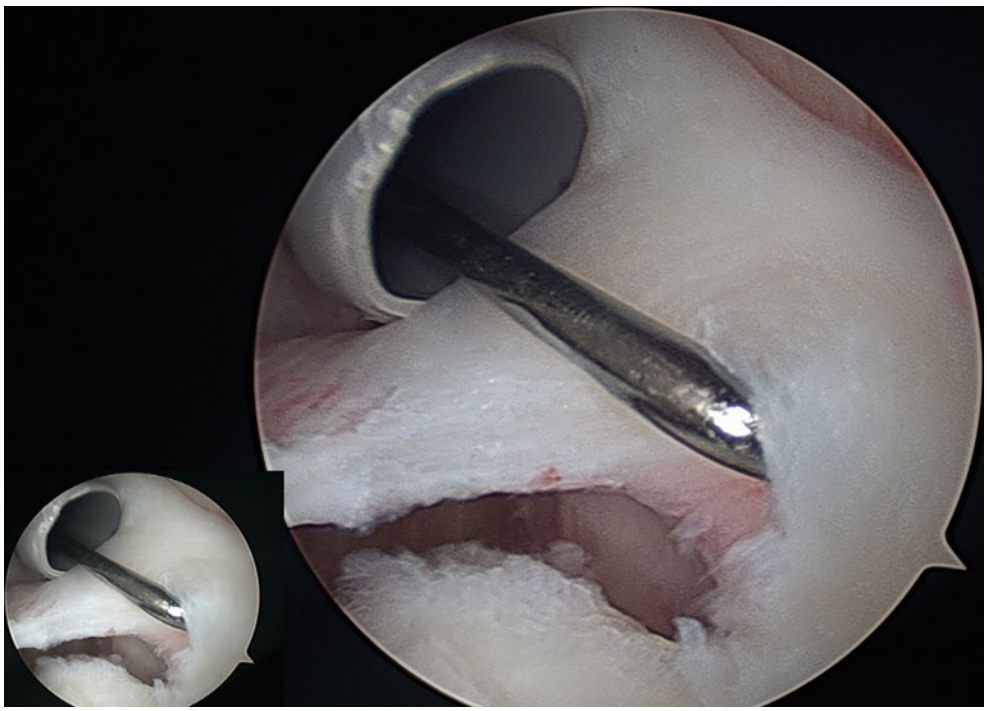

Fig. 20.2 Arthroscopic view of a SLAP II lesion. Note the detachment of the superior labrum with sublabral granulation tissue

that there is still much we do not understand about the painful throwing shoulder. The throwing athlete with a SLAP tear should be separately considered from non-throwers, with more specific outcomes measures [7, 8, 71], and an understanding of associated pathology. Alternatives to repair include tenotomy or tenodesis of the biceps, which is becoming more accepted as a treatment option in many patients. Concern remains, however, as to the effect of these treatments in the thrower, as the biceps has been shown to play an important role in stabilizing the shoulder during the pitch [52, 72–74], and no study has compared these two approaches directly. One recent report [75] noted the nonoperative management of type II SLAP lesions in the throwing athlete is associated with a greater rate of return not only to competition but to the same level of competition as well.

Complications of repair have been reported by Weber et al. [6] as "neither uncommon or insignificant with an overall rate of 4.7 %" in their study of SLAP incidence reported by American Board of Orthopaedic Surgery applicants. Other reports have noted that patients who do not do well after primary SLAP repair may present with pain, stiffness, and/or mechanical symptoms. Revision treatment of these patients' results in suboptimal outcomes compared to primary SLAP repairs [76, 77].

Arthroscopic Treatment: Surgical Technique

Patient Positioning

While either the beach chair or lateral decubitus position can be successfully employed in the arthroscopic treatment of the SLAP repair, we prefer the lateral position in conjunction with general anesthesia and an interscalene nerve block. After performance of the surgical time out to ensure laterality and antibiotic prophylaxis, both shoulders are examined under anesthesia. Particular attention is paid to the detection of catching or reproducible clicking which may be suggestive of mechanical blocking or loose bodies in the joint. Further, we are careful to document objective signs of translation, as superior labral tears may result in or exacerbate translation on examination.

Portals

In general, most SLAP tears can be addressed through standard posterior and anterosuperior portals. We begin with a standard posterior viewing portal, established approximately 2 cm below and 1 cm medial to the posterolateral acromial corner. Once entry is established, we confirm the presence of the SLAP tear and place the anterosuperior cannula slightly more superolateral than normal. This position is approximately 1 cm off of the anterolateral corner of the acromion, and the entry is made directly behind the biceps, just inferior to the leading edge of the supraspinatus (Fig. 20.3). This position allows for anchors to be placed at the 11:00 position posterior to the biceps without a separate portal or violation of the rotator cuff. On occasion, an accessory mid-glenoid portal can be placed to assist with suture management, and a trans-cuff or Neviaser's portal [78] can be established to aid in access to the posterior-superior labrum. The decision to add one of these portals is based on surgeon preference and takes into account the size of the patient, the stiffness of the capsular structures, and the associated pathology to be addressed. The use of the "portal of Wilmington" created just

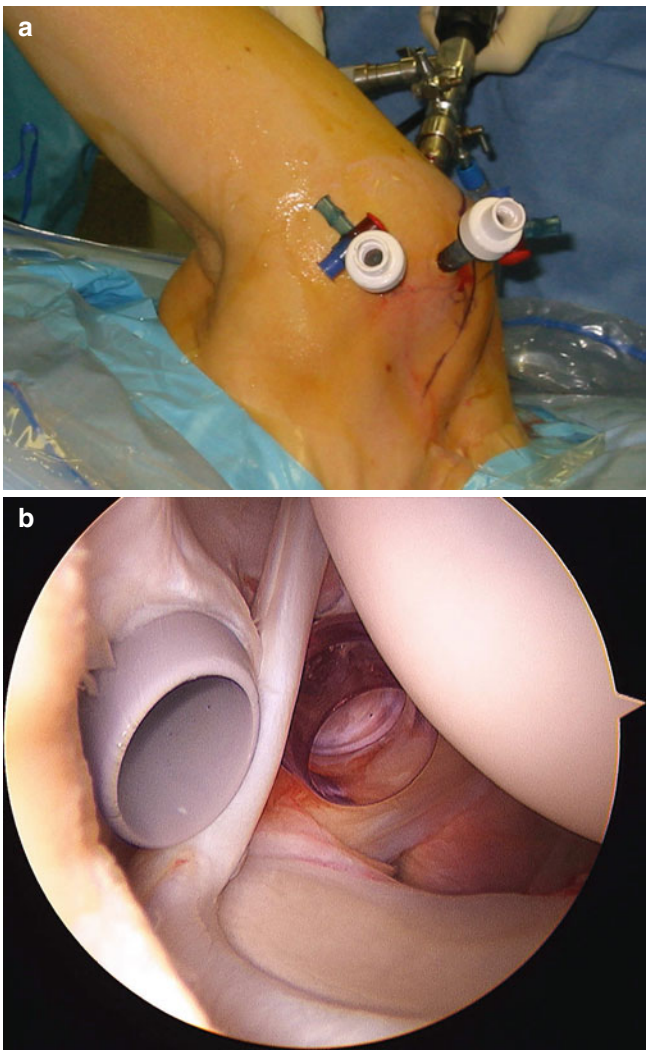

Fig. 20.3 External (**a**) and intra-articular (**b**) views of anterosuperior portal position for SLAP repair. Note that the position just posterior to the biceps tendon allows for optimal anchor positioning

anterior and inferior to the posterolateral corner of the acromion does provide access to the posterior-superior aspect of the glenoid and is very effective in addressing the posterior-superior type II SLAP lesion.

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

The diagnostic arthroscopy is a critical step in the procedure, and we use visualization as well as palpation from both the anterior and posterior portals to ensure that we have a solid grasp on the extent of the pathology. We determine the extent of the tear and other pathology with Snyder's 15-point shoulder examination [13]. We confirm a SLAP lesion by utilizing a similar process to Snyder's criteria for determination of a SLAP tear, including separation of the chondrolabral junction, erythema at the bicep anchor insertion, and a minimum of 5 mm of labral excursion [79]. Taking the arm out of traction and placing the arm in abduction and external rotation can also help in evaluating abnormal excursion if a "peel-back" phenomenon is present. Every biceps should also be tested with a probe pulling the biceps into the glenohumeral joint so that portion of the biceps within the bicipital groove can at least be partially viewed for additional distal pathology.

Step-by-Step Procedure (Box 20.1)

Box 20.1: Tips and Tricks

- Ensure portals are made in the correct position to be able to reach the desired posterior-superior anchor position. It is far better to establish an accessory trans-cuff or Neviaser's portal than to settle for a suboptimal anchor position.
- If using a suture shuttling technique, establish a mid-glenoid portal to ensure ease of suture management. While a single portal can be used to accomplish this, it is a simple step that costs little in time and can greatly aid in case progression.
- Do not neglect biologic preparation. Ensure bleeding cancellous bone at the top of the glenoid to ensure a good long-term result. Do not over-resect the superior glenoid.
- Ensure that suture material is well away from the articular surface at completion. Knotless systems or mattress sutures are excellent methods to aid here. Oblique mattress sutures optimize suture position and anatomic reattachment, avoiding over-constraint of the biceps anchor.
- Avoid placing anchors beyond the 12:30 position as SLAP lesions rarely extend beyond this position and the risk of external rotation loss is significant.

The first and most important step in the repair is biologic preparation of the lesion. Even a perfect repair will eventually fail if the underlying biology does not respond and heal the lesion. Thus, we spend the majority of our time ensuring that the tear is debrided and that the glenoid is taken down to a bleeding bed. All degenerative and fibrinous tissue is removed until bleeding cancellous bone is well visualized along the course of the planned repair (Fig. 20.4). This can be accomplished with an arthroscopic burr, but we prefer a rasp or shaver as the superior glenoid is relatively soft, and an effort should be made to avoid inadvertent bone resection which can compromise the anatomic attachment of the biceps anchor.

The next step is to plan the position of anchor insertion. Anchors should be placed in such a manner as to compress the tear against the superior labrum without excessive shear and with as low a profile as possible. For a typical 11:00–1:00 SLAP, anchors are placed at the 11:30 and 12:30 positions.

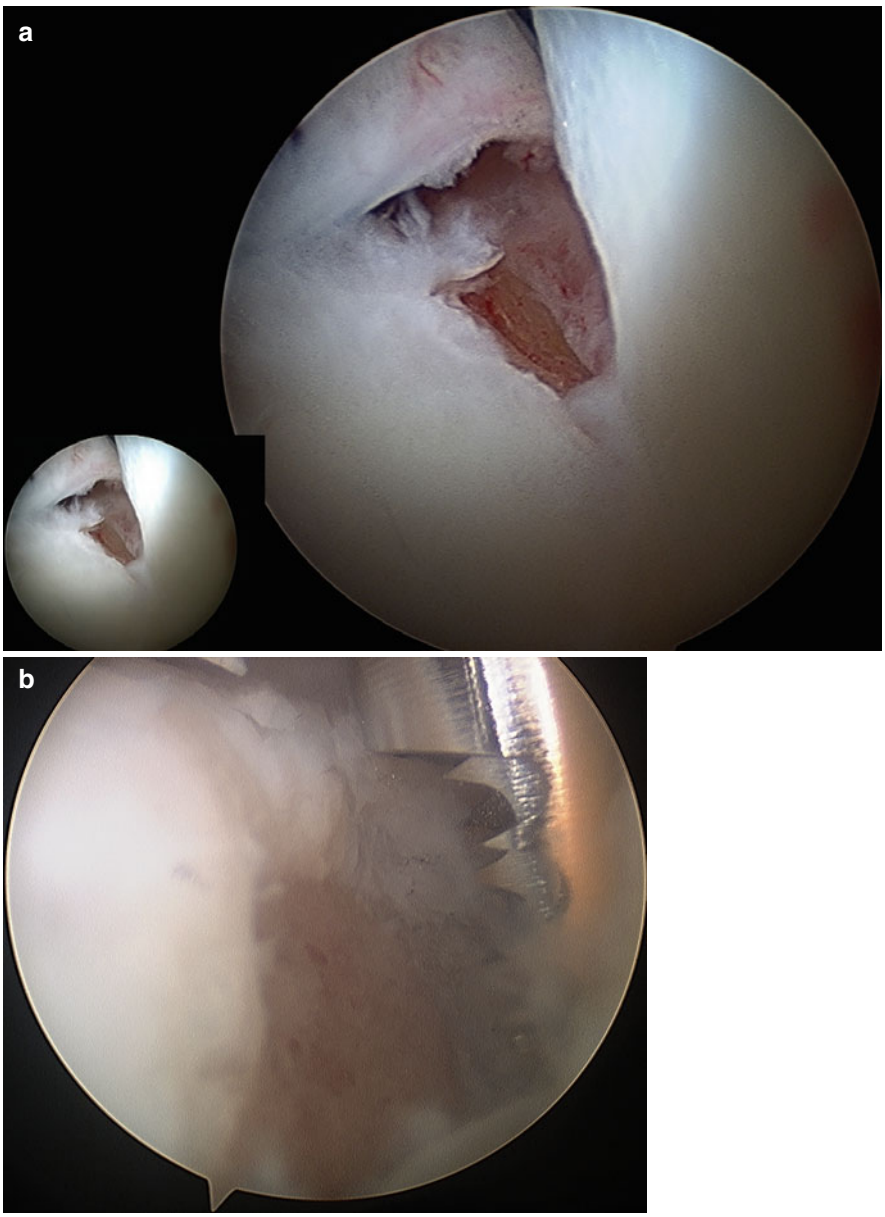

Fig. 20.4 Arthroscopic view of SLAP tear pre-repair (**a**). Note the preparation to bleeding cancellous bone without over-resection of the superior glenoid (**b**)

This allows direct compression of the tear without translating the labrum to a nonanatomic position. Anchor position should never be dictated by one's portal position, and additional portals should be liberally made to accommodate the correct position as opposed to settling for suboptimal anchor placement. Every effort should be made to not over-constrain the biceps anchor. Placement of sutures beyond the 12:30 position poses the risk of external rotation loss, in which very small differences in the overhead athlete can have devastating consequences. Needless to say, restoring anatomy, and not just stabilizing the labral attachment, is of critical importance in the high demand overhead athlete.

Suture passage can be accomplished with the use of a single-step tissue penetrator-suture grasper or can be done with the use of suture shuttles (Fig. 20.5). If the latter is used, we recommend converting to dual anterior portals, to ensure ease of suture management. As noted above, one can employ either simple or mattress configurations, and a multitude of knots are available to ensure loop security. Of utmost importance is ensuring that all suture material is well away from the articular surface upon completion (Fig. 20.6). Knotless suture anchors can obviate this concern and are an option in the treatment of the SLAP lesion.

Postoperative Care

Patients remain in an immobilizer-type sling for 3–4 weeks with scapular mobility, gentle passive range of motion (ROM) to 90° of scaption and 30° external rotation. Complex

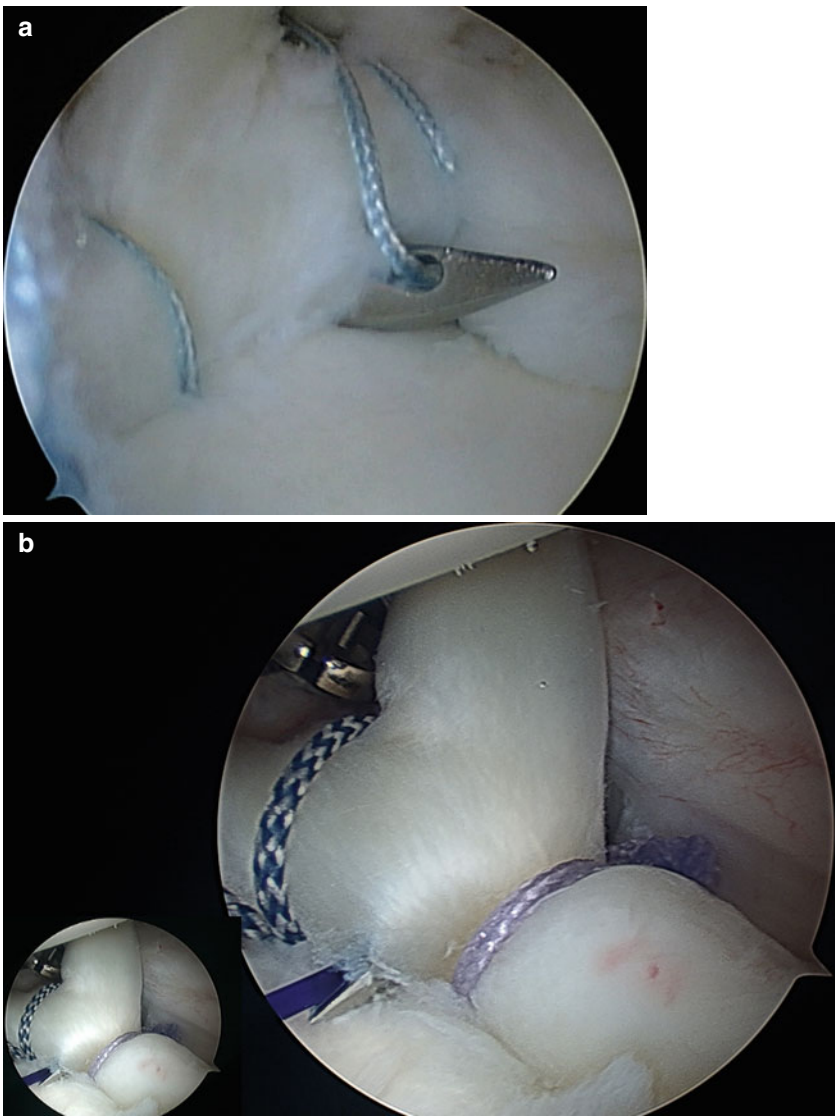

Fig. 20.5 Suture shuttle passage beneath biceps anchor using suture penetrator (**a**) or suture shuttle technique (**b**)

tears or those associated with instability in young patients may necessitate up to 6 weeks of immobilization. After 4–6 weeks, the patient gradually progresses to full range of motion, with active ROM allowed. Gentle rotator cuff strengthening begins 6 weeks postoperatively, and at 3 months, patients are allowed to return to a sport- and work-specific strengthening regimen, with resumption of full activities between 4 and 5 months. At 5–6 months, overhead lifting and sporting activities are allowed. Throwing and overhead sports often incur a longer recovery, and patients should be appropriately counseled [12, 80].

Literature Review

The evaluation of the literature on the outcomes of SLAP tears is no simple topic. Coexistent pathology, age, and activity level are just a few of the confounding influences on outcome, and many of the studies do not stratify these confounders in their results. Further, the technology of SLAP repair has changed from the absorbable tack to the suture anchor which may affect results. Finally, SLAP tears in pitchers should be considered a separate topic, as return to elite throwing is affected by not just surgical outcome but also by the level of competition.

For the properly diagnosed SLAP lesion, especially when isolated, most studies report very high rates of pain relief and subjective satisfaction. Recent studies which include the use of modern techniques report that up to 90 % of patients can achieve good or excellent results with regard to patient satisfaction and validated outcomes scores [25, 65–68]. These results, however, should be embraced with caution in the thrower, where a return to competitive throwing has been less reliable. As noted, this population should be separately considered in the analysis of outcomes, and the published studies on this specialized topic are summarized in Table 20.4.

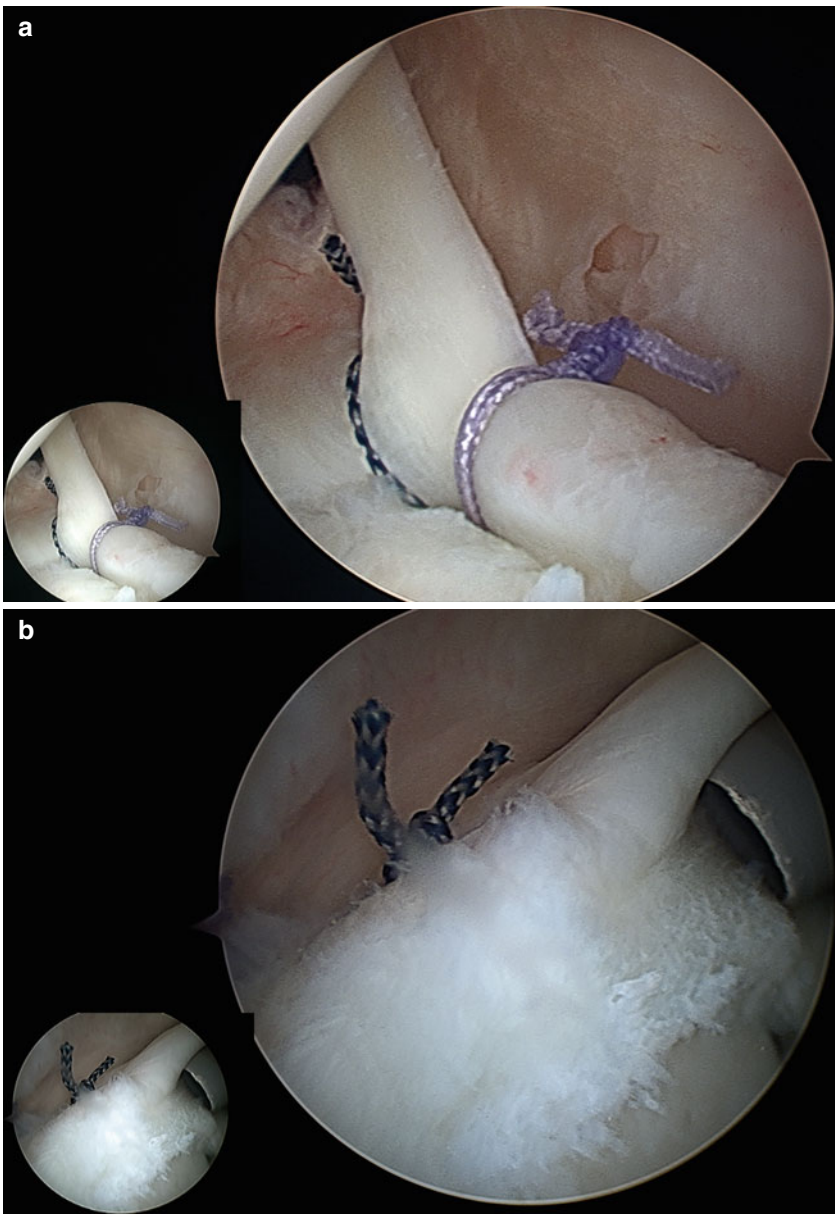

Fig. 20.6 Completed SLAP repair demonstrating a double-loaded anchor technique (**a**) and a mattress repair (**b**). Note in either case, care is taken to keep the suture knots off of the articular surface

Summary

Superior labral lesions remain a controversial and challenging diagnosis for the shoulder surgeon. Variation in anatomy, changes with aging, and a lack of conclusive findings on history, physical examination, and radiographic imaging combine to challenge even the most experienced clinician. A thorough and meticulous approach, combining these diagnostic tools can aid the treating physician in differentiating between normal variation and pathologic lesions. Surgical decision making must include the patient's age, chosen activity, and associated pathology. Recognizing and treating associated pathology is an important part of overall patient care, and treatment of the SLAP lesion itself should be approached with attention to detail by the surgeon. Asymptomatic SLAP lesions, especially in the older population, must be carefully considered when formulating a treatment plan. Postoperative rehabilitation is an important part of the overall management of these lesions, especially in the overhand athlete. Patient expectations must be managed, as pain relief is more reliable than is the return to previous levels of pitching. With a fastidious and discerning approach, treatment of these lesions can be successful and satisfying for both patient and surgeon.

Table 20.4 Return to throwing after SLAP repair

Author	Surgery	Athletes	Results	Noteworthy
Morgan (1998) [34]	Repair of SLAP	37 pitchers in 102 throwers	87 % returned to previous level of play	
Bradley (2008) [71]	Posterior capsulolabral repair throwers vs. non-throwers	27 throwers	89 % good and excellent, but 55 % returned to previous level	
Kim (2002) [66]	SLAP repair	18 overhead athletes	Only 22 % overheads returned to same level	
Andrews (2003) [80]	Repair ± thermal in throwers	130 athletes (105 pitchers)	87 % returned to play	Better results with thermal and repair than repair alone
Ide (2005) [67]	SLAP repair	19 pitchers	63 % returned to prev level	Results worse in baseball than other overhead athletes
Neuman (2011) [7]	SLAP repair	30 overhead athletes	ASES: 88, KJOC: 74	3.5 year retro review survey
Brockmeier (2009) [68]	SLAP repair	28 overhead athletes in larger series	71 % return to throw	Traumatic return to play higher (92 %) than insidious (64 %)
Cohen (2011) [81]	SLAP repairs from larger group of pitchers	22 pro pitchers	32 % return to play	
Neri (2011) [8]	SLAP repairs	23 elite throwers	57 % returned to preinjury level, worse with RC tear	KJOC score better than ASES in predicting outcome

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Subacromial Impingement

21

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The subacromial space is delimited above by the coracoacromial arch, which consists of the anterior part of the acromion, the coracoid process, the coracoacromial ligament (CAL), and the acromioclavicular (AC) joint and below by the humeral head covered by the rotator cuff. In 1972, Neer [1] first highlighted the correlation between degenerative changes of the coracoacromial arch and tendinopathy of the rotator cuff and long head of the biceps. He described the "subacromial impingement syndrome" as the main cause of shoulder pain due to a pathological reduction of the subacromial space that results in an attrition between the rotator cuff and the coracoacromial arch during the movements of flexion, abduction, and rotation of the limb, which can cause degenerative lesions of all the structures contained in the subacromial space.

Epidemiology

Subacromial impingement is the most common cause of shoulder pain, accounting for 44 % up to 65 % of shoulder disorders [2–5].

Several studies showed the prevalence of shoulder pain in different countries: a recent study in France identified subacromial impingement syndrome as the most common upper extremity disorder in the working population [6]; a Dutch study estimated the cumulative incidence of shoulder problems at 19/1,000 patients per year [7]; a review, which summarized 18 studies on the prevalence of shoulder complaints in the general population in the USA, UK, Scandinavia, Cuba, South Africa, Spain, and Nigeria, showed 4.7 to 46.7 % for 1-year prevalence [8].

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Prevalence is especially high in overhead athletes and manual workers, due to the athletic gesture and its repetition over time [9–12].

Pathophysiology

It is not easy to identify and understand the etiology of shoulder pain because the impingement syndrome is the result of the interaction between different factors, variously combined together [13]. Ellman and Gartsman [14] classified these factors as intrinsic, extrinsic, and secondary.

Intrinsic Factors

Intrinsic factors influence tendon morphology and performance. They can increase tendon thickness (swelling) and, at the same time, can lead to a functional imbalance of force couples between deltoid and supraspinatus tendon (see Chap. 2).

Intrinsic factors can be attributed to:

- Natural process of aging of rotator cuff tendons [15–18]
- Poor vascularity [19–24]
- Inferior biological and mechanical properties resulting in damage with tensile or shear loads [25–30]

The prevalence of tendon degeneration including partial- and full-thickness tears increases as a function of age, starting at 40 years [15–18, 31, 32]. Biomechanical studies suggested that there is a decreased elasticity and overall tensile strength of tendons with age [33]. Histological studies have shown degenerative changes such as calcification and fibrovascular proliferation in elderly subjects that were not present in younger subjects, both without a history of shoulder disorders [25]. Furthermore, there is a decrease in total glycosaminoglycan and proteoglycan content, an overall reduction of type I collagen content and an increased proportion of weaker, more irregularly arranged type III collagen fibers, and a greater tenocyte apoptosis [25, 26, 34, 35]. Obviously, these

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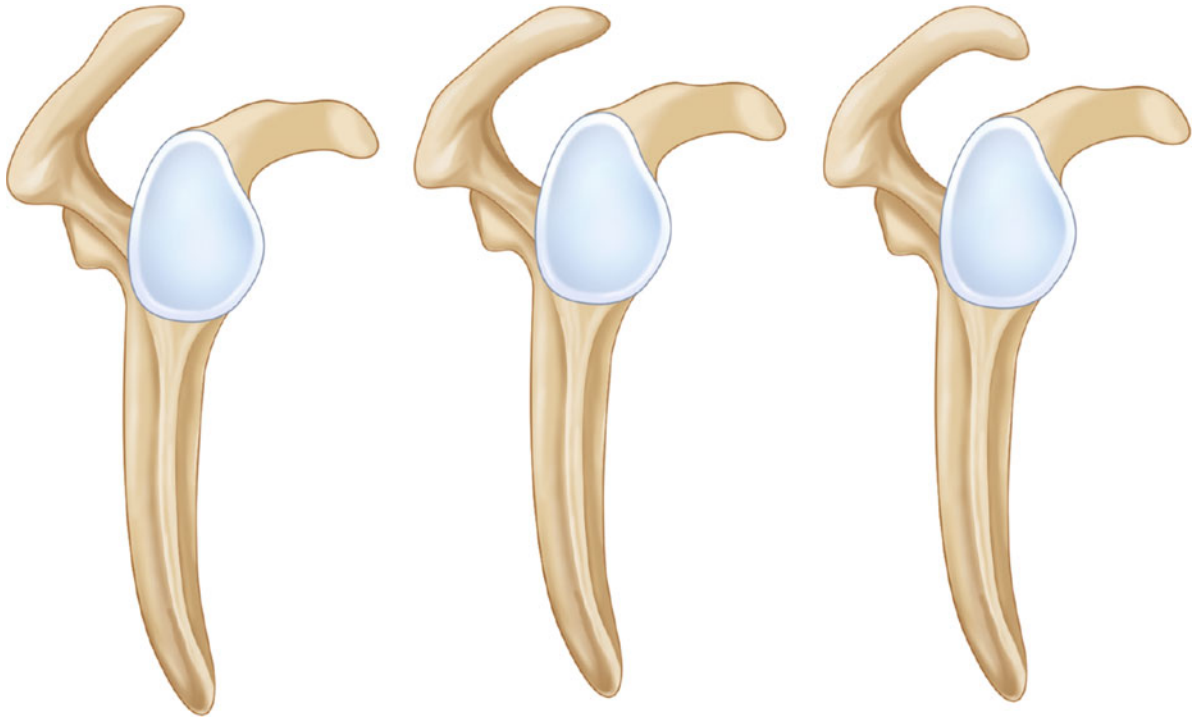

Fig. 21.1 Acromial morphology, according to Bigliani's classification

matrix alterations are concurrent with changes in tendon morphology characterized by an irregular tendon thickness [36].

The role of vascularity has not been fully elucidated. Codman first described the "critical zone," an area within the supraspinatus tendon approximately 1 cm from the insertion on the greater tuberosity with decreased vascularity, as the most common site for tendon injury [37]. In contrast, in vivo studies found no evident avascularity in the critical zone [38–40] nor evidence of hypovascularity in the articular side of the tendon [24, 41]. Conversely, several authors suggested an increased vascular response associated with degenerative changes and chronic rotator cuff tendinopathy [19, 20, 23, 25, 40].

Supporters of the intrinsic theory [20, 42] believe that the main factor triggering the subacromial impingement syndrome is tendon damage, especially of the supraspinatus. Degenerative changes or repeated microtrauma can weaken the supraspinatus tendon, which is then unable to keep the humeral head in the center of the glenoid cavity. Thus, the humeral head tends to shift upwards, causing narrowing of the subacromial space, which leads to an attrition between the rotator cuff and the undersurface of the acromion. Therefore, subacromial impingement is a consequence rather than the cause of cuff rotator injuries.

Extrinsic Factors

Extrinsic factors include etiological agents which can bring about anatomical reduction of the subacromial space, where the supraspinatus tendon engages. Neer was the first to define this condition as "outlet impingement" and to consider it the most common cause of impingement [43].

The reduction of the subacromial space is linked to a morphological alteration of the components of the coracoacromial arch:

- Acromial morphology: Bigliani et al. described three types of acromion: type I (flat), type II (curved), and type III (hooked) [44] (Fig. 21.1). Type III acromion has proved to be significantly associated with the development of impingement syndrome [45, 46].
- Acromial angle or acromial slope: a flatter slope or more horizontal position of the acromion is associated with subacromial impingement [47–50].
- Acromial tilt: Aoki et al. [47] noted a decreased acromial tilt (acromial posture in relation to the scapula) in the patients who had impingement.
- Osteophytosis of the inferior surface of the acromioclavicular joint [1, 51].
- Osteophytosis of the anteroinferior edge of the acromion with ossification of the CAL [1, 52, 53].
- Os acromiale: it results from failure of an acromial ossification center to fuse to the acromial process [54]. This can cause impingement because the hypermobile epiphysis can bend forward as a result of pulling by the coracoacromial ligament.
- Posttraumatic changes caused by fractures of the greater tuberosity, the coracoid, or the acromion [55–57].

Soslowsky et al. [58] carried out a study on animals (rats) and showed that extrinsic mechanical compression brings

about degenerative changes in rats exposed to overuse activities, but not in rats performing normal cage activities. Therefore, it is possible that bony anatomy can predispose to the development of an impingement syndrome without being the cause. Supporting this theory of a requisite overuse exposure, Yamaguchi et al. [59] have shown that cuff tendinopathy is more often symptomatic in the dominant limb.

Secondary Causes

Secondary impingement mechanisms are mainly related to biomechanical factors, such as alterations of the normal scapulohumeral kinematics, postural abnormalities, rotator cuff and periscapular muscle performance deficit, and reduced extensibility of pectoralis minor and of posterior capsule. Abnormalities in scapulohumeral kinematics represent a dynamic cause of subacromial space narrowing [60–62]. Postural abnormalities, muscle deficit, and retraction of the pectoralis minor and the posterior capsule directly affect scapular and humeral kinematics.

Patients affected by subacromial impingement generally have decreased scapular posterior tilting, reduced upward rotation, and increased internal rotation [63–65]. Consequently, secondary causes are the causes which somehow interfere with the normal stabilization mechanisms of the glenohumeral joint.

Instability

Instability is the most common cause of impingement in young patients. Anterior or multidirectional instability alters normal passive stabilization mechanisms thus preventing the supraspinatus from functioning at its best. This alters the dynamic equilibrium of the joint and causes the humeral head to shift upwards during elevation due to prevailing deltoid, thus resulting in subacromial impingement [66, 67]. Many throwing athletes affected by chronic subacromial impingement syndrome often suffer from associated glenohumeral instability. These patients are likely to suffer instability as a primary condition and rotator cuff tendinopathy as a secondary problem [68].

Overuse

Overuse damage of static stabilizers (common in throwing athletes) can cause alterations of the dynamic stabilizers, especially of the rotator cuff, resulting in the alteration of shoulder function. In fact, the stress caused by throwing can be greater than the compensation capacity of the static stabilizers [69]. This can result in excessive traction on the rotator cuff. Excessive traction can, in turn, weaken the rotator cuff and periscapular muscles and produce functional impingement, leading to the onset of a subacromial impingement syndrome.

Posterosuperior Impingement (Internal Impingement)

This is a particular form of impingement occurring between the articular surface of the supraspinatus tendon (and occasionally of the infraspinatus) and the posterosuperior edge of the glenoid [70–73]. This condition prevails in young overhead or throwing athletes. In this condition, impingement tests are usually negative whereas pain can be elicited by the apprehension test carried out in hyperabduction (extreme abduction-external rotation), which reproduces the mechanics of the impingement between the posterior glenoid and the articular surface of the rotator cuff.

Loss of Vault Suspension

Trapezius muscle paralysis or an inveterate AC dislocation, with an injury of the coracoclavicular ligament (conoid and trapezoid ligaments) can alter the coracoacromial arch, thus resulting in a limitation of the external rotation of the scapula during abduction and alteration of the scapulohumeral rhythm. The failed elevation of the vault during abduction leads to the premature engagement of the greater tuberosity under the acromion (30–40°) with the inevitable onset of a subacromial impingement [74].

Tightness of the Posterior Capsule

Tightness of the posterior capsule causes the anterosuperior migration of the humeral head, whereby the limb is moved in forward flexion and internal rotation [75]. This pathological condition, which is common in throwing athletes affected by painful shoulder, can trigger or worsen an impingement syndrome as it moves the humeral head against the acromion during forward flexion.

Scapular Dyskinesis

Scapular dyskinesis can be caused by anatomical, neuromotor, or kinematic abnormalities of the scapulothoracic region. Protraction and elevation of the scapula lead to the anterior tilt of the acromion, thereby reducing subacromial space (see Chap. 19).

Neurological Injuries

In the case of neurological injuries (cervical radiculopathy, suprascapular neuropathy), the deltoid prevails during abduction, and causes the upward migration of the humeral head, leading to impingement.

History

Each of the abovementioned factors (internal, external and secondary) can have an important role in the subacromial impingement syndrome. Moreover, in many cases the etiology is multifactorial; therefore, several factors can contribute together or sequentially to determine the clinical presentation of the syndrome. Whatever the predisposing cause, the evolution of this syndrome appears rather unidirectional since, once triggered, it becomes self-perpetuating.

Neer [1] described three progressive stages of impingement:

- Stage I: bursitis with subacromial edema and hemorrhage

- Stage II: onset of tendinopathy and initial development of a partial-thickness tear
- Stage III: progression from a partial-thickness tear to a full-thickness tear

Degenerative changes of the rotator cuff lead to a diminished capacity of the supraspinatus to keep the humeral head in the center of the glenoid; therefore, on the one hand, the deltoid tends to shift the humeral head upwards, while on the other hand, functional overload of the residual supraspinatus and of the other stabilizers impede an adequate balance of the force couples. This further worsens rotator cuff tendinopathy, leading to the progressive reduction of the subacromial space. Moreover, the coracoacromial arch, being subjected to continuous attrition, develops degenerative changes which, in the long run, will bring about a morphological alteration leading to a further anatomical reduction of the subacromial space. This tends to aggravate the impingement, progressively resulting in a full-thickness rotator cuff tear, degenerative changes of the long head of the biceps, and ultimately causing degenerative alterations of the glenohumeral joint.

Clinically, it will therefore not be easy to distinguish an impingement syndrome from a rotator cuff tear. Patient's age, sport activity level and work activities, traumatic or spontaneous onset of the symptoms, progression of symptoms, and the evaluation of strength will help to establish a diagnosis.

The main symptom in the subacromial impingement symptom is pain, varying in intensity. Patients may refer a vague discomfort in the shoulder or a sharp pain, generally described as "deep" or anterolateral, often exacerbated by the elevation of the limb beyond 90°. Sometimes it can be located in a single spot or, if the long head of the biceps is involved, the pain can run along its path.

Patients over 40 years of age, manual workers without history of trauma, but spontaneous and progressive development of symptoms without strength deficit are the typical patients affected by subacromial impingement.

However, the diagnosis of impingement syndrome should also be taken into consideration in young throwing athletes with chronic aspecific painful shoulder during elevation beyond 90°, without macrotrauma. Lastly, clinical examination and imaging will be crucial for the final diagnosis.

Clinical Examination

An accurate clinical examination will include the inspection of both shoulders, evaluation of the articular range of motion (ROM), static and dynamic evaluation of both scapulae, as well as carrying out specific tests to evaluate tenderness, muscle strength, and the possible involvement of the long head of the biceps.

The inspection should involve both shoulders so as to rule out muscle hypotrophy, which could otherwise be overlooked.

ROM should be assessed in elevation (on the scapular plane), abduction, and internal and external rotation, both actively and passively.

Pain is commonly localized on the anterolateral side of the affected shoulder at the level of the greater tuberosity and the insertion of the CAL. The long head of the biceps is often involved in subacromial impingement, especially in the advanced stages of this condition. In this case pain will be localized anteriorly at the level of the proximal region of the intertubercular groove. Tenderness to palpation of the AC joint can be a sign that the joint is involved in the impingement syndrome. Palpation and auscultation often make it possible to detect crackling noises during abduction due to inflammation and thickening of the subacromial bursa.

Impingement specific tests include:

- Painful arc: pain is elicited by active abduction on the scapular plane between 60° and 120° [76, 77].
- Impingement sign: pain is elicited by passive elevation of the limb on the scapular plane, with internal rotation of the humerus and the examiner's hand stabilizing the scapula. Pain is elicited by abduction greater than 70°.
- Hawkins test: the arm is forward elevated to 90°, then internally rotated and adducted thus making contact between the supraspinatus and the anterior portion of the acromion and the CAL.
- Yocum test: patient's hand is placed on the non-affected shoulder. The examiner asks the patient to lift up the bent elbow.
- Neer Test or impingement test: subacromial injection of 10 ml local anesthetic. Negative impingement sign after local anesthetic injection is indicative of a subacromial impingement.

Rotator cuff specific tests evaluate strength deficit. The main tests that should be carried out are Yocum test, Jobe test, drop-arm sign, Patte test, and lag sign (ER/IR). Lift-off, Napoleon test, and the Bear Hug test assess the involvement of the subscapularis tendon. Involvement of the long head of the biceps is assessed through several specific tests such as palm-up test, Yergason test, active-passive test of the long head of the biceps, and Ludington test. A detailed description of these tests is supplied in the dedicated chapters (Chaps. 3 and 23).

An accurate clinical examination of the scapulae is crucial to exclude secondary causes of impingement. The main tests for strength assessment are "wall push-ups" and "flip sign." There are basically two corrective maneuvers:

- Scapular assistance test (SAT): the examiner applies gentle pressure to push on the inferior medial scapular angle to assist scapular upward rotation and posterior tilt as the patient elevates the arm. A positive result is indicated by relief of painful symptoms related to the arc of impingement and on increased arc of motion.
- Scapular retraction test (SRT): the examiner places the scapula in a retracted position and manually stabilizes it, then repeats the tests to evaluate rotator cuff strength. The

test is positive if painful symptoms and strength improve during the tests.

Shoulder pain is involved in the differential diagnosis of several conditions. The first important finding to assess is whether pain is primarily located or radiating to the shoulder.

Differential diagnosis of impingement syndrome should include shoulder diseases commonly associated with local pain, such as rotator cuff tears, calcific tendonitis, adhesive capsulitis, peripheral neuropathy (i.e., entrapment of the suprascapular nerve), and glenohumeral osteoarthritis.

Clinical examination is not sufficient to discriminate between impingement syndrome and calcific tendonitis. Rotator cuff tears can have a traumatic as well as degenerative onset, and they are characterized by strength deficit, which is generally missing in impingement. However, intense pain can produce a loss of active motion and/or weaknesses.

Suspicion of adhesive capsulitis in the early stage (intense pain, limited loss of motion) arises from comorbidity frequently associated with the disorder (diabetes, thyroid diseases, cervical diseases) and in presence of limited passive ROM. The impingement test, by eliminating the pain, can be a valid help to rule out a real shoulder stiffness. Finally, neuropathy will be suspected if strength deficit occurs with muscle hypotrophy.

Pain radiating to the shoulder is common in cervical radiculopathy and in the thoracic outlet syndrome. Cervical disease is suspected in presence pain primarily located to the neck and scapular region, associated with varying degrees of sensory, motor, and reflex changes as well as dysesthesias and paresthesias to the whole upper limb with a dermatomal distribution (primary shoulder pain does not generally reach farther down than the elbow). Suspicion of thoracic outlet syndrome will easily be cleared by history and by carrying out specific tests.

Imaging

Imaging is crucial in differential diagnosis between subacromial impingement syndrome and other conditions which can commonly cause shoulder pain. At the same time, imaging is useful to establish the cause of impingement.

Standard radiographic examination should be carried out with impingement series: true anteroposterior, outlet, and axillary views. True anteroposterior view allows the evaluation of posttraumatic or degenerative alterations of the glenohumeral joint, changes in the acromiohumeral space, as well as calcifications. The axillary view rules out the presence of an os acromiale (Fig. 21.2). Outlet view allows the evaluation of the acromion morphology (Bigliani classification), of the acromial angle, and the possible existence of an acromial spur extending into the CAL. Snyder [78, 79] suggested a classification of acromial morphology based on the thickness of the acromion at the junction between anterior and middle third, outlining three acromion types:

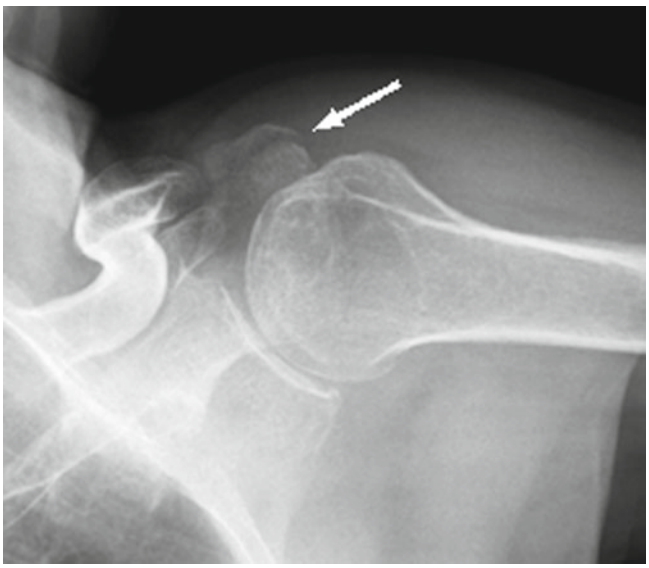

Fig. 21.2 Axillary X-ray view showing an os acromiale (*arrow*)

- Type A: thin acromion , thickness <8 mm
- Type B: medium acromion, thickness 8–12 mm
- Type C: thick acromion , thickness >12 mm

The evaluation of thickness of the acromion is important to make a correct preoperative planning, especially when an arthroscopic acromioplasty is indicated. Out of 200 patients with impingement syndrome, Snyder et al. [78] showed that 34 % of female patients with type III acromion, according to Bigliani classification, had acromion thickness <8 mm (type A). In these cases, subacromial decompression is associated with a higher risk of acromion fracture.

Magnetic resonance (MR) allows an accurate evaluation of the bursa and the rotator cuff tendons [80, 81]. It will be crucial in the ruling out of rotator cuff tear.

Ultrasound [82, 83] and MR arthrography (MRA) [84] have also been used. Ultrasound is a simple, noninvasive examination, but operator-dependent; MRA is undoubtedly highly specific and sensitive, but unlikely to be more decisive in diagnosing subacromial impingement compared to MR.

Treatment: Indications and Contraindications

Treatment of subacromial impingement is primarily conservative.

In case of impingement stage I–II, surgery consists in subacromial decompression and is appropriate only after failure of 6-month conservative treatment. Impingement stage III will need rotator cuff repair.

Subacromial decompression is a complex and aggressive surgery. It includes three surgical procedures: subacromial bursectomy, release of the CAL, and the removal of the anteroinferior edge of the acromion (acromioplasty). Indication is still being debated.

Subacromial decompression is not indicated for:
- Stage I–II impingement as first-line treatment
- Partial-thickness articular-side rotator cuff tears
- Irreparable rotator cuff tears
- Painful shoulder in athletes

On the other hand it is relatively indicated (as isolated or combined procedure) for:
- Partial-thickness bursal-side rotator cuff tears
- Full-thickness rotator cuff tear
- Calcific tendonitis

It is also appropriate when arthroscopy shows direct signs of impingement, such as abrasion and fibrillation of the undersurface of the acromion or of the CAL fibers at their acromial attachment.

Decision-Making Algorithm

Decision-making algorithm starts with an accurate evaluation of the patient. Patient's history and the clinical examination will guide the choice of the appropriate imaging modality. In particular:
- Pain without strength deficit will be indicative of impingement stage I–II, according to Neer [1] and standard radiographic exams can be considered.
- Strength deficit (positive rotator cuff specific tests) and loss of active ROM, with normal passive ROM will be indicative of stage III (rotator cuff full-thickness tear), according to Neer [1], and patients will undergo X-rays and MRI.
- If paresthesias or muscle hypotrophy are detected, electromyography (EMG) will also be useful.

Treatment choice will be influenced by the stage of the pathology and will be primarily aimed at reducing pain and restoring shoulder function:
- Stage I: conservative treatment characterized by rest, change in activities, non-steroidal anti-inflammatory drugs (NSAIDs) (if there are no general contraindications), subacromial injections of hyaluronic acid, isolated or associated with physical therapy; and single corticosteroid injection in the acute stage followed by rehabilitation.
- Stage II: conservative treatment. After failure of 6-month conservative treatment, the patient will be clinically reassessed and may be referred for subacromial decompression and/or rotator cuff repair.
- Stage III: arthroscopic rotator cuff repair.

In case of posttraumatic impingement syndrome (due to coracoid, acromion, or greater tuberosity fracture), the patient should be immediately referred for surgery aiming at removing the mechanical cause through subacromial decompression or reshaping of the greater tuberosity (tuberoplasty), associated with rotator cuff repair if indicated.

Subacromial impingement due to scapulohumeral kinematic abnormalities will be treated conservatively through an appropriate rehabilitation protocol (see Chaps. 6 and 19).

Arthroscopic Treatment: Surgical Technique

Patient Positioning

Surgery can be performed under general or regional anesthesia with the patient in lateral decubitus or beach chair position. We prefer regional anesthesia with possible sedation and beach chair position so that the acromion is parallel to the floor.

Portals

Three portals are sufficient to perform subacromial decompression:
- Posterior portal, used alternatively as viewing and operative portal
- Anterosuperior portal, used to control outflow or as accessory portal for the instruments
- Lateral working portal, used alternatively as viewing and operative portal

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

Evaluation under anesthesia, before starting surgery, is important to rule out possible signs of adhesive capsulitis or stiffness, instability, and assess subacromial crepitation, which may indicate an underlying rotator cuff pathology.

Now we can start the surgery. Position the arthroscope into the glenohumeral joint through the posterior portal. Air is blown into the joint with a 50 ml syringe, through the arthroscope sheath. The first examination is carried out in air: we evaluate the integrity of the subscapularis tendon, long head of the biceps and its pulley, glenoid labrum, glenohumeral ligaments, glenoid and humeral articular surfaces, and articular side of the rotator cuff.

During this evaluation, our attention will be influenced by the clinical examination previously performed, as well as by the patient's age: findings of glenoid labrum or glenohumeral ligament alterations will be of greater clinical significance in younger patients. If secondary impingement is detected, it will be necessary to treat instability findings, which are the first cause of the exacerbation of symptoms, and subacromial decompression will not be required. Intra-articular evaluation can be helped by palpation of anatomical structure using a probe or a switching stick inserted through the anterosuperior portal.

If no intra-articular lesions are detected, we can go on to the evaluation of the subacromial space. The subacromial

space is accessed by positioning the arthroscope in the posterior portal, but in this case, the arthroscope must run immediately under the inferior surface of the acromion. As soon as we enter the subacromial space, the subacromial bursa can impede vision; it will therefore be important to immediately set up all the portals and start with bursectomy. The inflow tube will be connected to the arthroscope sheath. The inflow will allow for space distension and therefore better visualization so that anterosuperior and lateral portal can be created. We will introduce a shaver or a radio-frequency instrument in the lateral portal in order to perform bursectomy. The anterosuperior portal will allow for outflow (preventing overflowing), and it can be used as an accessory portal to complete bursectomy. Once bursectomy is completed, the switching stick is positioned in the anterosuperior portal, and the rotator cuff is accurately examined and palpated for any alteration: hyperemic areas, thinning, or partial-thickness tears of the bursal side of the tendons. Then the arthroscope is rotated upwards to evaluate the undersurface of the acromion and the CAL for pathologic findings consistent with impingement, such as fibrillation or erosion of the undersurface of the acromion, degenerative changes of the insertion of the CAL, and os acromiale.

Step-by-Step Procedure (Box 21.1)

Box 21.1: Tips and Tricks

- To avoid inappropriate bone resection (too much or too little), it is necessary to carry out an adequate preoperative planning, and during the surgery it is important to evaluate the amount of bone to be resected, both from the posterior and lateral portals.
- During the resection of acromial osteophytes, the insertion site of the deltoid fascia must be preserved.
- During acromial resection through the posterior portal, the burr must be kept close against the posterior portion of the inferior surface of the acromion; this will make tangential resection of the inferior surface of the acromion possible in order to obtain a smooth, flat acromial profile.
- During acromial resection, the burr should be used in reverse mode to limit and check the aggressiveness of the instrument.
- If the bone is osteoporotic, acromioplasty is better performed with an aggressive shaver rather than a burr.
- Inappropriate handling of the burr or excessively aggressive aspiration can lead to poor intraoperative visibility and to accidental burring of the cuff. It will therefore be crucial to modulate aspiration and to keep the burr directed towards the acromion.
- During release of the CAL, starting from the bone surface and moving anteriorly towards the ligament fibers will reduce the risk of bleeding.
- Lowering systolic pressure and increasing the pressure and flow of the irrigation pump will improve hemostasis and visibility so that radio frequency can be used accurately.
- If bleeding occurs, the arthroscope should be directed on the bleeding spot so that the saline solution can help hemostasis and enable the surgeon to coagulate using radio frequency.

We perform only bursectomy if we detect a bursitis without changes of the rotator cuff tendons or the coracoacromial arch.

We perform bursectomy and superficial cauterization using a radio-frequency instrument if we detect hyperemic areas within the supraspinatus or the infraspinatus tendon, near the insertion site on the greater tuberosity. Hyperemia can indicate an acute tendonitis or calcific deposits (calcific tendonitis: see Chap. 28).

If partial-thickness bursal-side rotator cuff tears (Ellman grade I) are detected, bursectomy and debridement of the lesion will be performed. The arthroscope will be positioned in the posterior portal, the outflow cannula in the anterosuperior portal and the operative cannula in the lateral portal. Bursectomy and debridement will be performed using a shaver and a radio-frequency instrument through the lateral portal (Fig. 21.3).

If a more severe partial tear (Ellman grade II–III) is detected, the tear will be completed and subsequently repaired (see Chap. 22). In the case of a full-thickness rotator cuff tear, the tear will be repaired (see Chap. 23).

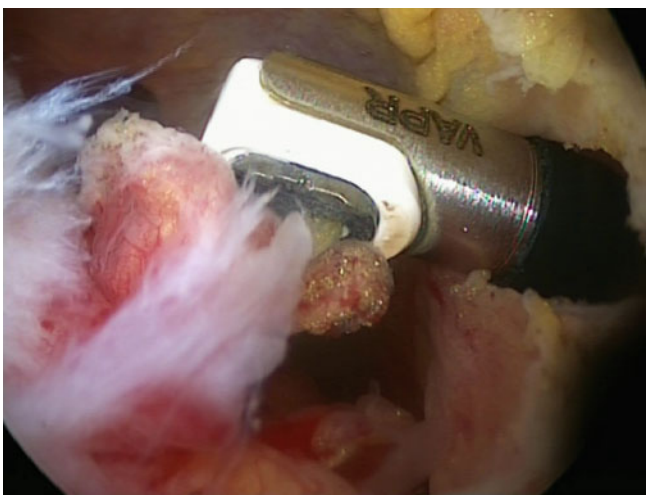

Fig. 21.3 Right shoulder. Bursectomy is performed using a radio-frequency instrument through the lateral portal. The arthroscope is in the posterior portal

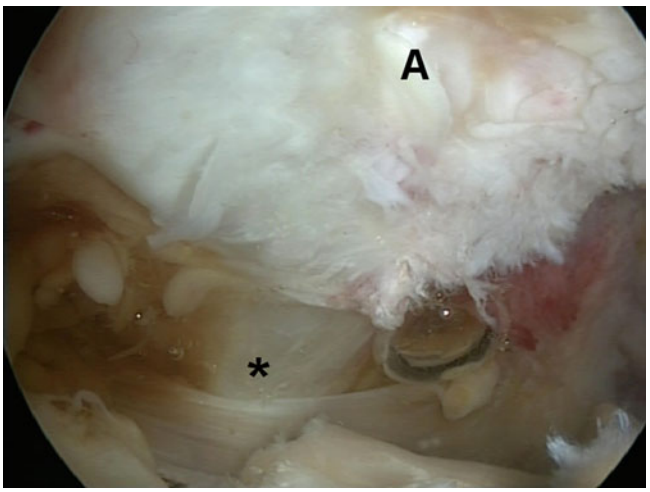

Fig. 21.4 Right shoulder. Arthroscopic signs of subacromial impingement. Fibrillation of the undersurface of the acromion can be observed (*asterisk* coracoacromial ligament, *A* acromion). The arthroscope is in the posterior portal

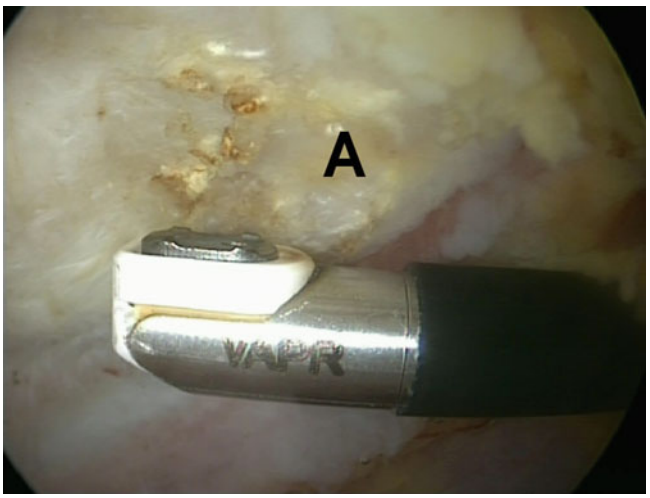

Fig. 21.5 Right shoulder. The radio-frequency instrument in the lateral portal is used to remove soft tissues from the undersurface of the acromion (*A*). The arthroscope is in the posterior portal

If an os acromiale is found, treatment will depend on the size of such finding: it can be arthroscopically removed with a motorized instrument, or, in the case of a large fragment, fixation could be indicated [85–88].

If we detect a type III acromion with clear signs of impingement (deep fibrillation or erosion of the undersurface of the acromion and/or spurring of the anterior edge of the acromion), a complete subacromial decompression will be performed (Fig. 21.4). The arthroscope will be introduced in the posterior portal and the outflow cannula in the anterosuperior portal. With a radio-frequency instrument in the lateral portal, we will remove and coagulate the periosteum and the CAL insertion site on the undersurface of the acromion (Fig. 21.5). Then we will proceed with a motorized instrument to allow for a better visualization of the anterior and lateral edges of the acromion and of the residual insertion site of the CAL. The CAL is then cut off the anterior edge of the acromion with radio frequency. The radio-frequency instrument must be kept close against the bone since the vessels within the ligament are 5–8 mm from the acromial edge, and a shift within the ligament fibers could lead to significant bleeding of the acromioclavicular branch of the thoracoacromial artery (Fig. 21.6). Release of the CAL can reveal an anteroinferior osteophyte of the acromion, close to the ligament insertion site. Acromioplasty is therefore performed. With an oval or a round burr, the anterolateral corner and the anterior edge of the acromion (usually 5–8 mm) are resected parallel to the anterior margin of the distal clavicle. Starting from the anterolateral corner of the acromion, we move medially towards the acromion-clavicular joint and remove the anteroinferior portion of the acromion and, where needed, the acromial osteophyte (Fig. 21.7). From the anterolateral corner of the acromion, we move posteriorly for about 1 cm

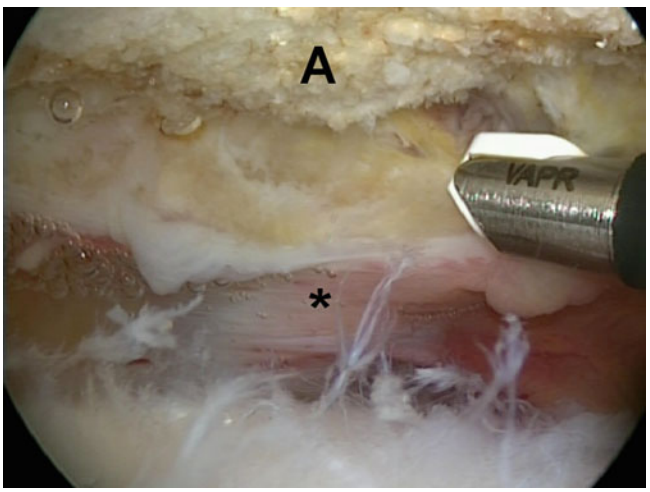

Fig. 21.6 Right shoulder. The release of the CAL (*asterisk*) is performed using a radio-frequency instrument through the lateral portal. The radio-frequency instrument must be kept close against the acromion (*A*) in order to avoid bleeding. The arthroscope is in the posterior portal

along the lateral edge of the acromion. Great care must be taken at this stage to preserve the deltoid insertion site. When the deltoid insertion site becomes visible, it means that resection is complete (Fig. 21.8). Posterior limit of the acromioplasty, according to the preoperative planning on X-ray, is then identified, taking the posterior surface of the distal clavicle as a reference landmark. When this level is defined, an oval burr is used through the lateral portal to create a 3 mm deep groove across the whole width of the acromion. This groove will be used as a reference for the posterior resection level and as bone edge for the oval burr (Fig. 21.9).

Position of the oval burr and arthroscope are now switched over, respectively, from lateral to posterior portal and vice

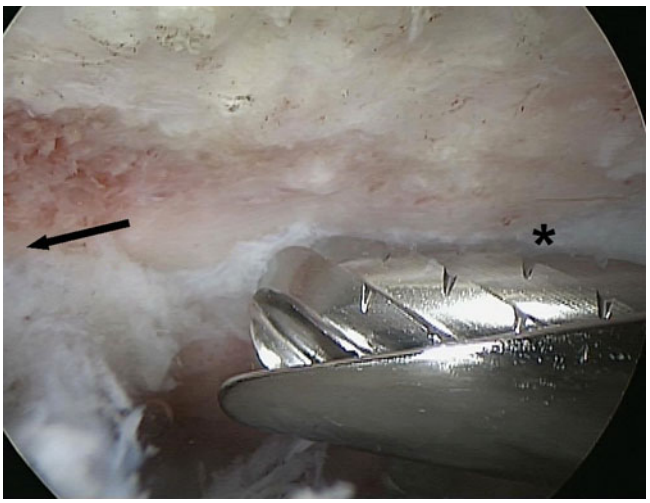

Fig. 21.7 Right shoulder. Acromioplasty is performed starting from the anterolateral corner of the acromion (*asterisk*) towards the acromion-clavicular joint (*arrow*). The anteroinferior portion of the acromion is removed using an oval burr. The arthroscope is in the posterior portal

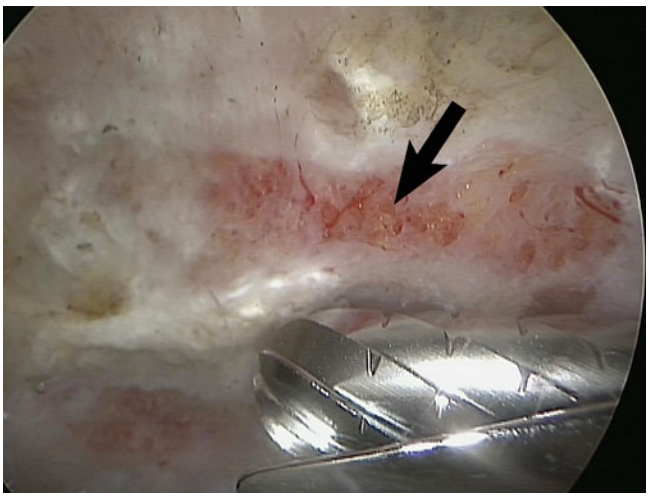

Fig. 21.9 Right shoulder. A 3 mm deep groove (*arrow*) across the whole width of the acromion is performed in line with the posterior aspect of the acromioclavicular joint. This groove is used as a reference for the posterior resection of the undersurface of the acromion. The arthroscope is in the posterior portal

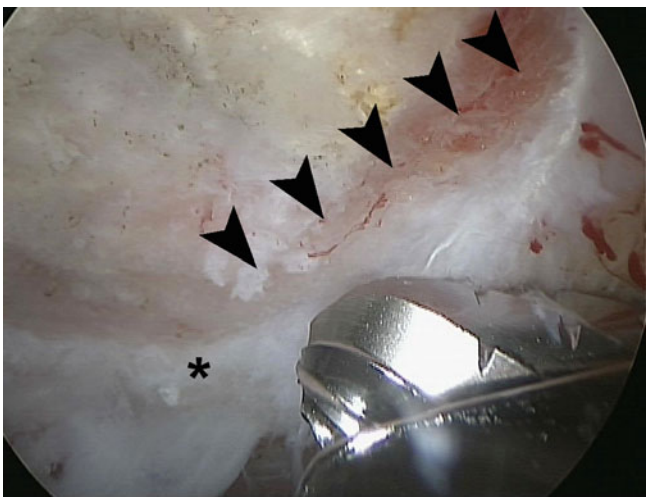

Fig. 21.8 Right shoulder. The oval burr is moved from the anterolateral corner (*asterisk*) to the lateral edge of the acromion (*arrow heads*). The deltoid insertion site must be preserved. The arthroscope is in the posterior portal

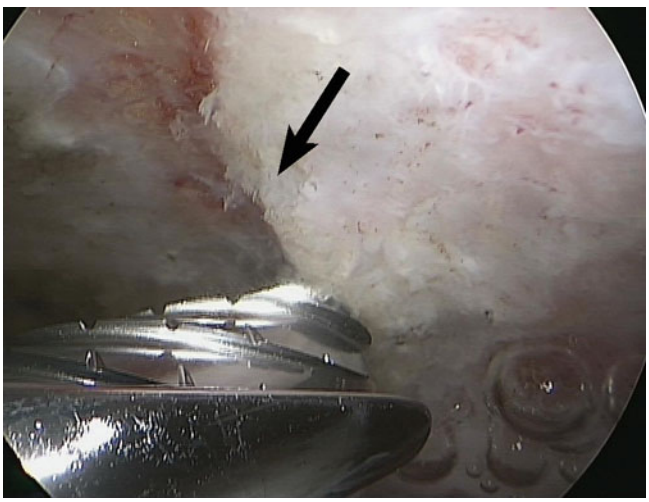

Fig. 21.10 Right shoulder. The oval burr is inserted in the posterior portal and used to perform tangential acromioplasty. The acromion is burred starting from the edge of the posterior reference groove (*arrow*), from posterior to anterior, from medial to lateral. The arthroscope is in the lateral portal

versa. The acromion is then burred or smoothed starting from the edge of the posterior reference groove, moving from medial to lateral till the whole inferior surface of the acromion is smoothed or burred. The burr must be kept close against the posterior portion of the inferior surface of the acromion; this will make tangential resection of the inferior surface of the acromion possible in order to obtain a smooth flat acromial profile (Fig. 21.10).

Once resection is complete, the arthroscope is positioned posteriorly to allow for better visualization and for the removal, where needed, of any residual roughness of the anterolateral corner of the acromion, which is difficult to see from the lateral portal (Fig. 21.11).

The arthroscope is eventually pointed towards the AC joint and the distal clavicle. If there is a significant degenerative change or an inferior osteophytosis of the clavicle, the inferior surface of the distal clavicle can be resected. The AC joint capsule is resected with a radio-frequency instrument. A shaver will then be used to perform the tangential resection of the inferior portion of the joint or the resection of the distal end of the clavicle (see Chap. 30).

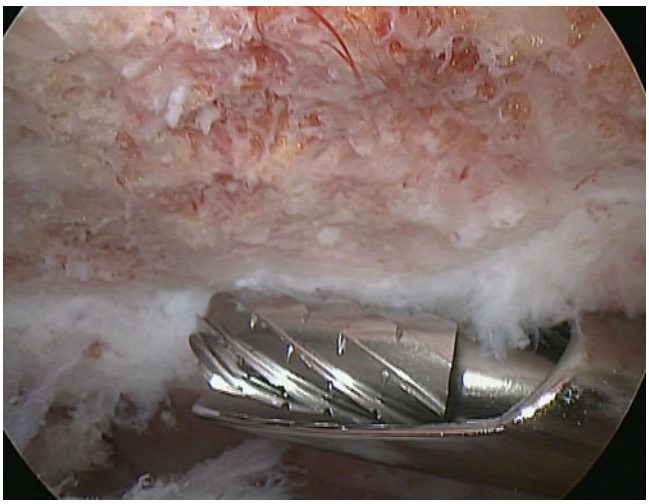

Fig. 21.11 Right shoulder. Acromioplasty is completed, the arthroscope is positioned again posteriorly and residual roughness of the anterolateral corner of the acromion are addressed with the oval burr from the lateral portal

Postoperative Care

Following isolated subacromial decompression, the arm is placed in a sling for 7–10 days (until stitches are removed), although pendulum exercises are allowed the first day after surgery. One of the postoperative complications is the development of scar adhesions which can limit the range of motion; therefore, it will be important to keep the postoperative immobilization period as short as possible and refer the patient for an early rehabilitative treatment aiming at an early recovery of passive range of motion.

If subacromial decompression has been associated with rotator cuff repair, immobilization will last longer to guarantee an adequate protection of the repair (see Chap. 23). After the sling is removed, rehabilitation program will be articulated in three phases (see Chap. 6), each lasting for 4 weeks:

- Phase one: prevention of scar adhesions, full recovery of passive, active-assisted, and active range of motion
- Phase two: closed kinetic chain exercises to strengthen the rotator cuff, the subscapularis tendon, and the scapular stabilizers
- Phase three: open kinetic chain exercises, proprioceptive and plyometric exercises, and postural rehabilitation of the kinetic chain (lumbopelvic, lumbar thoracic, scapulothoracic)

For athletes, this program will be followed by a training to recover the specific athletic gesture based on reprogramming of specific muscle activation patterns.

Return to sports activities and heavy manual work is allowed 6 months after surgery.

Literature Review

Subacromial impingement is the most common diagnosis of shoulder pathologies. First line treatment is always conservative based on oral NSAIDs, change in activities, subacromial injections, and/or specific exercise program and physical therapy. Surgery is taken into consideration only when conservative treatment fails. Actually, comparative studies on conservative and surgical treatment have shown overlapping results, with 65–80 % successful outcome 1 year after treatment [89–93]. These results obviously raise doubts about the necessity of surgical treatment. Holmgren et al. [94] performed a prospective study on 102 patients who suffered from subacromial impingement not responding to 6-month conservative treatment and who were, therefore, put on a waiting list for arthroscopic subacromial decompression. Patients took part in two physiotherapy protocols: one protocol was specific and focused on eccentric strengthening exercises for the rotator cuff and on eccentric and concentric exercises for the scapular stabilizers; the other protocol was unspecific (control group) and based on exercises for the neck and shoulders. Patients who underwent a specific rehabilitation protocol showed a significant improvement as regards pain and shoulder function compared to controls after 12-week treatment; moreover, 80 % of the specific exercise group opted out of surgery. These results support the intrinsic theory of subacromial impingement pathogenesis and claim that an appropriate rehabilitation strategy based on scapular muscle strengthening and on the re-equilibration of force couples for scapular control is sufficient to reduce symptoms induced by a degenerative disease of the rotator cuff. On the other hand, subacromial decompression following unsuccessful conservative treatment has its rationale in the extrinsic theory, which considers acromial morphology and the compression exercised by the coracoacromial arch as the major determining factor of rotator cuff tendinopathy.

Disadvantages associated with acromioplasty and sectioning of the coracoacromial ligament consist of weakening of the insertion of the deltoid muscle [95], scar formation in the subacromial space that can limit shoulder mobility [96], and risk of anterior-superior glenohumeral instability, especially in patients with irreparable rotator cuff tears [97, 98].

A recent literature review comparing acromioplasty versus isolated bursectomy showed that there are no significant differences between the two treatments [99].

Budoff et al. [100] reported on 79 cases with partial-thickness rotator cuff tears treated by cuff debridement without decompression. At an average 53-month follow-up, they observed 87 % of good or excellent results. At a longer follow-up (average 9.5 years), the same cohort showed satisfactory results in 79 % of the cases [101].

Gartsman and O'Connor [102] reported a prospective randomized study on 93 patients with an isolated full-thickness tear of the supraspinatus and type 2 acromion that were treated with arthroscopic rotator cuff repair with and without subacromial decompression. At an average 15.6-month follow-up, they did not observe significant difference in clinical outcome.

Milano et al. [103], in a prospective randomized study on 80 patients, compared arthroscopic rotator cuff repair with and without subacromial decompression. They showed that subacromial decompression did not significantly affect the clinical outcome of rotator cuff repair. More recent clinical trials [104, 105] confirmed the same findings, albeit McDonald et al. [104] found a higher reoperation rate in the group without acromioplasty. A recent literature review [106] confirmed no significant role of subacromial decompression on the clinical outcome of full-thickness rotator cuff repair.

Summary

Two different etiopathogenetic theories can explain the development of subacromial impingement: an intrinsic theory and an extrinsic theory. The intrinsic theory states that impingement is determined by a primary damage to the rotator cuff tendons (degenerative, vascular, or microtraumatic causes). According to the extrinsic theory, impingement is determined by alterations in the morphology of the coracoacromial arch. Biomechanical factors altering the scapulohumeral kinematics are secondary causes of impingement. An accurate clinical and imaging evaluation is crucial for establishing a correct diagnosis of impingement and for an appropriate therapeutic approach. Conservative treatment is the first-line treatment. Subacromial decompression is indicated after failure of conservative treatment. Bursectomy is often sufficient to eliminate symptoms. Acromioplasty should be limited to accurately selected cases.

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Partial-Thickness Rotator Cuff Tears

22

Rachel M. Frank, Jas Chahal, and Nikhil N. Verma

Epidemiology

Rotator cuff repair is one of the most common procedures performed in orthopedic surgery. The goal of repair is to restore normal rotator cuff kinematics in an effort to improve glenohumeral function and reduce pain. Recent data has revealed that healing and anatomic integrity of the rotator cuff repair site correlates with improved outcomes, particularly with regard to strength and functional recovery [1–6]. Rotator cuff pathology exists on a wide spectrum that ranges from cuff tendon edema and inflammation to partial- and full-thickness tears of one or more of the cuff tendons. Recent advances in diagnostic imaging modalities have led to an increase in clinician recognition of partial-thickness rotator cuff tears [7–9]. As elegantly described by Clark and Harryman [10], the anatomy of the rotator cuff insertion onto the humerus is complex as the tendons, articular capsule, coracohumeral ligament (CHL), and glenohumeral ligament complex essentially blend to form a confluent, layered sheet prior to insertion onto the tuberosities. On the articular side, the deepest layer of the cuff is reinforced at its attachment by the joint capsule. Understanding the tendon footprint anatomy is important, as the majority of older patients experience partial-thickness rotator cuff tears on the articular side of the supraspinatus tendon near its insertion on the greater tuberosity while younger, overhead-throwing athletes are more likely to experience partial-thickness rotator cuff tears at the supraspinatus-infraspinatus interval [9, 11].

Partial-thickness cuff tears are typically classified as articular sided, bursal sided, or interstitial (intratendinous). In 1990, Ellman [12] introduced a classification system for partial-thickness rotator cuff tears as seen arthroscopically. In this system, partial-thickness tears are classified by *location*: (A) articular surface, (B) bursal surface, or (C) interstitial; and *depth* (I) <3 mm, (II) 3–6 mm, or (III) >6 mm [12]. As the mean tendon insertion footprint of the supraspinatus is approximately 12–14 mm, Grade III (high grade) tears typically represent tears of greater than 50 % of the tendon thickness [12, 13]. Several eponyms have also been described in an attempt to further classify partial-thickness lesions, including the PASTA lesion, or partial articular supraspinatus tendon avulsion, as described by Snyder et al. [14], and the PAINT lesion, or partial articular tear with intratendinous extension, as described by Conway [15].

While the true incidence of partial-thickness rotator cuff tears is unknown, most authors agree that the vast majority of these tears occur in the supraspinatus tendon. Cadaveric data has demonstrated a substantially higher number of partial-thickness supraspinatus tears relative to full-thickness tears [16–19]. Interestingly, the majority of basic science studies describe intratendinous tears as more common than bursal or articular-sided tears, while the majority of clinical studies report on the substantially higher occurrence of articular-sided tears. Payne and colleagues found that 91 % of all partial-thickness tears were classified as articular sided in a population study of young athletes [20]. Articular-sided tears may be more common than bursal-sided tears due to the relative hypovascularity of the articular side of the cuff, as demonstrated by Lohr and Uhthoff [21]. This discrepancy between the clinical and basic science studies is likely multifactorial and related to the difficulty in actually diagnosing intratendinous tears clinically as well as the trouble in interpreting pathology on cadaveric specimens, which are typically much older than the patient population encountered in most clinical settings [7]. Further, it is important to recognize which partial-thickness cuff tears are clinically symptomatic, and which are simply incidental in nature, as multiple imaging studies have demonstrated the existence of both partial and full-thickness rotator cuff tears in asymptomatic individuals [22–25].

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Table 22.1 Intrinsic and extrinsic factors leading to partial-thickness rotator cuff tears

Intrinsic	
Age	Decreased cellularity, fascicular thinning, granulation tissue, dystrophic calcification, and decreased vascularity
Vascular	Relative hypovascularity of articular side of cuff, especially near insertion of supraspinatus on humerus
Histology	Thinner collagen bundles on articular side than bursal side leading to decreased ultimate stress to failure and increased "ease" of tear with lower-energy trauma on the articular side
Intratendinous lesions	Increased intratendinous strain especially at increasing amounts of abduction, leads to propagation of tear: Especially important in overhead athletes Often due to shear stress within the tendon itself between the deep and superficial layers
Extrinsic	
Subacromial impingement	Impingement of cuff tendons on subacromial osteophytes and/or coracoacromial ligament: More often leads to bursal-sided tears
Internal impingement	Internal impingement resulting from repetitive contact between the posterosuperior aspect of the glenoid and the undersurface of the cuff: Multifactorial Results from repetitive microtrauma during eccentric contraction during deceleration phase of throwing Associated with posterior capsular tightness Especially in overhead throwing athletes
Traumatic	Acute traumatic injury and/or chronic shoulder instability

Pathophysiology

The natural history of partial-thickness rotator cuff tears seems to involve a process of progressive extension of cuff pathology due to intrinsic and/or extrinsic factors. Intrinsic etiologies include biologic factors (age-related metabolic and/or vascular changes) leading to degeneration as well as interstitial lesions as a result of chronic shear stress. Extrinsic etiologies include acute traumatic injury, shoulder instability, repetitive microtrauma, subacromial impingement, and internal impingement. Many partial-thickness tears occur as a result of both intrinsic and extrinsic factors. The anatomic location of the tear, as determined by imaging and/or arthroscopy, may help determine the etiology of the lesion (Table 22.1).

As mentioned above, older patients are more likely to experience partial-thickness rotator cuff tears on the articular side of the supraspinatus tendon near its insertion on the greater tuberosity due to intrinsic tendinopathy from degenerative cuff changes, while younger, overhead-throwing athletes are more likely to experience articular-sided partial-thickness rotator cuff tears at the supraspinatus-infraspinatus interval [9, 11]. Bursal-sided tears may be more related to extrinsic factors including coracoacromial arch narrowing, impingement on the cuff from the distal clavicle, as well as trauma. As demonstrated in a cadaveric model by Ozaki and colleagues, the undersurface of the acromion is typically pathologic in bursal-sided tears while it is nearly always intact in shoulders with articular-sided tears [26]. Understanding the pathogenesis of partial-thickness rotator cuff tears is important as the specific pathology may dictate the treatment of choice. For example, in young, overhead-throwing athletes with partial-thickness cuff tears, it is important to recognize which factors are pathologic and which factors are adaptive to allow for repetitive throwing at a high level. Some factors may even be physiologic, such as decreased humeral retroversion, and may ultimately lead to poor throwing mechanics, internal impingement, and ultimately cuff tendinopathy and/or tendon tears.

Based on studies analyzing full-thickness tears, factors including patient age [1, 2, 4, 6], tear size [1, 2, 6, 27], muscle atrophy and fatty change [28, 29], chronicity [30], and smoking [31, 32] are all associated with worse outcomes. As described in detail above, partial articular-sided rotator cuff tears, while multifactorial, likely progress due to a lack of adequate healing response due to the relative hypovascularity within this region of the cuff. As such, these partial-thickness tears may subsequently progress to full-thickness tears. Ultimately, these factors contribute to the biologic properties of the tendinous footprint, and the ideal repair construct leads to biological healing of tendon to the footprint. Factors involved in optimal healing include footprint coverage, contact pressure, suture strength, loop and knot security, decreased motion at bone-tendon interface, and maximization of the biological response. Recently a systematic review analyzing published literature involving arthroscopic repair of partial-thickness rotator cuff tears demonstrated a progression rate of 6–35 % to full-thickness tears following debridement of tears less than 50 % with or without concomitant acromioplasty [11].

History

A thorough history, physical exam, and imaging studies are necessary to determine the best treatment options for a patient with suspected partial-thickness rotator cuff pathology. The

history must include assessment of the patient's age, functional status, expectations, activity level, and comorbidities. Mechanism of injury as well as previous episodes of shoulder pain, injury, or previous surgeries must be determined. Patients may complain of some or all of the following:

- Pain that is typically insidious in onset, over lateral arm and shoulder, radiating to deltoid insertion:
  - Possible history of trauma
- Pain that is often dull at rest
- Pain that often occurs at night, may awaken from sleep
- Overhead activities make it worse
- Difficulty with some activities of daily living including washing hair, holding hair dryer, and reaching back pocket
- Weakness in positions of abduction and/or forward flexion

Clinical Examination

Following a complete history, a focused physical examination of both shoulders is necessary in evaluating the patient with suspected rotator cuff pathology. Often, patient complaints are vague and nonspecific, and the clinician must utilize examination findings in conjunction with imaging studies to arrive at the diagnosis. Isolated rotator cuff pathology is difficult to ascertain on exam, and findings may be representative of other shoulder lesions, including adhesive capsulitis, impingement, acromioclavicular joint pain, and anterior, posterior, and/or multidirectional instability. Further, cervical spine pathology may mimic rotator cuff pathology. It is thus imperative to perform a complete examination of the shoulder and evaluate for other potential concomitant symptomatic lesions. As in any shoulder examination, the structure, function, neurological status, and strength of the injured shoulder should be compared to the opposite shoulder. If significant stiffness is noted, range of motion must be optimized prior to any operative procedure to avoid progressive loss of motion. Some specific physical examination findings in patients with partial-thickness rotator cuff pathology include:

- Supraspinatus atrophy
- Subacromial crepitation
- Decreased active ROM
- Tenderness to palpation over greater tuberosity
- Isolated rotator cuff muscle weakness:
  - Loss of muscle strength with resolution of pain after subacromial injection is consistent with a full-thickness cuff tear, while maintenance of strength with resolution of pain is more consistent with cuff inflammation and/or partial thickness tear.
- Possible liftoff or belly press for subscapularis tears:
  - Note these may be negative in high-level athletes.
- Associated pathology:
  - Biceps tendinopathy:
    - Speed test
    - Yergason's test
  - Impingement:
    - Hawkin's test
    - Neer's test
  - SLAP lesions:
    - O'Brien's test

Imaging

Diagnostic imaging is critical in the evaluation of patients with suspected rotator cuff pathology [33]. While plain radiographs are not helpful in the actual evaluation of the rotator cuff tendons, radiographs should be obtained to evaluate the patient's anatomy and to look for other potential causes for shoulder pain. A standard shoulder series including an anterior-posterior view of shoulder in the plane of the scapula, scapular Y view, and axillary view should be performed. In the latter, one can assess for the presence of a symptomatic os acromiale. A supraspinatus outlet view can also be performed to help evaluate the morphology of the coracoacromial arch and the osseous geometry of the acromion itself. Other specialized views can be obtained as indicated, including a Stryker notch view and/or West Point view to assess for Hill-Sachs lesions and/or glenoid bone loss, respectively.

Ultrasound is a very useful imaging modality in evaluating the rotator cuff integrity and is noninvasive, inexpensive, and available [34–40]. Nevertheless, this modality is completely operator dependent, and thus results can vary. The reported sensitivity and specificity of ultrasound for the diagnosis of partial-thickness rotator cuff tears are as high as 94 and 93 %, respectively [37]. However, other studies have shown a detection rate of only 41 % [34]. Recently, Ok and colleagues have demonstrated a comparable ability of ultrasound and magnetic resonance arthrography (MRA) to diagnose full-thickness cuff tears; however, ultrasound was less accurate for detecting partial-thickness tears and tear size itself [41]. A recent systematic review reported similar findings, stating ultrasound is superior in the detection of full-thickness tears when compared to partial-thickness tears [42].

Magnetic resonance imaging (MRI) and MRA [43–45] are comparable to ultrasound in the diagnosis of full-thickness rotator cuff tears and are superior in the evaluation of partial-thickness cuff tears. Partial-thickness tears will show increased signal in the cuff, without discontinuity, on T1-weighted images with corresponding signal increases on T2-weighted images. These signal changes are associated with identification of a lesion on the bursal or articular surface or within the tendon itself. The use of contrast enhancement increases the diagnostic ability of the MR over conventional MRI. A recent systematic review reported an overall sensitivity and specificity value of 80 and 95 %, respectively, for partial-thickness rotator cuff tears when using MRI [43]. A significant advantage of MRI/MRA is the ability to diagnosis

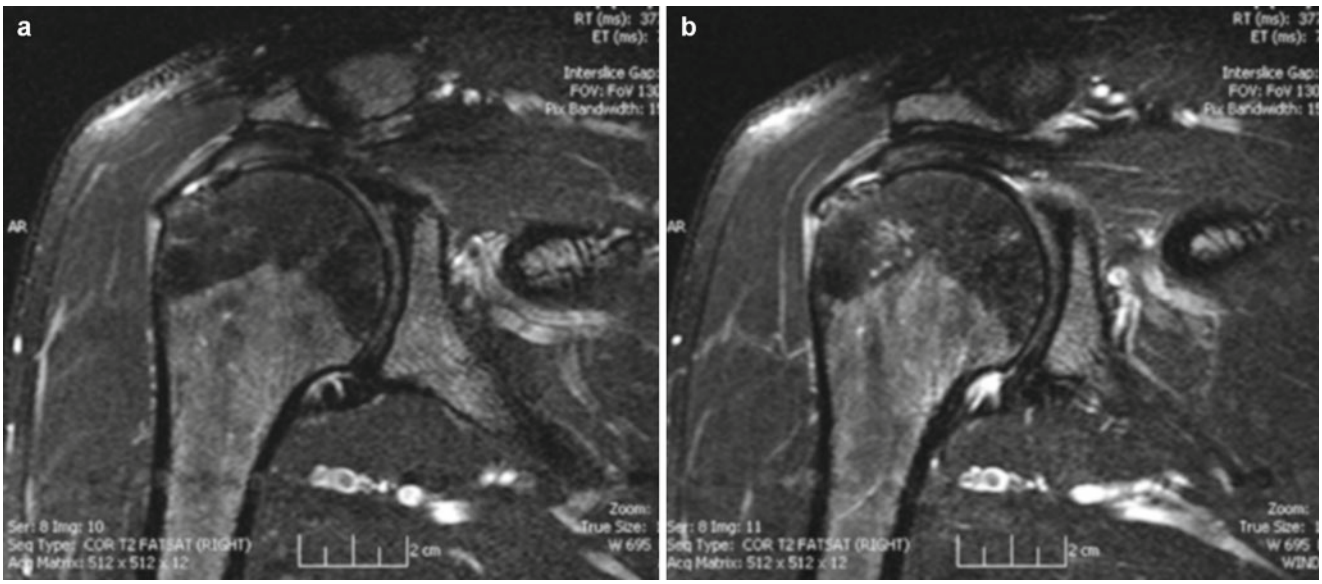

Fig. 22.1 T2-weighted MRI figures demonstrating a partial-thickness rotator cuff tear involving the supraspinatus tendon (coronal view) (**a**) and (**b**) sequential cuts of right shoulder coronal T2-weighed images with fat-saturation

concomitant pathologies, including labral tears, chondral lesions, and biceps tendon tears, among other common pathologies. The literature reports sensitivities up to 100 % for these modalities in the evaluation of partial-thickness cuff tears. MRI/MRA is extremely helpful in the assessment of tear retraction and degeneration of muscle, which ultimately affects surgical decision-making (Fig. 22.1).

Diagnostic arthroscopy remains the gold standard; however, it is obviously more invasive than any of the previously mentioned imaging modalities.

Treatment: Indications and Contraindications

In general, young patients presenting with acute traumatic weakness that can be attributed to a rotator cuff tear should be managed with early operative management while older patients can undergo a trial of initial nonoperative treatment. Other indications and contraindications include:

- Indication: persistent pain unresponsive to nonoperative measures
- Relative indication:
  - Poor function and diminished strength
  - Ability to comply with rehabilitation program
  - Medically fit for surgery
- Contraindications:
  - Active or recent infection
  - Medical comorbidities that preclude surgery
  - Advanced GH arthritis
- Relative contraindications:
  - Significant muscle degeneration
  - Fixed superior migration of humeral head
  - Significant stiffness (loss of passive ROM)

Decision-Making Algorithm

The decision-making algorithm for partial thickness rotator cuff tears can be challenging for even the most experienced of shoulder surgeons. In general, tears involving less than 50 % of the tendon width can be addressed with good outcomes. This can be accomplished with or without a formal acromioplasty. With tears involving greater than 50 % the width of the tendon, surgical options become more varied. Techniques including tear completion and repair, transtendinous repair, and transosseous repair have been described, also with good outcomes. Regardless of the technique of choice, patient-specific variables must be accounted for, including:

- Patient functional status, age, expectations, and comorbidities
- Associated pathologies (soft tissue injuries, bony involvement)
- Chronicity and quality of tear and morphologic features of tear

Arthroscopic Treatment: Surgical Technique

Patient Positioning

After an interscalene regional nerve block in the preoperative holding area, the patient is transported to the operating room. A combined regional anesthetic with conscious sedation is used as the preferred anesthesia during the entirety of the surgical procedure. Once sedated, the patient is positioned in the

modified beach chair position, and the operative limb is placed in a pneumatic arm holder to facilitate intraoperative limb positioning and movement during the case (Spider, Smith and Nephew, Andover, MA). The arm holder can be especially valuable when only a single surgical assistant is available.

Portals

A standard posterior viewing portal is established to perform a diagnostic arthroscopy. We prefer to place this portal 1 cm distal and medial to the posterolateral corner of the acromion. For diagnostic arthroscopy, a standard anterior portal is established using an outside-in technique just lateral to the coracoid process, and a 6 mm cannula is inserted.

In the context of a rotator cuff repair, the anterior portal described above can be used as an anterolateral working portal once in the subacromial space; a switching stick can be utilized to insert a 6 mm cannula when a repair is to be performed. A lateral portal that bisects the acromion is also established and an 8.25 mm cannula is inserted for instrumentation, passing sutures, and knot tying. Finally, several percutaneous portals are utilized as necessary for anchor insertion throughout the case.

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

Identifying associated pathology will help optimize outcomes following treatment of partial thickness rotator cuff tears. As such, the diagnostic arthroscopy involves a step-by-step examination of all pertinent intra-articular and subacromial anatomical structures. The goal is to identify all possible pain/symptom generators and see if the observed findings are concordant with the concerns that the patient initially presented with.

Specifically, the surgeon must examine glenoid and humeral chondral surfaces for possible osteochondral injury; the long head biceps pulley and tendon for evidence of instability, tears, or inflammation ("lipstick" sign); the labrum for degenerative or unstable tear patterns; capsular and synovial irritation; and loose bodies. The arthroscope must alternately be placed into anterior and posterior portals to afford a comprehensive evaluation [46]. In regards to evaluating rotator cuff pathology, partial articular-sided tears of the supraspinatus are routinely evaluated from the posterior portal, while those of the infraspinatus are viewed from the anterior portal [46]. The bare area of the posterosuperior humeral head defines the interval between the supraspinatus and infraspinatus when viewing the intra-articular tendon footprint. The subscapularis insertion can be viewed from the posterior portal while a manual posterior force is applied to the proximal humerus; alternatively, a 70° arthroscope can be utilized from the posterior portal to view the insertion of the subscapularis tendon. The diagnosis of internal impingement can be made with a dynamic evaluation – when the shoulder is placed in the abducted and externally rotated (throwing) position, a partial articular-sided tear of the posterior aspect of the supraspinatus abuts the posterosuperior glenoid and adjacent labrum [46–48].

In the subacromial space, a thorough anterior, posterior and lateral bursectomy is performed to optimize visualization of the entire bursal surface of the rotator cuff, the acromion, coracoacromial ligament (CAL), and acromioclavicular (AC) joint. Prominent spurs on the undersurface of the acromion should be smoothed. Areas of focal rotator cuff injury or hyperemia may suggest subacromial impingement, which can be treated with subacromial decompression. If the patient has AC joint-related symptoms, then a concomitant AC joint resection may also be warranted. In general, the need to perform an acromioplasty alongside rotator cuff debridement or repair is left to the discretion of the surgeon. In a meta-analysis conducted by Chahal et al., there was no difference in short-term functional outcome scores or reoperation rates among patients treated with and without an acromioplasty in the context of arthroscopic repair of full-thickness rotator cuff tears [49]. Longer-term follow-up is required to determine if acromial morphology may contribute to recurrent pain or tearing.

It is important to note that intratendinous tears can be difficult to identify due to relatively normally appearing bursal and articular surfaces. Firm palpation of suspected areas of pathology with a probe may suggest subsurface irregularity, and unroofing of the tear with a shaver may be necessary for full appreciation of cuff pathology [46]. Correlation with preoperative imaging studies to identify the region of abnormality is required.

Step-by-Step Procedure (Box 22.1)

Box 22.1: Tips and Tricks

- Debridement with a full-radius synovial resector will remove damaged tissue but will not violate healthy tendon fibers
- For transtendon repairs
  - The shoulder should be kept in the adducted position while introducing anchors and instruments to avoid iatrogenic injury to the articular surface [46]
  - Introduce the spinal needle for the shuttle suture from lateral to medial and almost parallel to the footprint. Entering too far medial on the bursal surface will create a 'rent' when sutures are tied [46]
  - Try to pass sutures such that a triangular configuration is created – allowing for recreation of the normal insertion footprint

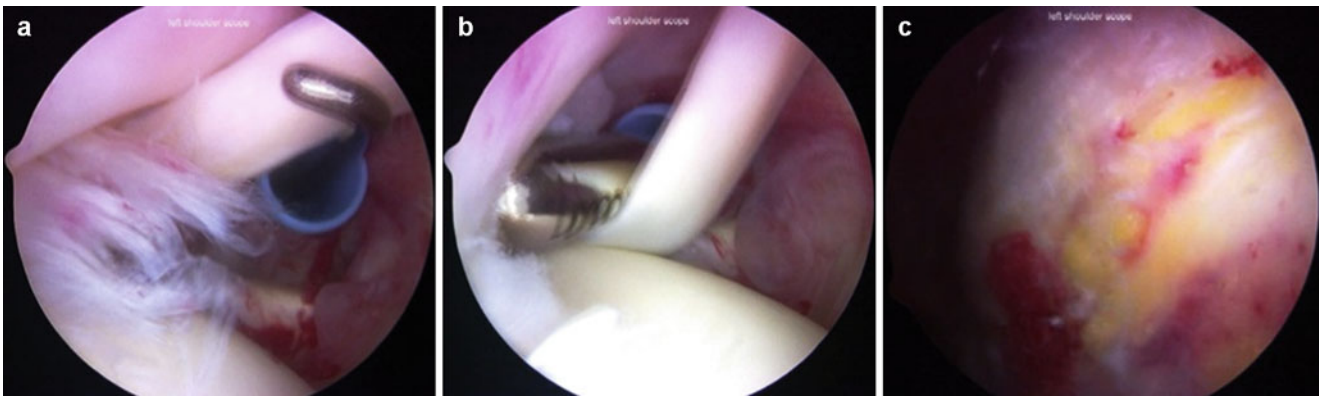

Fig. 22.2 Arthroscopic images of a left shoulder demonstrating partial-thickness rotator cuff tear (**a**), debridement of the tear (**b**), and preparation of the surface to a bleeding surface (**c**)

Prior to the diagnostic arthroscopy, an examination under anesthesia is performed to document associated shoulder instability and possible deficits in range of motion in all planes. Such findings are taken into consideration if correlative pathology is observed during the diagnostic arthroscopy. Hypotensive anesthesia (systolic blood pressure approximately 100 mmHg) will minimize bleeding and improve visualization [46].

Our preferred approach to the surgical management of patients with partial-thickness rotator cuff tears is guided by an evidence-based treatment algorithm developed at Rush University and published by Strauss et al. [11]. The first step in treatment involves an inspection of the rotator cuff footprint on the articular side. Any irregular and frayed areas of tendinous attachment is probed and debrided with an arthroscopic shaver. Measurement of the exposed footprint in conducted using a probe to calculate the percentage of tendon involved. Following this, the suspected site of partial-thickness rotator cuff tearing is marked with a monofilament suture using a percutaneously inserted 18-guage spinal needle (from the anterolateral corner of acromion). The arthroscope is then advanced into the subacromial space, and the bursal side of the rotator cuff is examined to rule out a full-thickness defect. In the absence of articular sided pathology, the bursal side of the rotator cuff is also examined to look for partial-thickness tearing.

According to the Ellman classification [12] of partial-thickness rotator cuff tears, the depth of the tear is then determined. Tears that involve less than 50 % of the tendon thickness are managed with debridement alone [11]. Biomechanical evidence supporting debridement of tears <50 % and repair of tears greater than 50 % has recently been presented [1]. For tears that are more than 50 % of tendon thickness, the literature would support a repair using any one of the following techniques: (1) completion of the tear with subsequent repair, (2) transtendinous repair, and (3) transosseous repair [11]. Our preferred approach includes a transtendinous technique for partial articular sided tears; for bursal tears or high-grade articular sided tears (>80 % thickness), we prefer to complete the rotator cuff tear and repair the resultant full-thickness defect.

Debridement (Fig. 22.2)

For both articular and bursal-sided tears, a full-radius synovial resector is used to debride the tear until healthy tissue margins are created. Subsequently, an instrument of known size is used to estimate tear depth as well as the anteroposterior dimension of the tear in order to confirm whether debridement alone will be a sufficient treatment.

Transtendinous Repair (Fig. 22.3)

Transtendinous repairs are ideally indicated for articular surface tears of the supraspinatus that involve more than 50 % of tendon thickness. Following debridement of the articular-sided repair, a thorough subacromial bursectomy, and examination of the bursal surface of the rotator cuff, the arthroscope is reintroduced into the glenohumeral joint. Precise portal placement is vital – typically 6 and 8.25 mm cannulas are inserted: the 6 mm in the anterosuperior position within the rotator interval and the larger cannula in the lateral position within the subacromial space. Greater tuberosity preparation is performed with an arthroscopic bone shaver (BoneCutter, Smith and Nephew, Andover, MA). Percutaneous placement of double-loaded suture anchors is accomplished through the intact bursal portion of the rotator cuff into the medial portion of the greater tuberosity. Specifically, an awl or tap is passed through the intact rotator cuff tendon fibers to the central aspect of the articular margin of the exposed greater tuberosity defect. A 4.5 mm double-loaded suture anchor is then passed through the intact portion of the rotator cuff and screwed into the greater tuberosity while surgeon in visualizing the joint. We prefer the use of PEEK material to prevent cystic reaction during anchor resorption while allowing for postoperative imaging. A single anchor is used for tears 1.0–1.5 cm or less in the anteroposterior direction, and two anchors are used for tears larger than this [50]. One limb of

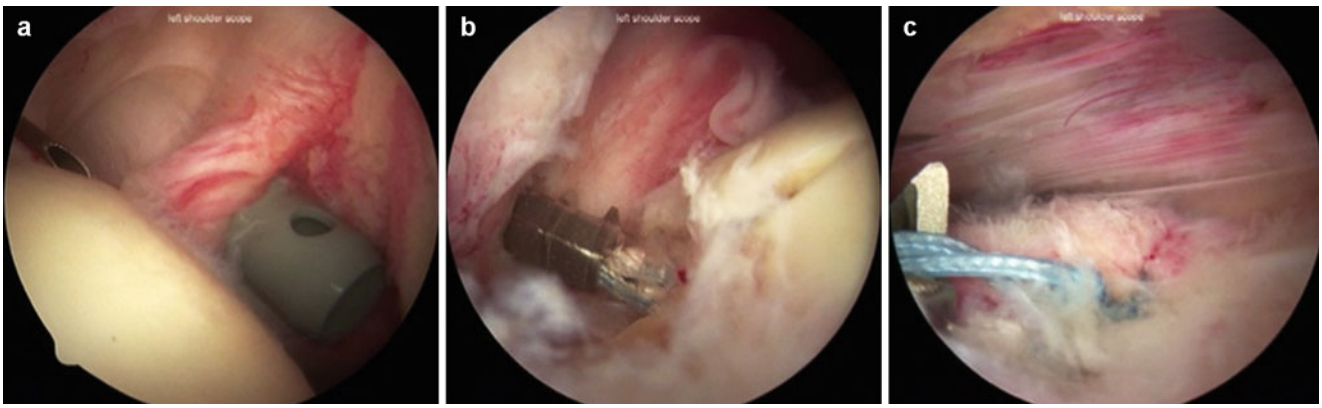

Fig. 22.3 Arthroscopic images of a left shoulder demonstrating PASTA lesion (**a**), use of a 4.5 mm double-loaded fully threaded PEEK medial anchor (Twinfix, Smith and Nephew, Andover, MA) suture anchor (**b**), and PASTA lesion after suture anchor repair (**c**)

suture from each pair is retrieved through anterior working cannula. After initial localization with a spinal needle, a tissue penetrator is passed through the anterolateral portal 1 cm medial to the intact margin of the rotator cuff crescent [51]; subsequently, 1 suture limb from the anterior portal is retrieved and pulled through the intact healthy portion of the rotator cuff and pulled out through the anterolateral portal. Care must be taken to avoid taking an excessive bite of tissue, which may result in over-tensioning of the cuff tendon. The second suture limb is then retrieved in a mattress fashion approximately 5 mm posterior to the first.

For the second pair of sutures, the tissue penetrator is passed for a second time in a similar fashion but 5–7 mm posterior to the first suture, and the second suture is pulled through. Alternatively a spinal needle can be passed through the rotator cuff and a monofilament suture used as a shuttle to individually pass sutures through the torn tendon where they are retrieved in the subacromial space. Suture limbs are retrieved in the subacromial space and are tied in standard fashion; the suture passed through the intact rotator cuff is used as a post [51]. It is important to try to pass sutures such that a triangular configuration is created – allowing for recreation of the normal insertion footprint [11]. After the arthroscopic knots are tied in the subacromial space, the arthroscope should be reintroduced into the glenohumeral joint to evaluate the repair. It is critical to complete the subacromial bursectomy prior to suture passage to facilitate suture retrieval and knot tying after suture passage.

Completion of Tear with Repair (Fig. 22.4)

At our institution, this technique is utilized for high-grade partial bursal-sided rotator cuff tears and occasionally for high-grade (greater than 80 %) articular-sided tears. Following debridement, the tear is marked with a monofilament suture during visualization in the joint, the area of the marking suture or the region of partial bursal tear is identified in the subacromial space, and the tear is completed on the bursal surface with an 11-blade scalpel or beaver blade and debridement of degenerative tendon tissue is performed with an arthroscopic shaver. Completion and mobilization of the torn rotator cuff are confirmed with an arthroscopic probe, and the greater tuberosity is prepared with a bone-cutting shaver (Smith and Nephew, Andover, MA). Once a full-thickness defect is created, our preferred repair technique is to perform a transosseous equivalent repair. Once again, precise portal placement is vital – typically a single 8.25 mm cannula is inserted in the lateral position within the subacromial space

When performing a transosseous equivalent repair, the first step is to confirm tension free reduction of the torn tendon edge anatomically to the lateral edge of the rotator cuff footprint of the greater tuberosity. As discussed, we use a bone-cutting shaver to prepare the greater tuberosity in order to create a bed for bleeding. The first fully threaded 4.5 mm PEEK medial anchor (Twinfix, Smith and Nephew, Andover, MA) is percutaneously placed 2–3 mm posterior to biceps (for supraspinatus tears) and 5 mm lateral to articular surface. Sutures are then retrieved through lateral cannula. Using a spectrum device (Linvatech, Largo, FL), an indirect suture passing technique is undertaken in order to pass sutures through the rotator cuff. Specifically, suture passage is performed with a horizontal mattress configuration taking approximately 12 mm bites of tissue. In doing so, the scope is placed in an accessory lateral portal for visualization, while the spectrum is placed via the posterior portal for suture passage. Sutures should be placed just lateral to the musculotendinous junction, which will restore the normal anatomic footprint. As the sutures are passed, they are then shuttled from the lateral working portal to an anterior storage portal moving sequentially from posterior to anterior direction along the rotator cuff. Furthermore, sutures are placed 4–5 mm apart in the horizontal mattress configuration. Additional anchors are inserted as needed 4–5 mm off articular surface, and the aforementioned steps are repeated. Alternatively a direct suture-passing device (Elite-Pass, Smith and Nephew, Andover MA) can be used via the lateral cannula to pass sutures sequentially

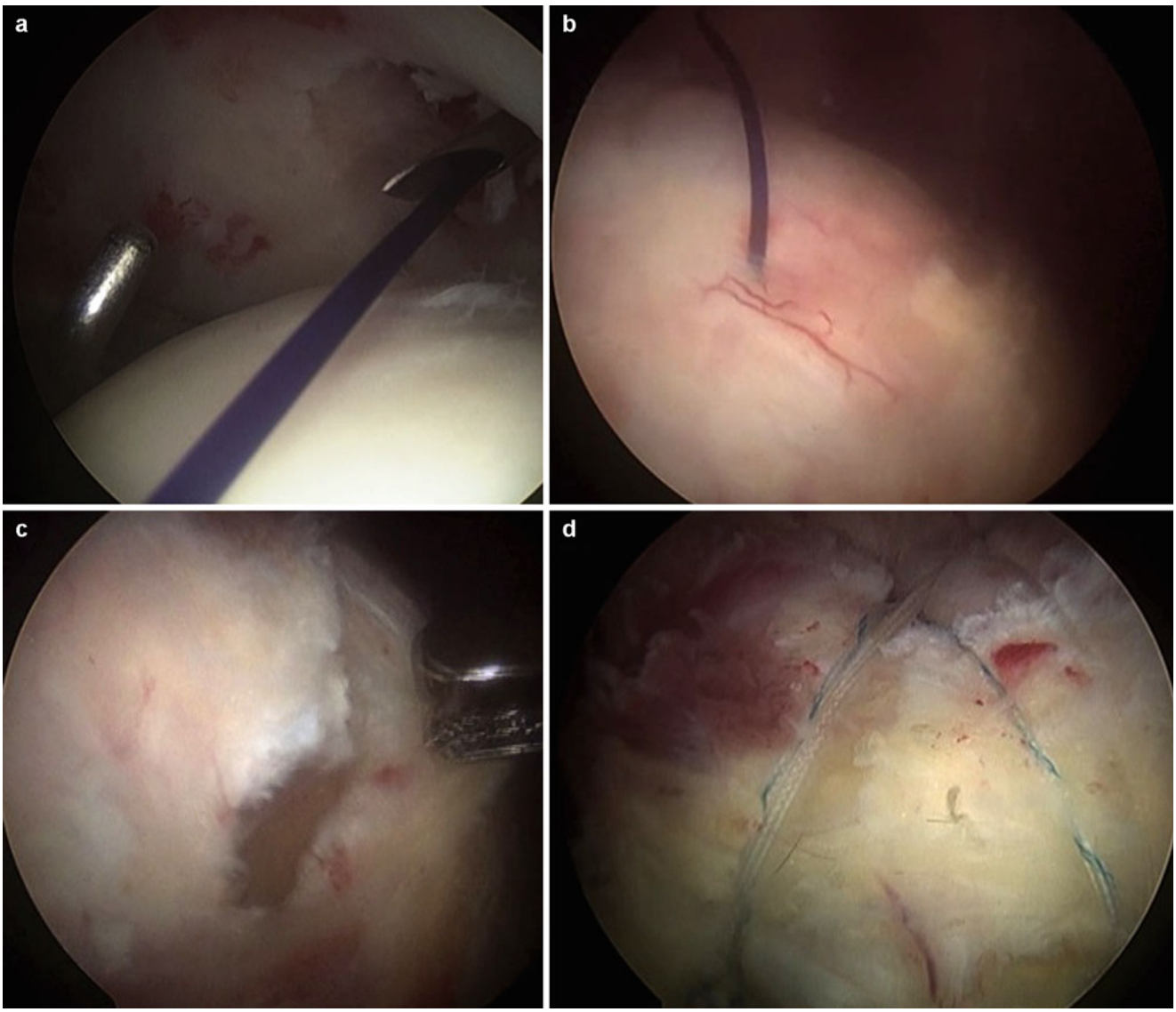

Fig. 22.4 Arthroscopic images of a left shoulder demonstrating use of an indirect suture passing technique in order to pass sutures through the rotator cuff (**a**, **b**), grasping of the rotator cuff tendon (**c**), and complete repair construct (**d**)

form anterior to posterior while visualizing from the posterior portal. Once all medial row anchors are inserted and sutures are passed, we begin tying sutures anteriorly and then progress posteriorly. As sutures are tied, they are shuttled to an accessory anterior portal for storage and later use in the lateral row construct. Alternating half-hitch stitches are utilized, as they do not require sliding through the anchor and/or tissue.

For lateral row fixation, the arm is abducted and externally rotated. The anterolateral pilot hole is placed 5–7 mm off lateral edge of the tuberosity and 5 mm posterior to bicipital groove while the posterolateral pilot hole is placed 5–7 mm lateral to the posterior aspect of the tear. In general, sutures from the medial row are secured laterally using 2–3 sutures from medial anchors to each lateral row implant. As the lateral row anchors (Footprint, Smith and Nephew, Andover, MA) are inserted sequentially, the sutures are also tensioned and securely fixed to the lateral row implant thus completing the repair. The completed construct should demonstrate apposition of tendon to bone with restoration of normal anatomic footprint of the rotator cuff and compression of tendon to bone providing optimal contact force and area between the tendon and greater tuberosity.

For bursal sided tears, Koh et al. [52] have suggested that instead of debriding all of the remaining tissue to achieve a complete tear and hole to visualize the glenohumeral joint, just enough tissue should be debrided to allow a shaver and grasper to pass through into the shoulder joint ("small window" technique). This small window in the robust medial footprint of rotator cuff preserves as much of the remnant healthy tissue as possible. After debridement and minimal decortication of the footprint, percutaneous placement of

Table 22.2 Outcomes

Author	Year, journal	*N*	Surgical technique	Average follow-up (months)	Results
Wright and Cofield [53]	1996, *JSES*	39	Open acromioplasty, debridement, tendon suturing	55	59 % excellent, 26 % satisfactory, 15 % unsatisfactory (4/6 with unsatisfactory results had had previous unsuccessful surgery); no reoperations in entire cohort
Weber [59]	1999, *Arthroscopy*	55	Mini-open side-to-side repair ($n=33$) vs. debridement with acromioplasty ($n=32$) of tears >50 % width of tendon	42.9	Debridement group: 3/32 extended to full-thickness tears and 3/32 required mini-open repair for moderate symptoms; average UCLA 22.7 vs 31.6 (Repair group)
Ide et al. [50]	2005, *AJSM*	17	Transtendon	39	Average UCLA and JOA significantly improved ($P<0.01$); 2/6 overhead athletes returned to previous level, 3/6 to lower lever, and 1/6 unable to return
Deutsch [55]	2007, *JSES*	41	Conversion to full-thickness, suture anchor repair	38	All with improvement in motion, strength, pain relief, and ASES (42–93, $P<0.001$); 98 % satisfied
Brockmeier et al. [56]	2008, *Arthroscopy*	8	Intratendinous repair	5	Initial outcomes encouraging (all athletes including 5 high-level baseball players and 3 high-level tennis players)
Shin [57]	2012, *Arthroscopy*	48 *2 groups of 24	Transtendon repair ($n=24$) vs. tear completion with anchor repair ($n=24$)	31	22/24 in each group satisfied; Repair group with improved ASES ($P=0.037$) and Constant ($P=0.019$); Repair group with better pain control at 3 months postop; Repair group with 2/24 MRI-demonstrated retear (compared to transtendon: 0/24)
Kim et al. [58]	2012, *KSSTA*	32	Arthroscopic transtendon suture-bridge repair	17.4	Significant improvements in UCLA, ASES, Constant, VAS, and FF

* just indicates that of the 48 shoulders, there were 2 groups of 24 shoulders (Transtendon repair (n = 24) vs. tear completion with anchor repair (n = 24))

double-loaded suture anchors is accomplished. The technique utilized is similar to the technique described above for use in completed articular-sided tears. With the arthroscope in the posterolateral portal, a spectrum device (Linvatech, Largo, FL) preloaded with No. 0 PDS (Ethicon) is introduced through the anterolateral or anterior cannula in order to penetrate the full thickness of the tendon approximately 5–10 mm medial to the torn margin of the RC tendon, one anteriorly and another posterior. Once the suture hook penetrates the full thickness of the tendon, the PDS is relayed inside the glenohumeral joint. Through the "small window" [52] created earlier, a grasper is used to retrieve PDS from joint. One limb of the suture anchor is relayed with the PDS within the cannula, and the other limb was relayed in the same fashion. Arthroscopic knots are then tied in a standard fashion.

Postoperative Care

Postoperatively, patients undergo 6 weeks of shoulder immobilization with an abduction derotation sling with supervised gentle passive range of motion (ROM). This is followed with active-assisted exercises at 6 weeks postoperatively with stretching focusing on forward elevation, external rotation at the side, and internal rotation up the back. Isometric strengthening, muscle reeducation, and scapular stabilization start at 6 weeks postoperatively. Resistive exercises are added at 10 weeks.

Literature Review

A review of recent outcomes [50, 53–58] following repair (various techniques) of partial-thickness rotator cuff tears is presented in Table 22.2. Overall, current evidence shows similar outcomes when comparing tear completion and repair to transtendinous repair of tears greater than 50 % the width of the tendon. There are no studies, however, that completely support one technique over another. Further, while not discussed specifically in Table 22.2, debridement with or without formal acromioplasty has been a reliable treatment option for tears less than 50 % the width of the tendon.

Summary

Partial-thickness rotator cuff tears represent an increasingly recognized clinical entity on the spectrum of rotator cuff pathology. These tears are now diagnosed earlier in their disease process and with more accuracy as a result of increased

physician awareness and incredible improvements in diagnostic modalities. The nature of partial-thickness cuff tears is dependent on both intrinsic and extrinsic factors and often results from a combination of both. Arthroscopic repair remains the treatment of choice; however, specific technique choices are dependent on the specific tear characteristics. Future research is needed to evaluate long-term outcomes following arthroscopic repair of partial-thickness rotator cuff tears.

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Full-Thickness Rotator Cuff Tears

23

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Epidemiology

Rotator cuff tears are the most commonly encountered shoulder disorder. They can be degenerative or traumatic.

Degenerative rotator cuff tears have a closely age-related prevalence of between 15 and 51 % [1–7]. In individuals over 65 years of age, 50 % of rotator cuff tears are bilateral [5]. Only one-third of these lesions cause pain [7]. A recent study of 588 patients with unilateral shoulder pain demonstrated that 35.5 % of patients with a symptomatic tear also had an asymptomatic rotator cuff lesion in the contralateral shoulder; patients with a partial tear or intact rotator cuff and shoulder pain instead had much lower percentages of full-thickness tears in the asymptomatic contralateral shoulder: 4.3 and 0.5 %, respectively. Finally, the symptomatic tears were, on average, 30 % larger than the asymptomatic ones [5]. The development of pain and limitation in daily activities was, in fact, associated with expansion of the lesion, understood not only as an increase in the size of a full-thickness lesion but also as conversion of a partial lesion into a full-thickness one [8]. Other factors have been found to be associated with the development of pain: age, dominance, fatty infiltration of the muscles of the rotator cuff, and alterations in glenohumeral kinematics [9–12]. It has been estimated that more than half of asymptomatic tears become symptomatic in around 3 years [3]. In the past, several authors have shown that degenerative lesions of the rotator cuff mainly involve the supraspinatus tendon, typically starting from the anterior portion of its humeral insertion near the long head of the biceps and propagating posteriorly [13–16]. More recent studies have instead demonstrated that rotator cuff tears start from the infraspinatus tendon [17–19]. Kim et al. [19], in an ultrasound study of 360 shoulders with either a partial-thickness or a full-thickness rotator cuff tear, found that the lesions arose in a region 13–17 mm posterior to the long head of the biceps tendon, near the junction of the supraspinatus and infraspinatus tendons. These results are confirmed by the rotator cable-crescent theory developed by Burkhart et al. [20] (see below).

Traumatic rotator cuff tears are caused by a fall or trauma to an abducted, externally rotated arm, and they generally occur in individuals with a mean age of around 55 years, who are thus almost 10 years younger than the population affected by degenerative lesions [13, 21]. Traumatic tears also tend to be larger in size than degenerative ones and often also involve the subscapularis tendon. In 50 % of cases they are, in fact, large or massive lesions [21]. If promptly repaired, traumatic tears are, in theory, associated with a very good outcome, precisely because the patients are younger, and there is less retraction and less fatty degeneration. The rate of successful healing after repair of traumatic injuries ranges from 65 to 69 % [21–24].

Pathophysiology

The rotator cuff is a musculotendinous structure composed of the supraspinatus, infraspinatus, teres minor tendons, which arise on the posterior surface of the scapula and insert on the greater tuberosity of the humerus, and the subscapularis

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tendon, which arises on the anterior surface of the scapula and inserts on the lesser tuberosity of the humerus.

The intrinsic, extrinsic, and secondary causes associated with the development of rotator cuff disease, including full-thickness tears, have already been examined in the chapter on impingement (Chap. 21).

The anatomy of the rotator cuff is the direct result of its function. Rotator cuff acts as a dynamic stabilizer of the glenohumeral joint, keeping the humeral head centered in the glenoid in all phases of movement. From a biomechanical point of view, its main function is to contribute to glenohumeral joint kinetics by balancing force couples in two planes: coronal (superior-inferior) and transverse (anterior posterior). Force couples acting on the coronal plane are the result of the balance between the deltoid and the upper part of the rotator cuff; in the transverse plane, the balance is between subscapularis muscle (anteriorly) and the posterior cuff (infraspinatus and teres minor) (see Chap. 2) [25–28].

In 1993, Burkhart et al. [20] described the theory of the rotator cable-crescent complex. The intact rotator cuff is characterized by an arching cable-like thickening of the coracohumeral ligament located at the margin of the avascular zone. Anteriorly, it inserts on the greater tuberosity of the humerus (just posterior to the long head of the biceps) and posteriorly closes to the inferior border of the infraspinatus tendon. This structure acts as a "suspension bridge": stress exerted on the rotator cuff muscles is transferred to the rotator cable, thereby reducing the stress on the weaker, thinner avascular region. The suspension bridge and rotator cable theory also applies in the case of a rotator cuff tear, in which the free margin of the tear corresponds to the rotator cable and the anterior and posterior margins correspond to the supports at each end of the cable's span. Therefore, also in the case of damage to the avascular region of the supraspinatus, the tendon is able to exert its compressive effect on the humeral head distributing tensions along the "suspension bridge." This would seem to explain the absence of active movement deficits in patients with small -and medium-sized tears and also the success of functional repairs of large or massive lesions, even without complete repair of the cuff [28].

From a pathological point of view, the size and extent of rotator cuff tears can vary. Partial tears can occur on the bursal or articular side of the rotator cuff or be interstitial; complete lesions involve the full thickness of the tendon.

The evolution of surgical techniques for repairing rotator cuff tears (from open to arthroscopic) has allowed an ever expanding knowledge of the anatomical structures involved and thus the development of different classification systems able to describe the lesions in increasing detail and strictly related to the possibility of repair.

Currently, full-thickness tears can be described in relation to their location, shape, area, retraction, and reducibility. Listed below are the classifications that have been proposed over the years:

- Codman [29] divided rotator cuff lesions into incomplete, complete, and pure transverse.
- McLaughlin [30] classified them as transverse, vertical split, and retracted.
- Wolfgang [31] divided them into transverse, triangular, and massive.
- DeOrio and Cofield [32] categorized them according to the length of the greatest diameter of the tear: small (less than 1 cm), medium (1–3 cm), large (3–5 cm), and massive (greater than 5 cm).
- Harryman et al. [33] divided them into:
  - Type 0: intact cuff
  - Type IA: partial tear
  - Type IB: full-thickness supraspinatus tear
  - Type II: full-thickness supraspinatus and infraspinatus tear
  - Type III: full-thickness supraspinatus, infraspinatus, and subscapularis tear
- Matsen et al. [34] divided them according to the extent of the lesion and the structures involved:
  - Stage I: full-thickness supraspinatus tear (≤2 cm)
  - Stage II: full-thickness supraspinatus and partial infraspinatus tear (2–4 cm)
  - Stage III: full-thickness supraspinatus, infraspinatus, and subscapularis tear (5 cm)
  - Stage IV: cuff tear arthropathy
- Snyder [35] proposed a classification based on the location and the severity of the lesion, dividing them into partial articular side (A), partial bursal side (B), and complete (C). The complete lesions were further subdivided as follows:
  - C/0: partial articular and bursal tear
  - C/1: full-thickness tear less than 1 cm
  - C/2: full-thickness tear, between 2 and 3 cm, with minimal retraction, usually involving only the supraspinatus tendon
  - C/3: tear involving the supraspinatus and part of the infraspinatus tendon
  - C/4: massive tear involving at least two tendons
- Burkhart and Lo [28] proposed a geometric classification based on arthroscopic identification of the shape of the lesion (Fig. 23.1). Each type of lesion corresponds to a certain type of repair so as always to ensure a tension-free repair and balanced force couples. They classified rotator cuff tears as follows:

– Type I, crescent-shaped: classic lesions that have excellent mediolateral mobility regardless of their size and can be repaired with minimal tension.
– Type II, U, L, or reverse L shaped: U-shaped tears extend more medially than crescent-shaped ones, their apex being located more adjacent or medially to the glenoid rim. L- or reverse L-shaped tears are characterized by a

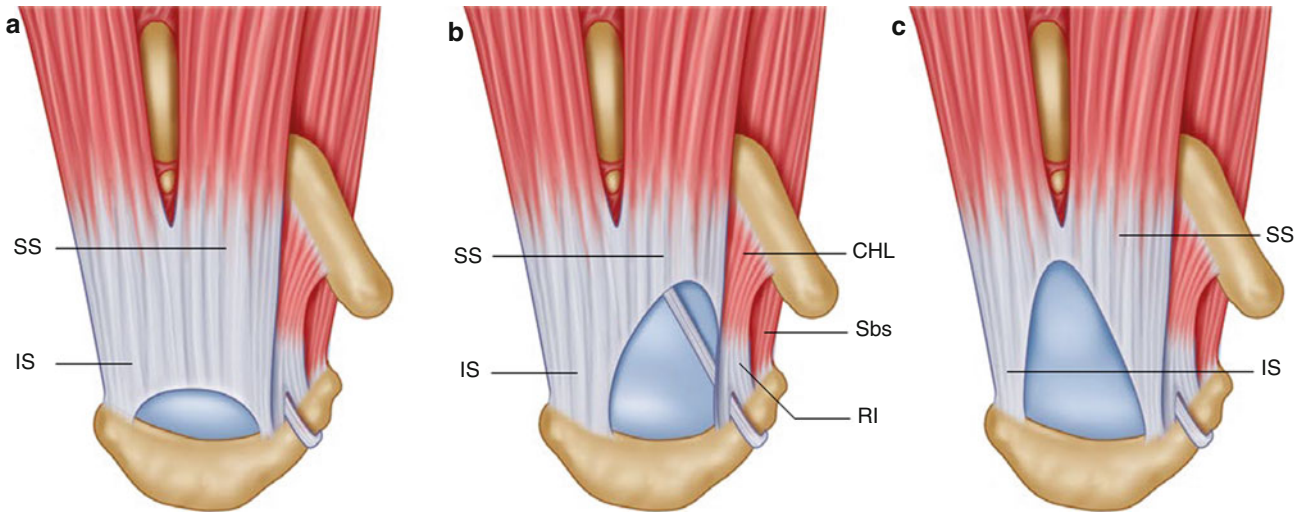


Fig. 23.1 Geometric classification of full-thickness rotator cuff tears. (**a**) Crescent. (**b**) Reverse L shaped. (**c**) U shaped (*SS* supraspinatus, *IS* Infraspinatus, *RI* Rotator interval, *SbS* Subscapularis, *CHL* coraco-humeral ligament)

mobile leaf. In L-shaped tears, the anterior leaf is the more mobile, while in reverse L-shaped tears, the posterior one is the more mobile. It is crucial to recognize the shape of the tears and in particular to identify the apex of the tear, as they are not suitable for direct tendon-to-bone repair, but require, rather, the use of "margin convergence" techniques designed to reduce tension and draw the free edge of the tear towards the tendon footprint.

- Type III, massive, retracted, and immobile: tears characterized by mediolateral or anterior-posterior immobility that require the application of specific mobilization techniques before they can be repaired.
- Type IV, cuff tear arthropathy.

History

According to Neer's classification [36] (see Chap. 21), degenerative rotator cuff disease evolves through three stages. The second stage, in particular, is characterized by the development of a partial-thickness tear leading to the appearance, in the third stage, of a full-thickness tear.

Actually, the natural evolution of rotator cuff tears is still debated. Some authors assert that a partial-thickness tear, once established, will inevitably progress towards a full-thickness tear, just as a full-thickness tear will tend to enlarge over time [3, 5, 37]. Other authors have instead shown that isolated supraspinatus tears do not always progress [10, 38]. This finding has been confirmed by a recent study of 24 patients aged under 65 years with an isolated supraspinatus tear (of traumatic or nontraumatic origin) diagnosed by MR arthrography and reevaluated after a mean follow-up of 42 months. The authors showed that, at follow-up, the tear was no longer detectable in two shoulders and was reduced in size in nine; in nine patients it showed no change, whereas in only six cases was the lesion found to have increased in size [39]. Conversely, there is no doubt that large and massive lesions tend to increase in size over time [10, 40].

A careful history and a detailed clinical and radiological assessment are therefore fundamental for a correct diagnosis and, consequently, the most appropriate choice of treatment, as well as for the definition of possible outcomes associated with each treatment.

The patient's age and dominance, the traumatic or spontaneous onset of the shoulder pain, the type of work, the loss of strength, the presence of nocturnal pain, and the duration of the symptoms will help to establish a diagnosis.

Degenerative tears should be suspected in patients older than 60 years, who perform manual activities and complain of gradual onset of nocturnal pain in the shoulder of the dominant arm, followed by exacerbation of pain during the day, and associated loss of strength.

In the case of traumatic tears, on the other hand, the patient is more likely to be younger and able to link the onset of symptoms to a specific traumatic event. However, the presence of traumatic tears cannot be excluded in patients over 60 years of age. In these patients, the traumatic event could well have increased the size of a preexisting degenerative tear.

Small full-thickness or partial-thickness rotator cuff tears caused by repetitive microtrauma can also be diagnosed in young overhead athletes complaining of limitation of the athletic gesture and pain lasting several hours after participation in sporting activities.

A patient with a rotator cuff tear, regardless of his age and pathogenesis of the lesion, will always complain of pain and loss of strength. Clinically, loss of strength will suggest a

Neer stage II or III. These symptoms display a progressive course over time. The patient will describe the pain as located laterally (or anterolaterally if there is involvement of the long head of the biceps), irradiating to the arm or neck and exacerbated by elevations above 90° or overhead activities; he will report nocturnal pain and may also complain of a limited active range of motion and of limitations in daily living activities. A typical sign is progressive difficulty reaching objects placed above head level.

The clinical history must nevertheless be integrated with the results of careful clinical and imaging evaluation before the diagnosis can be confirmed.

Clinical Examination

The clinical examination always starts with an evaluation of active and passive range of motion, presence of any subacromial crepitus, inspection of both shoulders in order to detect any muscle atrophy and deformities, and assessment of both scapulae and of joint kinematics. It is also necessary to perform various specific clinical tests. A general overview of rotator cuff pathology and specific tests for impingement is provided in the relevant chapters (see Chaps. 3 and 21).

Assessment of muscle strength is a critical clinical aspect in the diagnostic work-up of a rotator cuff tear. Listed below is a series of specific clinical tests for evaluation of rotator cuff tears:

- Yocum test: evaluates supraspinatus tenderness and strength.
- Jobe test: this test is performed by having the patient abduct the arm in the scapular plane against resistance. The arm is internally rotated (thumb pointing down). The onset of pain, or a reduction in strength, indicates a supraspinatus tear or tendinopathy.
- Infraspinatus test: this test is performed by having the patient perform an external rotation against resistance with the elbow adducted. The onset of pain, or a reduction in strength, indicates an infraspinatus tear.
- Patte test: this test is performed by having the patient perform an external rotation against resistance with the shoulder abducted to 90° and the elbow flexed. The onset of pain, or a reduction in strength, indicates an infraspinatus tear.
- Drop-arm sign: the examiner is positioned next to the patient; the patient's arm, with elbow flexed to 90°, is placed in 90° forward elevation in the scapular plane and completely externally rotated. The patient is asked to hold the externally rotated position. The wrist is then released, leaving only the elbow supported. The test is positive if the forearm drops, indicating the presence of an infraspinatus tear.
- External Rotation Lag Sign (ERLS): the examiner is behind the patient; the patient's arm, with elbow flexed to 90°, is placed in 20° forward elevation in the scapular plane and almost completely externally rotated. The patient is asked to hold the externally rotated and elevated position, and the wrist is then released. The test is positive if the forearm drops, indicating the presence of a supraspinatus and infraspinatus tear.

The possible involvement of the subscapularis tendon is evaluated using lift-off test, Napoleon test, and bear-hug test, while specific tests for involvement of the long head of the biceps are palm-up test, Yergason test, active-passive test of the long head of the biceps, and Ludington test (see Chap. 23).

It is crucial to remember that patients suffering from a full-thickness rotator cuff tears complain of functional limitation, which is related not only to the pain and loss of strength caused by the muscle-tendon lesion but also to the alteration of the joint kinematics resulting from the altered balance of force couples [25, 28]. An understanding of this concept is the basis both of conservative and surgical treatment.

Imaging

Several imaging procedures are used to diagnose rotator cuff pathology.

Conventional Radiology

Standard X-ray shoulder series (impingement series, see Chaps. 4 and 21) are helpful to evaluate the acromiohumeral distance and the shape of the acromion, and to rule out concomitant conditions, such as osteoarthritis, os acromiale, or calcifying tendinopathy.

Ultrasounds

Ultrasounds (US) are a noninvasive, low-cost imaging tool widely used to diagnose rotator cuff diseases. Some studies revealed a high sensitivity and specificity of US in diagnosing a full-thickness rotator cuff tears [41–43], with results comparable to those obtained with magnetic resonance (MR) [44] and MR arthrography (MRA) [45]. In a recent study, Ok et al. [43] showed that US are less accurate in sizing the lesion compared with MR. US have the same sensitivity and specificity of MR in assessing fatty infiltration of rotator cuff muscles [46]. Nevertheless, it should be kept in mind that US are still an operator-dependent procedure.

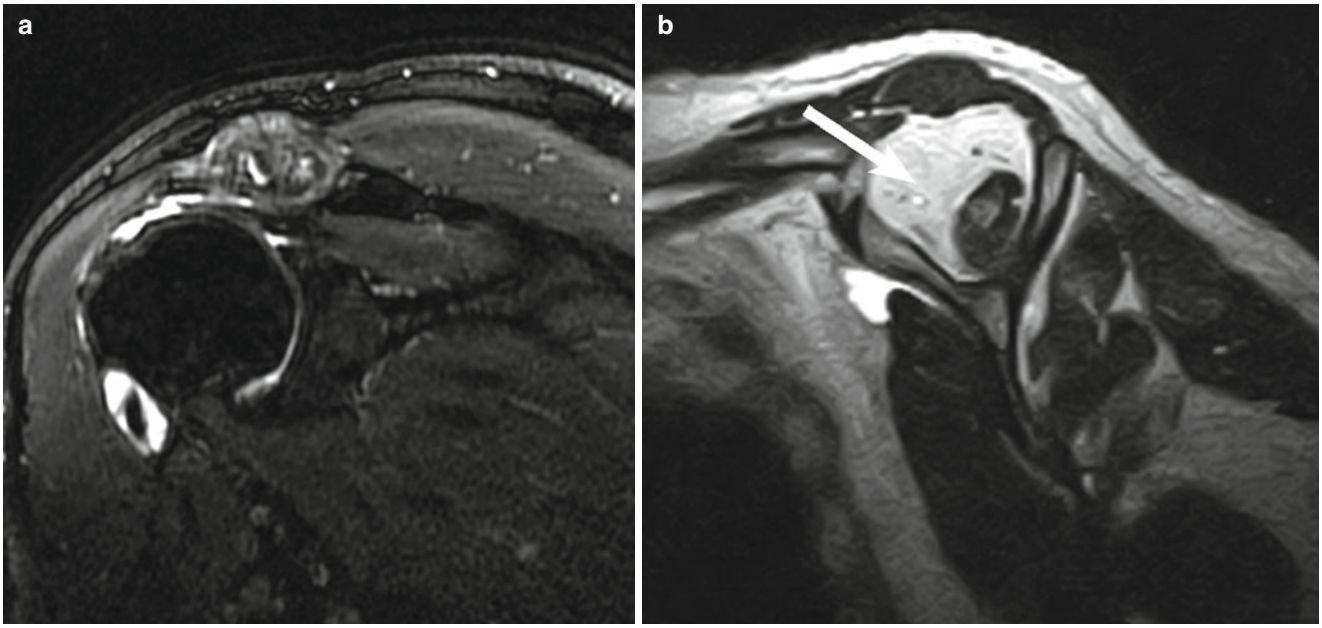


Fig. 23.2 MR of a full-thickness rotator cuff tear. (**a**) Oblique coronal view shows tendon retraction to the humeral head. (**b**) Sagittal view shows severe fatty infiltration of the supraspinatus (*arrow*)

Magnetic Resonance

MR is a very accurate procedure for diagnosing rotator cuff tears, but it is also very expensive [47, 48]. Recent studies recommend high-field MR (3.0 T) for its great accuracy [49, 50]. Using the coronal oblique, sagittal, and axial series is possible to evaluate the subacromial bursa, the thickness of the tendons, and also the presence of tendon tears (Fig. 23.2a).

Patte's classification [51] is the most widely used for evaluating the rotator cuff tears based upon the number of tendons involved and tendon retraction. The number of tendons involved is evaluated on sagittal images and the lesions are divided into:

- Segment 1: subscapularis
- Segment 2: rotator interval
- Segment 3: supraspinatus
- Segment 4: supraspinatus and the anterior part of infraspinatus
- Segment 5: supraspinatus and Infraspinatus
- Segment 6: massive tear

Tendon retraction is evaluated on coronal images and classified in:

- Grade 1, without retraction
- Grade 2, retracted to the humeral head
- Grade 3, retracted to the glenoid

Recently, Davidson and Burkhart [52] proposed a geometric classification to evaluate preoperatively the lesions on the MR, by measuring the maximum tear length (L) on the coronal T2-weighted series and the maximum tear width (W) on axial T2-weighted series. Rotator cuff tears are classified in:

- Type I: $L \leq W$ and $L < 2$ cm
- Type II: $L > W$ and $W < 2$ cm
- Type III: $L \geq 2$ cm and $W \geq 2$ cm (performing accessory procedures like interval slides or partial repair is necessary in 75 % of cases)
- $L \geq 3$ cm and $W \geq 3$ cm (performing accessory procedures like interval slides or partial repair is necessary in 100 % of cases)
- Type IV: glenohumeral arthritis and absence of subacromial space

MR also allows to evaluate fatty infiltration and muscle atrophy on sagittal series (Fig. 23.2b). Gouttallier et al. [53] first described a classification of fatty infiltration on computed tomography arthrography (CTA). Later, Fuchs et al. [54] showed that MR has a better interobserver reliability than CT scans for the evaluation of fatty infiltration.

Gouttallier's classification [53] identifies five stages of fatty infiltration:

- Stage 0 – normal muscle
- Stage 1 – some fatty streaks
- Stage 2 – less than 50 % fatty muscle infiltration
- Stage 3 – 50 % fatty muscle infiltration
- Stage 4 – greater than 50 % fatty muscle infiltration

Recent studies reported contrasting results about the reliability of this classification [54–58]; for this reason some authors [54, 58–60] proposed a simplified version of this classification that identifies only three stages:

- Absent or minimal fatty infiltration
- Moderate fatty infiltration
- Fat more than muscle

Other classification systems are reported in the literature to evaluate muscle atrophy of the rotator cuff on MR sagittal series [61–63].

Computed Tomography

CT is also very useful in diagnosing full-thickness rotator cuff tears. Its sensitivity and specificity are enhanced using an intra-articular contrast agent (CTA). However, diagnostic accuracy of CT is lesser than that of MR; for this reason it is indicated in patients that cannot undergo MR exam [48, 64].

Magnetic Resonance Arthrography

Although MRA has the highest sensitivity and specificity, it is an invasive and expensive diagnostic tool. Nowadays, its role in diagnosing rotator cuff tears is limited, while it is the golden standard for the evaluation of injuries of the glenoid labrum [48, 65]. Some studies reported the importance of MRA to evaluate the potential healing of repaired rotator cuff tears [66, 67].

Treatment: Indications and Contraindications

Although rotator cuff tears are very frequently observed in clinical practice, there is still no consensus about their treatment. Recent literature reviews [68–70] pointed out the role of the nonoperative treatments, such as physical therapy, nonsteroidal anti-inflammatory drugs (NSAIDs), subacromial infiltration of corticosteroids, or hyaluronic acids, for treating small or asymptomatic tears; nevertheless, surgical treatment has shown better results than nonoperative management at middle-long-term follow-up. Therefore, it is possible to summarize surgical indications as below:

- Small tears not responsive to conservative management
- Asymptomatic tears converted into symptomatic
- Symptomatic or asymptomatic middle- or large-sized tears
- Recent traumatic tears (less than 4 months) [71]
- Massive tears

Relative contraindications to surgery are represented by:

- Elderly
- Fatty infiltration greater than stage 2 according to the Gouttallier's classification [53]
- Inveterate traumatic tears

Contraindications to surgery are represented by:

- Articular stiffness (loss of passive range of motion)
- Severe osteoarthritis
- Tendon retraction to the glenoid
- Severe fatty infiltration
- Severe muscle atrophy
- Ongoing systemic or local infections
- Neurologic diseases or comorbidities that preclude surgery

Decision-Making Algorithm

The decision-making algorithm is based upon an accurate history taking and clinical evaluation of the patient. Subsequently, the clinical suspect will be confirmed by imaging studies. Specific radiographic views and MR will be requested if a rotator cuff disease is suspected. In particular cases, if there is muscle atrophy and/or neurological symptoms, often derived from a trauma, an electromyography will rule out a primary neurological or muscular disease.

Pain reduction and functional recovery are the main goals of a rotator cuff repair. The surgical outcome can be influenced by some predictors, which should be considered before starting the appropriate treatment; these factors are patient's age and functional demand, duration of symptoms, tendon retraction, fatty infiltration, and muscle atrophy.

Recently, the MOON group [12] evaluated 389 patients with atraumatic symptomatic rotator cuff tears; the authors identified some modifiable factors significantly related with pain and functional impairment, such as scapulothoracic dyskinesis, deficit of active abduction and forward elevation, and loss of strength in abduction and elevation. Age, duration of symptoms, tear size, narrowing of the subacromial space, and smoking habit represented non-modifiable factors, not significantly related with poor outcome [12]. These statements justify an initial nonoperative treatment for patients with small asymptomatic rotator cuff tears, particularly if they are older than 60 or younger but with low functional demand. In these cases, addressing alterations of the scapulothoracic kinematics might result in pain reduction and function recovery. If the conservative treatment fails, a surgical option will be considered. On the contrary, for high-demand patients, surgical treatment is the first choice. When the rotator cuff tear is medium to large sized and symptomatic, without contraindications, surgical repair is mandatory.

Clinical Case/Example

A.C., 66-year-old female, diabetic, manual worker complained of a pain to her right shoulder during the last year. Pain was exacerbated during overhead activities (putting plates into the cupboard), but she never received any treatment, neither consulted an orthopedic surgeon.

Four months ago she fell down out in the street while she was carrying shopping bags. From that moment she complained of a worsening of the symptoms with severe shoulder pain irradiating at the arm, which prevented her from moving it. She referred to emergency, where a fracture was ruled out, and a MR of the shoulder was prescribed. She came to our attention 2 weeks after the trauma, still complaining of pain, mainly upon trying to raise her arm, and not responsive to common analgesic drugs.

Clinical evaluation revealed a marked limitation of the passive range of motion. Neurological deficits were ruled out. MR revealed the presence of rich intra-articular effusion, massive rotator cuff tear with retraction to the glenoid, also involving the subscapularis tendon.

At the beginning, she was treated conservatively to recover almost the full passive range of motion. This is because the stiffness of the shoulder was surely due to trauma, but we did not want to underestimate her metabolic disease (diabetes), which could predispose her to develop stiffness. After 4 weeks of rehabilitation, she was evaluated again, and we observed an improvement of the passive range of motion, even if active movements were still limited. She continued physical therapy for other 2 weeks, when complete passive range of motion was regained. A new clinical evaluation showed positive Yocum test, external rotation lag sign, and Napoleon test. Surgical indication was established for arthroscopic rotator cuff repair.

Arthroscopic Treatment: Surgical Technique

Patient Positioning

Surgery can be performed in general or regional anesthesia with an interscalenic block or in blended anesthesia. Interscalenic block is favorable because it permits the patient to collaborate in his positioning on the operative table and allows a better and longer control of postoperative pain (see Chap. 9). The patient can lie in lateral decubitus position or in beach-chair position, depending on the surgeon's preference. We prefer a regional anesthesia associated sometimes to sedation and the patient placed in the beach-chair position with the affected arm in traction (2–3 kg).

An evaluation under anesthesia of the preoperative range of motion is performed to rule out shoulder stiffness.

Portal Placement

It is very useful to mark bony landmarks with a dermographic pen: the spine of the scapula, the acromion, the clavicle, and the coracoid process. These landmarks will guide the portal placement during surgical procedure when soft tissues are swollen (see Chap. 10).

Four portals are generally used for a standard arthroscopic cuff repair. In some cases, if the tear is large or massive, additional portals might be necessary for the optimal positioning of the suture anchors and for suture retrieving.

Portal used by the authors are:

- Posterior portal: from this portal we usually perform the intra-articular diagnostic arthroscopy. Sometimes it can be used for passing and retrieving sutures. Posterior view can be also improved by an additional posterolateral portal (see Chap. 10).
- Anterior-superior portal placed with the outside-in technique. It permits to approach the joint passing through the rotator interval. This portal is useful to perform procedures on the long head of the biceps and the subscapularis tendon, for passing and retrieving sutures and knot tying. Moreover, it can be used for controlling the outflow and as secondary operative portal for powered or radiofrequency instruments.
- Standard lateral portal: it is used as viewing portal to evaluate the shape, location, and mobility of the tendon tear; as operative portal it is used to introduce powered and radio-frequency instruments and for suture management.
- Superior-lateral portal: this portal allows correct suture anchors placement. It is performed with an 18-gauge needle inserted adjacent to the middle third of the lateral margin of the acromion (or at the middle part of the rotator cuff tear with the scope positioned in the posterior or posterior-lateral portal).

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

All the arthroscopic procedures start with a diagnostic evaluation. Once the posterior portal has been established, the scope is introduced into the joint and the articular space is distended with 30 cc of air inflated with a syringe through the arthroscopic sheath. The evaluation of the intra-articular structures proceeds in this order: subscapularis tendon, long head of the biceps and pulley, articular side of the rotator cuff, humeral and glenoid articular surfaces, and glenoid labrum. In elderly patients without a history and clinical findings of instability, it is not necessary an accurate evaluation of the glenohumeral ligaments.

Combined lesions of the subscapularis tendon and/or the long head of the biceps, if present, should be addressed from the intra-articular side before passing the scope into the subacromial space (see Chaps. 25 and 26).

Once the intra-articular phase has finished, we will pass the scope into the subacromial space through the posterior

portal. A new posterior-lateral portal can also be performed (see Chap. 10). The trocar is passed under the inferior part of the lateral edge of the acromion to have a complete access to the operative field. The inflow will distend the subacromial space. An accurate subacromial bursectomy is performed using a powered or a radio-frequency instrument through the lateral portal in order to have a good visualization of both the free tendon edge and the tendon footprint on the greater tuberosity. Switching the scope in the lateral portal and the radio-frequency instrument in the posterior one is possible to remove the posterior curtain of the subacromial bursa. Although some authors underlined the importance of preserving the subacromial bursa to enhance tendon healing (it contains blood vessels and cells) [72], a good visualization of the posterior cuff is obliged to adequately accomplish the procedure. In fact, bursectomy may facilitate the following steps:

- To expose the tendon tear.
- To distinguish the bursa from the tendon (that are mobile); if a retracted tear is present, it is very important to release and mobilize the tendon by grasping it and splitting it from the bursa or scar tissue using a radio-frequency device.
- To understand the shape of the tear.
- To ease suture management and knot tying.

Now it is possible to proceed to the cuff repair. With the scope in the lateral portal, the size, the retraction, and the geometry of the tear according to Burkart and Lo [28] are evaluated; mobility and reducibility of the tendon can be assessed using a grasper or a probe through the other portals (Fig. 23.3). Once the surgeon have evaluated these findings, it is possible to repair the rotator cuff tear anatomically and without excessive tension.

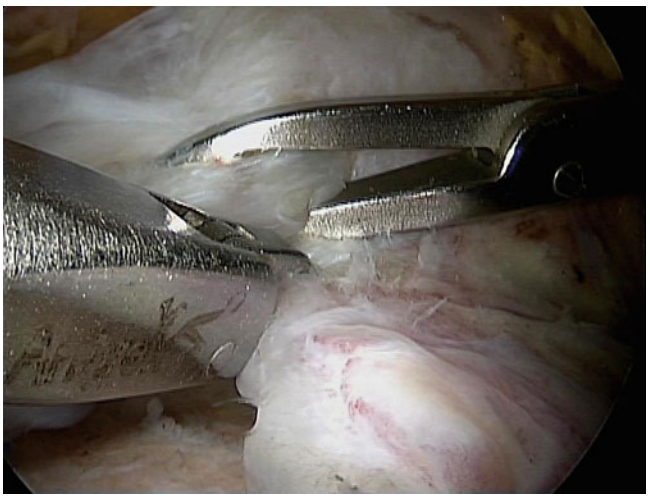


Fig. 23.3 "En face" view of a full-thickness rotator cuff tear (left shoulder; the scope is in the lateral portal). The shape and the mobility of the tear are evaluated using two graspers from the anterosuperior and the posterior portals

Step-by-Step Procedure (Video 23.1) (Box 23.1)

Type of repair is determined according to the shape of the lesion:

- Crescent-shaped tears can be easily mobilized and repaired with minimal tension directly to the bone using suture anchors (Fig. 23.4). If tears show atrophic edges, the debridement of the free margin of the tear may be performed with a basket to vivify the tear edge and favor healing.

Box 23.1: Tips and Tricks

- Bursectomy can be performed with a motorized instrument, but since the tissue is generally well vascularized, the use of a radio-frequency instrument will ensure a better control should bleeding occur.
- Inadequate bursectomy can make it extremely difficult to visualize suture management.
- Attention must be paid to the mobilization of tear margins: if we do not identify the tear shape and the direction of force vectors, it will be impossible to achieve tension-free repair.
- Attention must be paid to cortical abrasion of the greater tuberosity: excessive abrasion increases the risk of anchor pullout.
- It is not always easy to achieve placement of the anchors at the right angle because of the steric impediment of the lateral edge of the acromion. Inserting the anchor through a metal cannula will allow to reach the right position without pushing directly against the anchor handle.
- Attention must be paid to the distance between anchors: if anchors are too close, they may be converging, and in the case of osteoporotic bone, this increases the risk of pullout.
- When we retrieve the sutures before passing them through the tendons, attention must be paid to pass the strand of the suture always in the same way: before or behind the other sutures preloaded on the same anchor (not between them as far as possible).
- Always use cannulas for suture management: this will prevent the creation of false route or the interposition of soft tissues during the passage and retrieval of sutures.
- When placing the cannula in the anterior-superior portal to retrieve posterior sutures, it is useful to push the cannula down to the sutures to be retrieved, so as to avoid picking the wrong strand or letting other suture get in the way.
- Make sure the suture knots are on the cuff and not on the bone so as to increase contact pressure between bone and tendon.

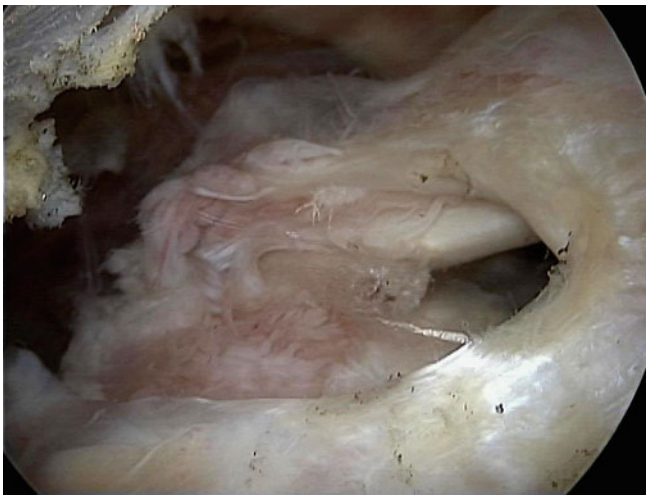


Fig. 23.4 Full-thickness rotator cuff tear (left shoulder; the scope is in the posterior portal). Crescent-shaped tears can be repaired directly to the bone with suture anchors

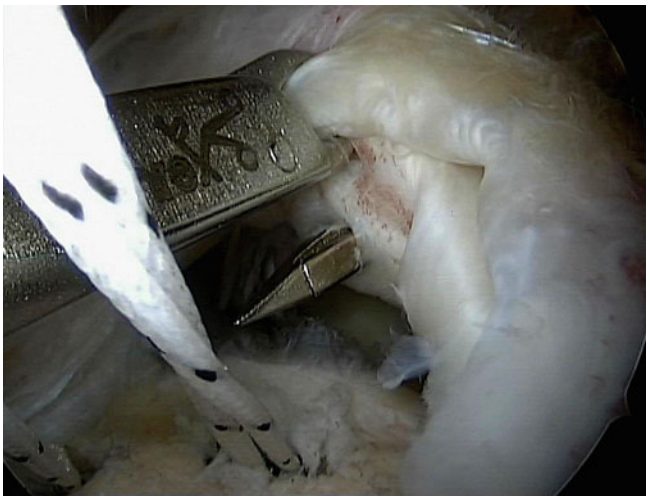


Fig. 23.5 Full-thickness rotator cuff tear (left shoulder; the scope is in the lateral portal). The posterior leaf of the tear often shows delamination. A direct retrograde suture passer is used to include all the tendon layers in the repair

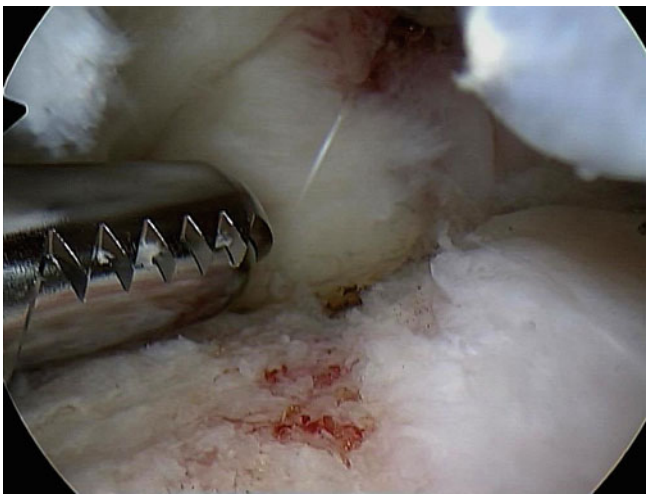


Fig. 23.6 Preparation of the tendon footprint (left shoulder; the scope is in the posterior portal). The shaver is used to abrade the cortical bone

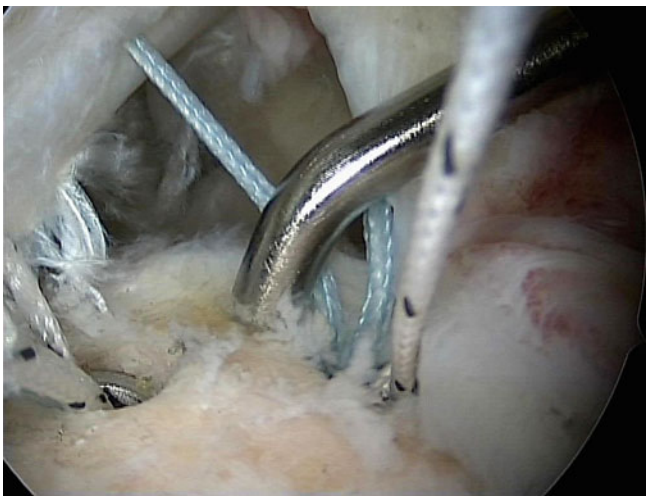


Fig. 23.7 Microfractures of the greater tuberosity (left shoulder; the scope is in the lateral portal). After anchor placement and sutures passing, multiple vents on the greater tuberosity are performed from medial to lateral with an arthroscopic awl. This technique is used alternatively to cortical abrasion to stimulate bone-tendon healing

- U-shaped, L-shaped, or reverse L-shaped tears will require an appropriate mobilization of the leaves as well as margin convergence techniques (side-to-side suture) to reduce tension and restore the crescent shape of the tear so that it can then be repaired with suture anchors. Side-to-side sutures can be performed in different ways: with separate knots or as a basket-shoes continuous suture. We prefer the separate knot technique (see Chap. 13). In order to carry out these sutures appropriately, it is essential to identify the tear apex: the first (medial) suture will run through the tear apex. In the case of U-shaped tears, the anterior and posterior leaves will be equally mobilized; therefore, once we pass the sutures from medial to lateral, the portal from which sutures will be knotted makes no difference (generally it is the anterior-superior portal). On the other hand, it is important to remember that in L-shaped tears (where the anterior edge is more mobile), sutures will pass from medial to lateral, from anterior to posterior; the post will be the strand of the suture that is passed through the anterior edge, and sutures will be knotted from the posterior portal. On the contrary, in the case of reverse L-shaped tears (where the posterior edge is more mobile) sutures will pass from medial to lateral, from posterior to anterior, the post will be the strand of the suture that is passed through the posterior edge, and sutures will be knotted from the anterior-superior portal. Attention must be paid to posterior cuff delamination and to the inclusion of all layers in the suture: including only the surface layer will lead to partial repair and therefore

greater tension in the repair and a high risk of re-tear (Fig. 23.5).

Once we have identified the tear pattern, mobilized the tear, and reduced the tension, we can achieve tendon repair through different techniques: single-row technique, double-row technique (or transosseous equivalent), and arthroscopic transosseous repair.

Before placing the anchors we must prepare the footprint. The arthroscope will be reinserted into the subacromial space through the posterior portal, a radio-frequency instrument will be introduced into the lateral portal to remove soft tissues from the footprint, and a motorized instrument will subsequently be used to abrade the cortical bone to stimulate healing (Fig. 23.6). In anterior tears, the accessory anterior-superior portal may be used to complete footprint preparation with a motorized or a radio-frequency instrument.

As an alternative to cortical abrasion of the greater tuberosity of the humerus, recent studies [73–75] have demonstrated the efficacy of microfractures in stimulating bone-tendon healing after arthroscopic repair of rotator cuff tears. The advantage of microfractures is that they do not weaken the cortical bone, especially in osteoporotic bone, thus reducing the risk of anchor pullout. We prefer to perform microfractures after placing anchors, from the articular margin of the humeral head to the lateral edge of the greater tuberosity over the whole footprint area and before knotting sutures (Fig. 23.7). We use a small joint perforator which allows for 5 mm deep, 1.5 mm wide microfractures set about 4 mm from one another following the original technique described by Steadman for focal chondral lesions of the knee [76].

Single-Row Technique

The single-row technique implies repairing the tear with a single row of medial anchors.

Different types of anchors are available according to size, screw thread pattern, material (metal, biodegradable, or PEEK), number of preloaded sutures, and type of fixation (knotted or knotless). Vented anchors are now available. They are supposed to favor the migration of bone marrow elements to the repair site through the holes of the anchor. For single-row repair we use knotted metal anchors preloaded with two sutures of different colors.

As a rule, one or two suture anchors are sufficient according to the size of the tear. Large tears may require up to three anchors.

While keeping the arthroscope in the posterior portal, we create a superior-lateral portal along the lateral edge of the acromion to place the anchors. An 18-gauge needle is used to determine the exact point of anchor entry. In extremely anterior tears, it will sometimes be possible to use the anterosuperior portal to place the anterior anchor.

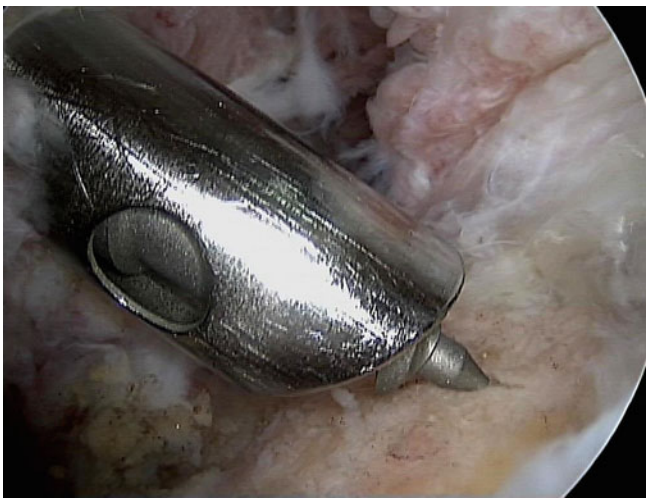


Fig. 23.8 Anchor placement (left shoulder; the scope is in the posterior portal). The anchor is placed through the superior-lateral portal, at a 45° "deadman" angle, close to the articular margin of the humeral head. A metallic cannula is used to place the anchor

After creating the portal, we prefer to use a metal cannula to place the anchor. In this way, if modifications are necessary to place the anchor correctly, this can be achieved by pushing on a rigid metal cannula rather than directly on the anchor handle, which could bend or break.

Anchors are placed along the articular margin of the humeral head, in the center of the tear, or at the anterior or posterior edge of the tear, respectively, for one anchor or two anchor repair, at a 45° angle relative to the direction of the tendon, so as to reduce the risk of anchor pullout (deadman's angle theory) [77] (Fig. 23.8). We direct the anchor laser marker so that the sutures are perpendicular to the tear edge, and we perform simple sutures with direct antegrade and/or retrograde suture passers. Anchors should always be placed from anterior to posterior, and sutures will be knotted from posterior to anterior according to the direction of the force vectors (see Chap. 13). The most appropriate knot type will be chosen according to tendon quality and the possibility for suture to run through the tear edges without tension (see Chap. 14).

In L-shaped or reverse L-shaped tears, one of the anchors will be placed at the corner of the lesion, and it will therefore be possible to perform mattress sutures. The anchor laser marker will therefore be directed in such a way that sutures run parallel with the tear margin: it will therefore be possible to pass one limb of the suture in the anterior edge of the tear and the other limb of the same suture through the posterior

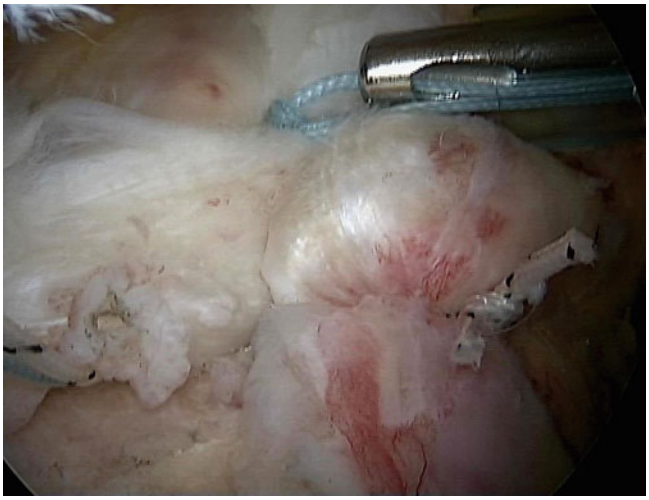


Fig. 23.9 L-shaped tear (left shoulder; the scope is in the lateral portal). After reduction of the tear and anchor placement, both limbs of one suture are passed through the tendon, creating a horizontal mattress configuration

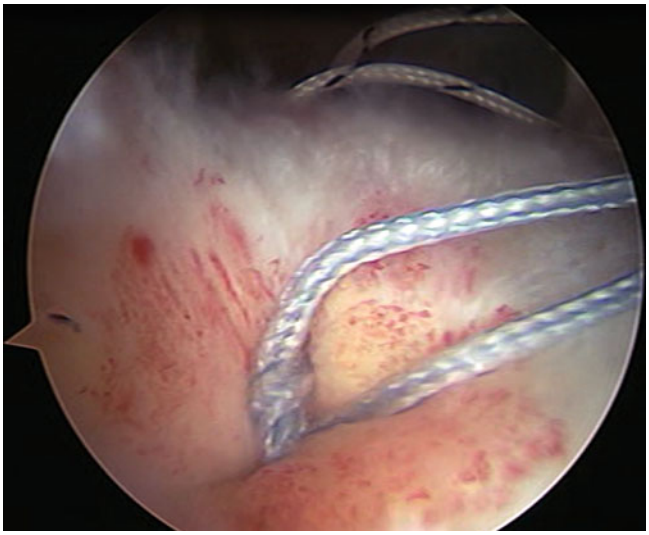


Fig. 23.10 SutureBridge technique (right shoulder; the scope is in the lateral portal). After medial anchor placement, both strands of each of the two anchor sutures are passed through the tendon and then tied from posterior to anterior

edge so as to create a side-to-side suture on the anchor; alternatively it is possible to pass both limbs of the thread through the posterior edge (in L-shaped tear) to reduce tension on the less mobile leaf (Fig. 23.9).

Double-Row Technique

The double-row technique implies two rows of anchors: a medial row and a lateral row. This technique arises from the necessity to increase the contact surface between tendon and bone so as to favor healing.

This technique was first described by Lo and Burkhart [78]. Over time, different configurations have been described: separate knots for the medial and lateral rows [78]; *SutureBridge*, which implies 2 medial anchors preloaded with 2 sutures and 2 knotless lateral anchors [79]; diamond back repair with 2 medial anchors preloaded with 2 sutures and 3 knotless anchors laterally with different connections between the sutures [80]; SpeedBridge, which implies the use of FiberTape (Arthrex; Naples, FL, USA) instead of sutures and knotless anchors both for the medial and lateral rows; and SpeedFix, which uses FiberTape without anchors in the medial row, secured with a knotless anchor laterally.

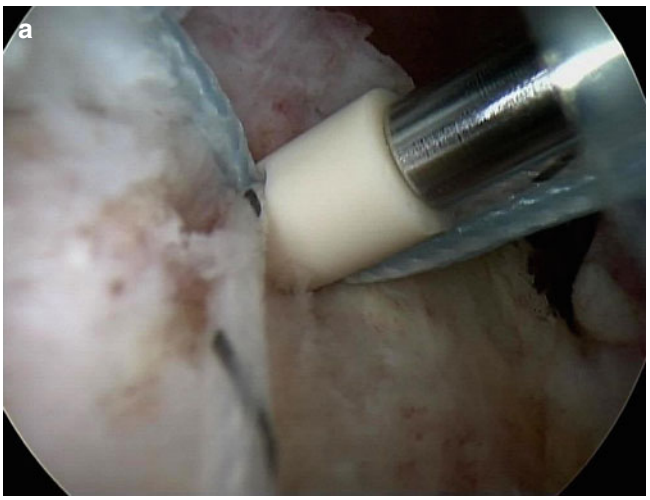
We use the SutureBridge configuration.

After preparing the footprint of the lesion and appropriately mobilizing the tear margin, medial anchors are placed as previously described. For each anchor, the laser marker is directed so that the sutures run parallel to the tear margin and all four limbs of the mattress sutures are passed. Sutures are knotted from posterior to anterior, and only the knots of the two middle sutures are cut: we will be left with two limbs of one posterior suture and two limbs of one anterior suture, possibly, of different colors (Fig. 23.10).

With the arthroscope in the posterior portal and the operative cannula in the lateral portal, we use a suture grasper to retrieve from the lateral portal the most anterior strand of the anterior and posterior sutures; these will be secured with a knotless anchor placed laterally at the level of the anterior anchor of the medial row (Fig. 23.11). Subsequently, the remaining two limbs of the anterior and posterior sutures are retrieved from lateral portal and secured with a knotless anchor in the lateral row at the level of the posterior anchor of the medial row. We will thus obtain a crisscross suture configuration. Besides ensuring a large contact area and high contact pressure to the bone tendon interface, this configuration helps distribute tension repair in several places, thanks to the different connections between the sutures (Fig. 23.12).

Arthroscopic Transosseous Repair

The possibility to perform arthroscopic transosseous suture without anchors is a recent development. The ArthroTunneler (Tornier) is a single-use device which allows to create two converging bone tunnels: one where medial anchors are generally placed and a lateral tunnel, in line with the former. It is possible to pass up to three sutures through each transosseous hole (best if of different colors) so that different suture configurations can be performed (Fig. 23.13). Actually there are no clinical studies confirming the efficacy of arthroscopic transosseous repair; therefore, the theoretical benefit of this kind of repair lies in the complete absence of anchors, which



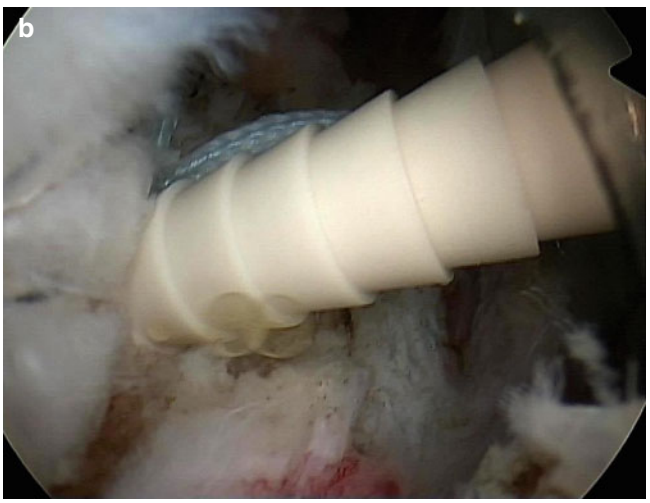


Fig. 23.11 SutureBridge technique (right shoulder; the scope is in the posterior portal). (**a**) One strand of each suture is retrieved from the lateral portal and loaded in the eyelet of the knotless anchor. (**b**) The lateral knotless anchor is placed

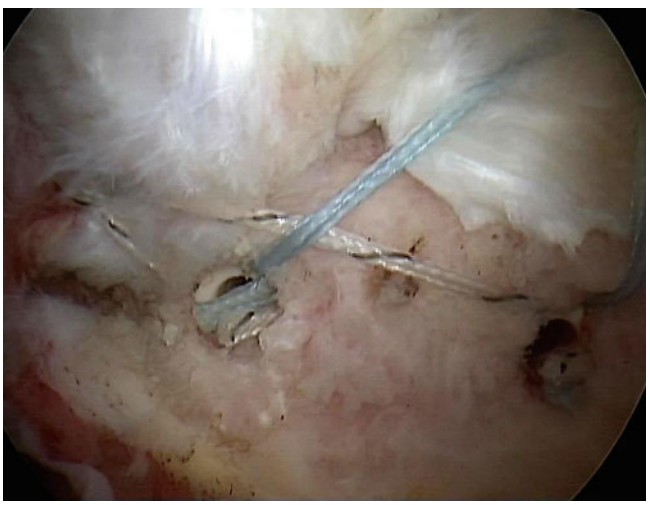
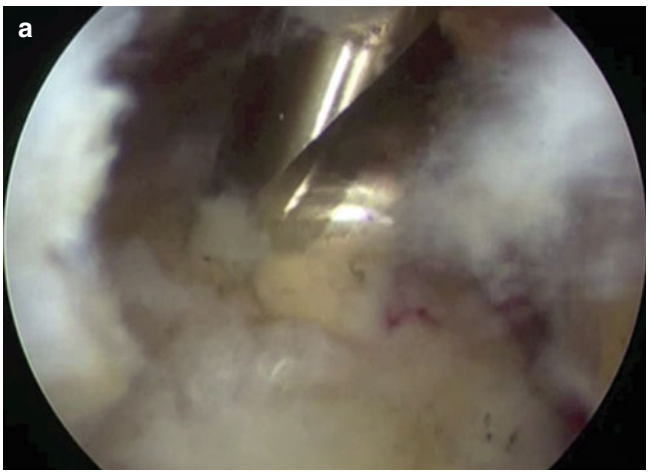
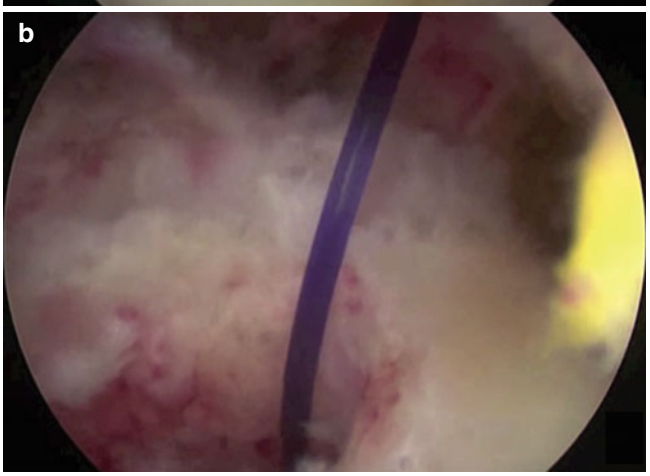


Fig. 23.12 SutureBridge technique (right shoulder; the scope is in the lateral portal). The crisscross suture configuration guarantees great fixation strength and large contact area of tendon to bone





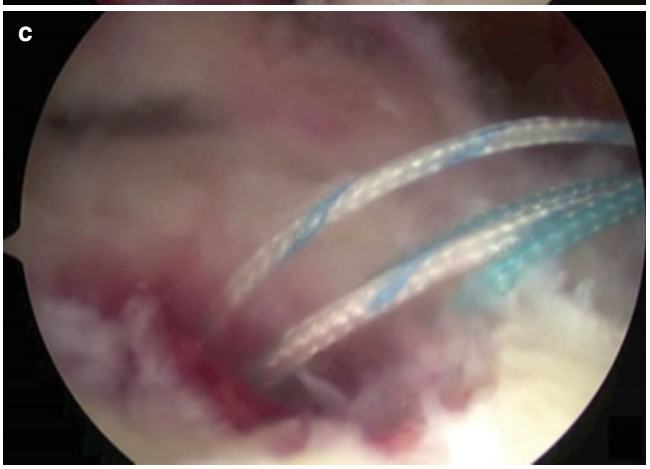


Fig. 23.13 Arthroscopic transosseous repair (right shoulder; the scope is in the posterior portal). (**a**) Preparation of the vertical transosseous tunnel. (**b**) Vertical tunnel with shuttle wire. (**c**) Vertical tunnel with sutures in place

is not only a strictly economical benefit but also a clinical benefit because it eliminates the risk of anchor pullout and facilitates future revision intervention while making it possible to obtain appropriate contact pressures at the bone tendon interface. However, a recent biomechanical study [81] has shown lower failure loads in this type of repair as

compared to double-row technique, regardless of the type of configuration.

Postoperative Care

Regardless of the type of repair, any patient who has undergone arthroscopic rotator cuff repair will remain immobilized in a sling with neutral rotation and 20° abduction for 4 weeks.

After the sling is removed, the patient will follow this rehabilitation protocol:

- Stage 1 (4–8 weeks after surgery): massotherapy and lysis of scar adhesions, passive mobilization for range of motion recovery
- Stage 2 (9–12 weeks after surgery): closed kinetic chain exercises to strengthen the rotator cuff, the subscapularis, the biceps, the deltoid, the pectoralis major, and the scapular stabilizers
- Stage 3 (13–16 weeks after surgery): open kinetic chain exercises, proprioceptive and plyometric exercises, and postural rehabilitation of the kinetic chain (lumbo-pelvic, thoracolumbar, and scapulothoracic muscles)

Return to heavy manual work and competitive sports activities is allowed 6 months after surgery.

Literature Review

Rotator cuff tear repairs are performed with open, mini-open, or arthroscopic techniques with comparable clinical results [82–84]. Arthroscopic techniques present undeniable advantages: lesser morbidity of the surgical site, shorter surgery time, and early recovery after surgery [84, 85].

Despite the good clinical results after arthroscopic repair [86–91], the rate of re-tear is high, especially in case of large massive tears [33, 92–94]. Different imaging methods (ultrasounds, CT, MR, and MRA) and classification systems have been used to assess structural integrity of the rotator cuff after repair [59, 67, 95–108]. Whether there is a link between clinical result and structural integrity is still controversial. Some studies did not show a significant difference between patients affected by complete or partial re-tear and patients with healed rotator cuff [99, 109–111]. On the other hand, other studies have shown that clinical results were significantly better in patients with a healed rotator cuff [33, 95, 100–104, 106, 108, 111–115].

In order to reduce re-tear rate, different arthroscopic surgical techniques have been developed over the years, so that they could lead to tension-free anatomical repair with an appropriate contact area at the bone-tendon interface. Although several studies showed better biomechanical properties after double-row repair [116, 117], recent literature reviews [91, 118, 119] do not show any advantage of double-row over single-row technique, neither from a clinical [94, 120–125] nor from a structural point of view as appears from studies using CTA and MR [120, 121, 124, 126]. Moreover, clinical studies on the re-tear pattern after double-row repair have shown that most re-tears occur along the medial row [127–129]. Therefore, although double-row repairs increase the contact area between bone and tendon, thus favoring healing at the interface, they also seem to increase the tension of the repair along the medial row. The use of FiberTape with medial knotless anchors is a recent development. However, a recent literature review [130] has shown that biomechanical results are improved when the medial row of a transosseous-equivalent rotator cuff is tied compared with a knotless repair.

In spite of technical improvements, structural integrity after rotator cuff repair is negatively affected by a number of biological factors: female gender [131], symptom duration [111], tear size [33, 112, 132, 133], fatty infiltration beyond Goutallier grade 2 [53] and muscular atrophy [60–62, 111, 134–141], smoking with a linear correlation between the number of cigarettes and the tear size [142–144], bone mineral density [145], diabetes [146], and vascularization of the repair area [147, 148].

Age is a confounding factor. While some studies suggest a negative correlation between old age and anatomical outcome [33, 95, 149, 150], others have shown good results after arthroscopic rotator cuff repair in elderly patients [113, 151–154]. Fatty infiltration and muscular atrophy appear to be the most important predictors of a negative surgical outcome both from a clinical and anatomical point of view [59, 99, 111, 135, 136, 138, 140]. They advance gradually, probably irreversibly, although repair seems to be able to block progression [111, 155, 156].

Finally, two recent studies have highlighted the fact that most re-tears occur in the first 3 months following surgery [23, 89]. A possible explanation might lie in the vascularization of the repair area, which seems to peak 1–2 months after surgery and gradually decreases in time [147, 148].

Summary

Rotator cuff tears can be degenerative or traumatic. Degenerative tears are the most common pathologies among shoulder disorders. Clinical assessment and MR evaluation are crucial. In elderly patients, pain onset and strength deficit may indicate the widening of a preexisting tear. Small tears can initially be addressed with conservative treatment. Small tears which fail to respond to conservative treatment as well as medium and large tears must be referred for surgical treatment. Different arthroscopic techniques have shown comparable clinical and structural

efficacy. The key to success for the arthroscopic rotator cuff repair is tear pattern recognition.

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Large-to-Massive Rotator Cuff Tears

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Epidemiology

Rotator cuff injuries have held a place in the surgical literature for well over a century, since Codman's reports of successful repair in 1911 [1]. Rotator cuff tears can be classified in three major ways – chronicity, thickness, and size. When a definitive injury is present, tears may be classified as acute tears when the injury occurred within 6 weeks of presentation [2]. Otherwise, they are labeled as subacute or chronic tears. Tears without a definitive history of injury can be classified in a similar manner based on the timing of the onset of symptoms. Tears may be partial thickness and involve either the bursal side or articular side or cause delamination and present as intertendinous. Large and massive tears are full-thickness rotator cuff defects that include multiple tendons.

One method of tear classification involves the size of the tear, determined either by MRI, ultrasound, diagnostic arthroscopy, or direct observation during surgery. Full-thickness tears are labeled small if they are <1 cm, medium if 1–3 cm, large if 3–5 cm, and massive if >5 cm [3, 4]. Another technique to quantify cuff tears is to classify multiple tendon tears as either large or massive and to use anterosuperior or posterosuperior to describe the functional deficit [5, 6]. In general, prognosis and natural history are poorer for larger tears than smaller tears, increased age of the patient, and chronicity of the tear [7].

The true incidence and prevalence of large and massive rotator cuff tears is difficult to define, as many tears likely go undiagnosed in asymptomatic individuals [8]. Tears may begin as smaller lesions and then progress in size if diagnosis is delayed or missed. Patients may be able to maintain relatively good active range of motion in spite of having a large or massive tear, but such maintenance of function requires significant increase in force generation by the intact portions of the rotator cuff and by the deltoid and scapular muscles [9]. Furthermore, it has long been known that even after surgical repair of a rotator cuff tear, functional outcome does not always correlate with integrity of the repair [10]. The prevalence of rotator cuff tears has been shown to increase with age [8, 11]. Smaller, well-compensated tears may progress with time to become larger and develop fatty infiltration and atrophy [12, 13]. Following repair of large and massive tears, the healing rate and overall satisfaction may decrease with chronicity [5, 14].

The causes of large and massive rotator cuff tears are multifactorial. Factors contributing to the pathogenesis of a tear can be traumatic, degenerative, developmental, capsuloligamentous, neuromuscular, inflammatory, infectious, and iatrogenic. In addition, large and massive rotator cuff tears may be associated with several other forms of pathology in and around the shoulder. Associated pathology of the glenohumeral joint including labral lesions, biceps tendon tears, and changes of the articular cartilage are common findings in many patients with full-thickness cuff tears. In some cases, large and massive tears may ultimately progress to rotator cuff tear arthropathy – an end-stage condition characterized by superior escape of the humeral head and painful degenerative changes of the glenoid and the acromion [15].

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History

The history at presentation given by patients with large or massive rotator cuff tears is often different from the acute smaller tear. While a history of trauma is common, the episode can be a minor traumatic event such as a minor fall or straining to lift a moderate object. There may be a history of prior similar episodes, even years before. Prior treatment is common, and patients complain of difficulty lifting overhead or away from their bodies. In some cases, the onset is insidious and symptoms may fluctuate with pain and weakness following the initial episode. Nighttime pain is frequent, and many patients find daytime symptoms can be improved by activity modification. While one shoulder is usually more symptomatic, bilateral involvement occurs in 50 % of patients older than 65 [16]. While pain is the most common complaint, disability is also noted in many activities of daily living. As weakness increases, patients become more symptomatic as tear size progresses. The term pseudoparalysis is used to describe patients who have lost their ability to elevate their arm due to the large rotator cuff defect (Fig. 24.1). Pseudoparalysis is seen in massive tears and is demonstrated by the patient's inability to actively elevate the arm, felt to be due to anterior or superior escape of the humeral head through the cuff defect.

Patients with a minimally symptomatic tear may experience increased pain if the tear size increases [17]. Large tears may continue to extend and produce pain, weakness, and loss of function [18]. Progression to arthritis is thought to occur in 4 % of patients with large or massive rotator cuff tears. Asymmetrical loading due to proximal migration of the humeral head and humeral acromial abutment are thought to contribute to cartilage erosion [19].

Differential Diagnosis

Pain at the deltoid insertion is classic, and radicular complaints associated with weakness occur and may be confused with cervical radiculopathy. A number of patients may exhibit marked weakness when attempting shoulder elevation and have significant cervical disc disease. An MRI of the shoulder would suggest a small or absent rotator cuff tear, which would be insufficient to explain this degree of arm weakness. Cervical evaluation, electromyography, and MRI imaging of the neck would be important in making the correct diagnosis. Other neurological causes of weakness include brachial plexopathy and cerebral vascular disease. Paraglenoids cysts can compress the suprascapular nerve and produce weakness of both the supraspinatus and infraspinatus. Tear extension and retraction can also injure the neurologic innervation to the cuff muscles [20]. Large ganglions associated with labral tears and readily identified by MR imaging can cause neurologic compression findings.

Clinical Examination

Atrophy of the rotator cuff muscles can be seen in large and massive tears and involves both the supraspinatus and infraspinatus muscles. Indentation of the supraspinatus and

Fig. 24.1 Pseudoparalysis: profound weakness in arm elevation due to loss of superior stability in a patient with a massive rotator cuff tear

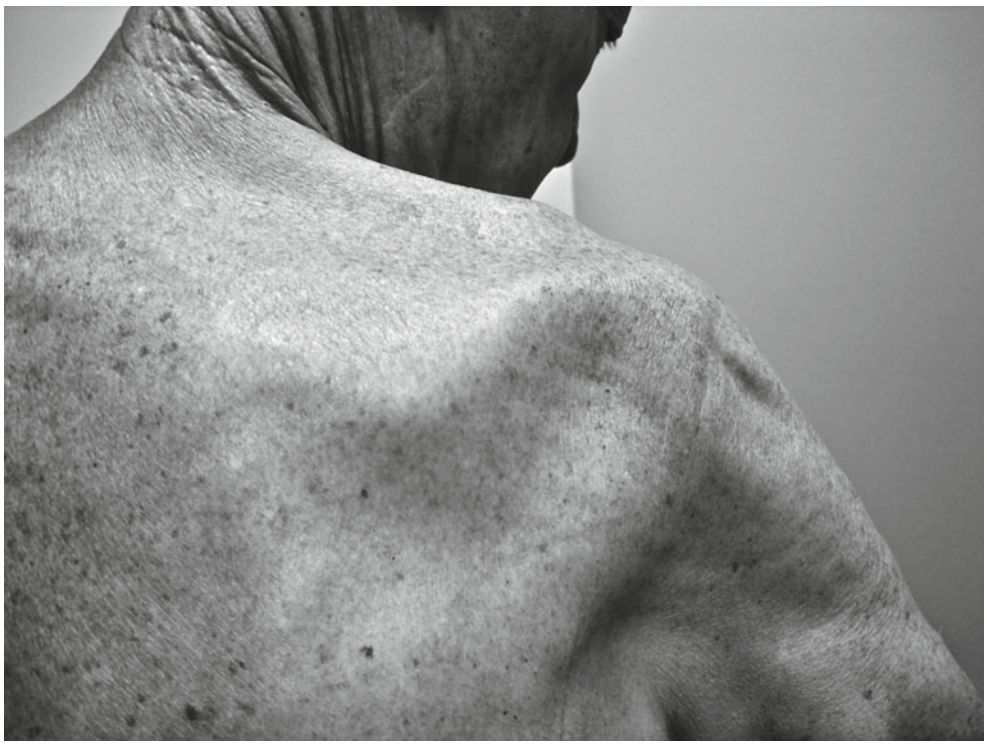

Fig. 24.2 Significant parascapular atrophy seen in patient with chronic large cuff tear

infraspinatus fossa is seen and can be compared to the uninvolved shoulder (Fig. 24.2).

While pseudoparalysis is found in patients with multiple tendon tears, some will have discovered ways to elevate their arm by flexing at the elbow and using the deltoid muscle and triceps muscles to produce elevation. Shoulder hiking or profound scapular elevation can be pronounced, particularly if there is superior escape or proximal migration of the humeral head. Impingement signs such as the Neer impingement sign [21] and Hawkins sign [22] are often positive, but not specific to large or massive rotator cuff tears. Crepitus is common and may be subacromial or glenohumeral. It is important to test each rotator cuff muscle to determine the extent of the tear. Weakness, as tested by static strength testing in specific planes, will indicate specific tendon involvement. Weakness to resisted internal rotation may indicate subscapularis involvement (Fig. 24.3). Weakness of abduction suggests supraspinatus damage and weakness to external rotation resistance is found in infraspinatus and teres minor involvement.

Differentiating weakness from pain can be difficult, as patients may guard during certain examinations. Some authors have suggested an injection of local anesthetic into the subacromial space as a means of differentiating apparent weakness due to pain, from true weakness due to tendon defects [23].

A substantial difference between active and passive motion on examination of the shoulder is perhaps the hallmark of large and massive rotator cuff tears. The lack of full passive motion does not preclude rotator cuff involvement. Osteoarthritis involving the glenohumeral joint often presents with weakness combined with a reduction of passive motion. An examination of the contralateral shoulder is helpful. Lag tests may be helpful in demonstrating profound

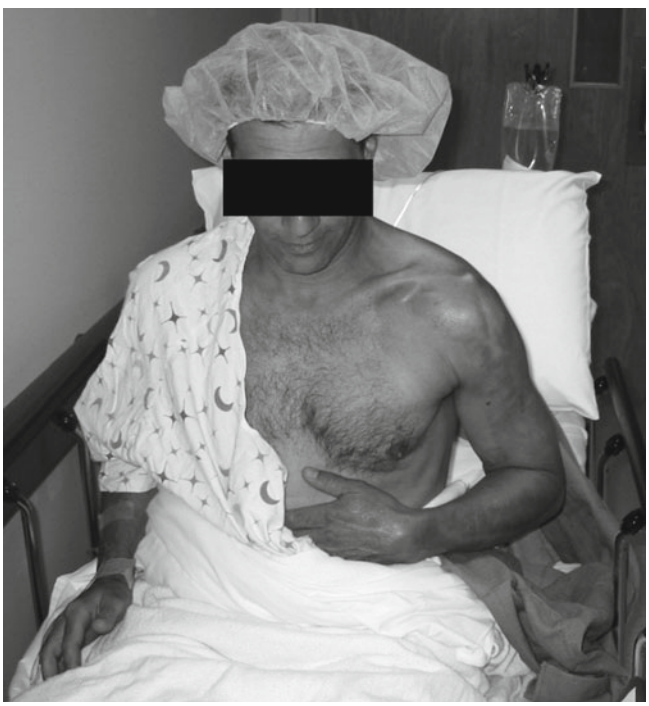

Fig. 24.3 Subscapularis weakness detected with belly-press maneuver. Patient is unable to maintain neutral wrist alignment when pressing on his abdomen

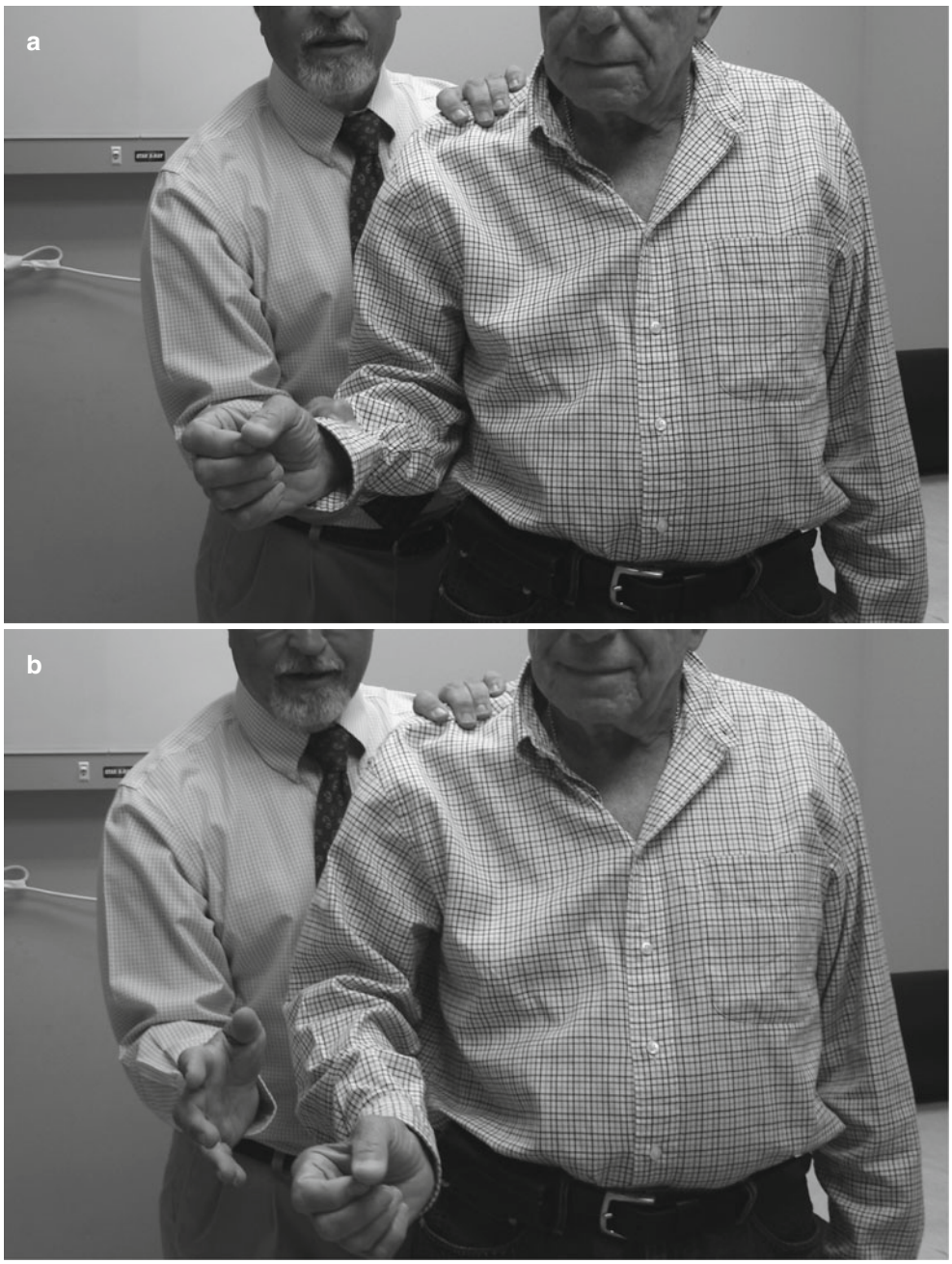

Fig. 24.4 A lag test for external rotation weakness. (**a**) The arm is positioned by the examiner and asked to maintain this position. (**b**) The examiner releases the arm and the arm rotates back to the torso

weakness in patients with satisfactory passive motion. Here, patients are unable to maintain a position due to profound weakness [24] (Fig. 24.4).

Lesions of the tendon of the long head of the biceps are commonly found in tears >5 cm with an incidence between 5 and 100 % [25]. Positive findings include pain at the biceps groove, pain with forearm supination, or Speed's forward flexion test. Though not always present, visual inspection of the biceps contour may reveal the typical "Popeye" deformity (Fig. 24.5).

A thorough neurological exam is essential in differentiating large and massive tears from weakness seen from neurological disorders. Weakness of distal muscles, sensory changes, or abnormal reflexes indicate a neurologic disorder and are not seen in large and massive rotator cuff tears. The examiner should confirm profound weakness is due to glenohumeral instability and cuff deficiency prior to considering reparative surgery.

Imaging

Imaging for massive rotator cuff tears most often includes X-ray and MRI. Occasionally, CT, CT arthrogram, and ultrasound are used as well. X-rays should be the first imaging

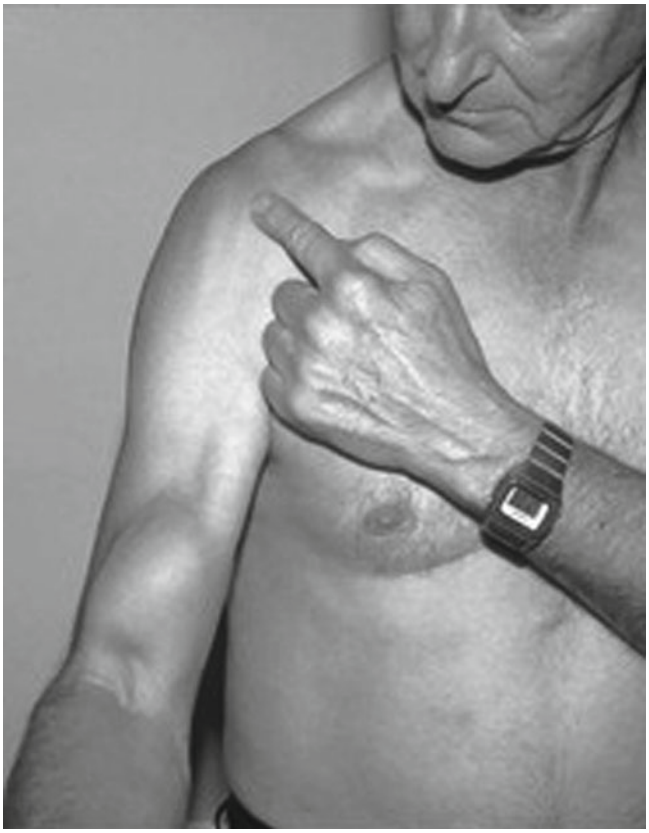

Fig. 24.5 A "popeye" biceps deformity can be seen in patients with a rupture and retraction of the long head of the biceps tendon

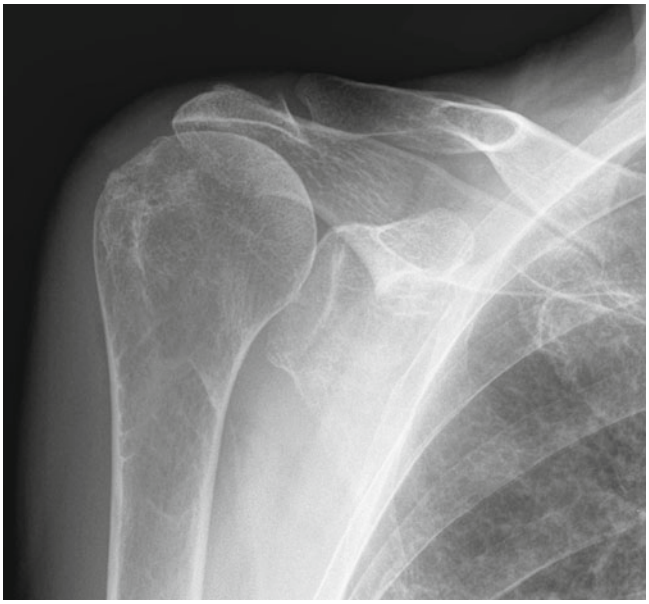

Fig. 24.6 A radiograph of an upright patient with superior migration of the glenohumeral joint. There is a significant reduction in the acromiohumeral distance

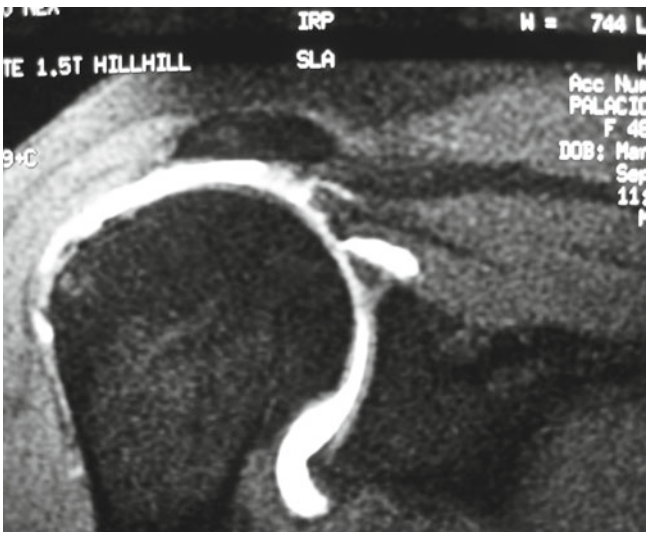

Fig. 24.7 An MRI in the coronal view demonstrating a retracted supraspinatus tendon

modality that is performed after obtaining a history and physical examination. Plain radiographs are useful to demonstrate degenerative changes in the shoulder. Anteroposterior images in internal and external rotation, a scapular Y lateral view, and an axillary image are typical images utilized. Superior elevation of the humeral head in neutral arm rotation during a seated or standing radiograph is indicative of chronic significant rotator cuff damage. When the acromiohumeral distance is measured to be less than 6 mm, superior escape of the humerus should be considered and rotator cuff repair should be considered with caution [26]. When the humeral head elevation becomes static or fixed, combined with degenerative changes and acetabularization of the acromion, a rotator cuff repair has a high risk of failure [27] (Fig. 24.6). Humeral head radius, mean lateral acromion angle, glenoid inclination angle, coracoid tip positioning, and acromial index are other measurements completed on plain radiographs that have been used and studied with variable results. Other findings on plain radiographs that can provide useful information include cysts underlying the rotator cuff footprint. These cysts can influence rotator cuff repair and anchor insertion. In revision situations, evaluation of the previous hardware can help in preoperative planning and counseling of the patient. The acromial morphology should be considered on the scapular Y view as well. Other findings on plain imaging may be unrelated to the rotator cuff tear but should be noted, including AC joint arthritis, calcification of the tendon, heterotopic ossification, and any spurring.

Large and massive tears are well visualized with MRI imaging with or without the use of articular contrast. Coronal oblique sequences can demonstrate the tendons that are injured and the extent that they retract (Fig. 24.7). The supraspinatus, infraspinatus, and teres minor are often best visualized in the coronal view, and the individual tendons and their

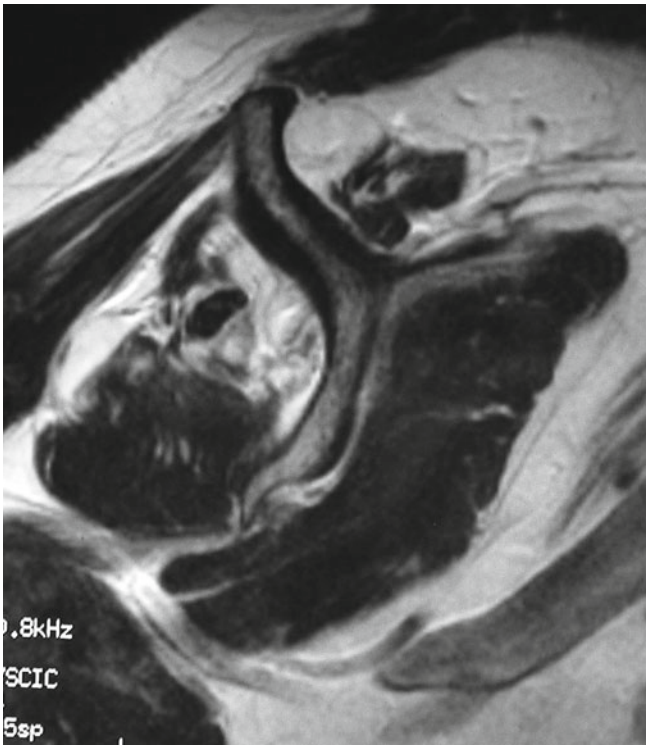

Fig. 24.8 A medial sagittal view MRI demonstrating chronic changes to the supraspinatus and infraspinatus muscles

insertion should be examined. In addition to identifying tendons that are torn, an estimation of retraction that each of these three tendons can be made. The axial imaging sequence will show the subscapularis tendon attachment, degree of retraction, and the amount of exposure of the footprint. The sagittal images can show the humeral attachments of all of the rotator cuff tendons well. In addition, this sequence medially demonstrates the quality of the muscles of the rotator cuff with regard to fatty infiltration and atrophy [28]. In this sequence medial to the humeral head, the spine of the scapula can be visualized with the supraspinatus muscle lying in the supraspinatus fossa (Fig. 24.8). If the muscular portion of the supraspinatus muscle is bisected by the line connecting the superior surface of the scapula and the superior surface of the spine of the scapula bisects the muscle, the muscle is normal or nearly so. If the line does not intersect the muscle at all, the rotator cuff may be inappropriate for repair because of fatty infiltration and atrophy. The MRI may be helpful to diagnose labrum, biceps, articular surfaces, and the acromioclavicular joint. MRI imaging can be variable in quality, and higher field magnets will produce superior quality of images. In recurrent rotator cuff tears, MRI is generally considered sensitive in diagnosing tears but has been shown to be less accurate than would be desired for determining the tear size and tends to overdiagnose tears in postoperative patients [29].

CT scanning can be a useful adjunct in shoulder evaluation. Contrast can be used to assist in evaluating the rotator cuff especially in cases where previous metallic hardware obscure MR imaging. In revision settings, the previous hardware can be best evaluated with CT scanning in many cases [30]. The quality of the muscle in massive rotator cuff tears appears to be better noted in MR imaging when compared to CT arthrography (CTA) [31]. In revision cases where there is metal hardware in the humerus, tendon visualization is obstructed by artifacts, and MR arthrography (MRA) has been shown to better demonstrate partial-thickness rotator cuff tears [32].

Ultrasound is a technique to evaluate rotator cuff integrity. The rotator cuff insertion can be reasonably well seen with ultrasound, but accuracy of ultrasound has been demonstrated to be less than that of MR imaging in recent studies [33]. The accuracy of imaging of rotator cuff tears that are full thickness with ultrasonography is typically better than the estimation of partial-thickness tears in inexperienced hands [34]. The quality of the muscle in rotator cuff tears has not traditionally thought to be as well noted as in MR imaging via ultrasound, but this has been recently disputed in the literature [35]. One large advantage of ultrasound over MR is the ability to see the rotator cuff in the setting of hardware. Hardware can occasionally make evaluation with MR more challenging. Bilateral shoulder evaluation can be performed to make comparison. Ultrasound also provides a much cheaper and faster evaluation of the cuff, but its utility has been clearly associated with user experience.

Our typical imaging protocol in massive rotator cuff tears includes plain radiographs to evaluate degenerative changes and other pathology. In addition, plain radiographs can easily evaluate acromial morphology and migration of the humeral head. MRI is used as well to most accurately evaluate the extent of the tear of the rotator cuff, the amount of retraction, and the quality of the muscle in addition to other pathology. CT with or without arthrogram is added to evaluate hardware placement in revision, especially when metallic implants have been previously used, and is a common technique in Europe. Ultrasound is typically reserved for post repair evaluation as a screening tool and as a dynamic study with humeral head rotation.

Treatment: Indications and Contraindications

Patients with large and massive rotator cuff tears may be selected for cuff repair if there is a reasonable chance for improvement of their symptoms. The most common reason a patient is considered for surgical repair is pain. Some patients present with exacerbation of their symptoms after a moderate trauma due to an extension or enlargement of their tear. Other patients have a significant increase in their pain due to symptoms resulting from the long head of the biceps. A more

recent change in the patient's pain or function can improve the prognosis and reparability of the muscle and tendon. Chronicity may lead to significant fixed tissue retraction, muscular atrophy, tendon deterioration, and loss of flexibility of the soft tissues. In carefully selected patients, an improvement in patient's pain can be anticipated after arthroscopic rotator cuff repair [36, 37]. Some elderly patients with multiple morbidities may experience pain relief from a biceps tenotomy, avoiding the postoperative restrictions [38].

A measureable change in active elevation may be due to pain, weakness, or loss of glenohumeral superior stability. Early repair of the subscapularis insertion and the posterosuperior cuff can restabilize the shoulder, allowing for improved force couples which assist in shoulder elevation. Some patients that cannot achieve a complete "watertight" closure have experienced pain relief and improved motion following arthroscopic rotator cuff repair. Residual defects of the supraspinatus often demonstrate weakness, but pain relief has been reported [39, 40]. Common surgical procedures including subacromial decompression or acromioplasty should be limited, to avoid potential anterosuperior escape in these patients.

Contraindications for arthroscopic repair are patients who have anterosuperior escape, true pseudoparalysis of the shoulder, and those that cannot comply with the postoperative restrictions. Certain patients have true pseudoparalysis and are unable to lift their arm, even if pain can be reduced due to chronic cuff deficiency. This is often due to the anterosuperior escape of the humeral head through the cuff defect combined with inadequate containment beneath the acromion and anterior arch structures. In an acute setting of less than 3 months, younger, more active patients can consider an attempt at repair. As these tears become more chronic, there is less chance of reversal of these findings. Neurological disorders may mimic rotator cuff disease and include cervical disc herniation, brachial plexopathy, syringomyelia, and central cord syndrome. Another contraindication is advanced arthritic joint changes. As passive motion becomes limited due to these changes, rotator cuff repair would have little benefit to functional gains.

There are several elderly patients who have moderate osteopenia at the time of their presentation. The addition of a cuff repair may exacerbate joint compression forces and lead to humeral head collapse. Avascular necrosis has followed rotator cuff repair, and this may be multifactorial. It would not appear to be directly related to potential vascular compromise from suture anchors. Older female patients with osteoporosis should be considered at risk for developing avascular necrosis and humeral head collapse following repair of large cuff defects.

A lidocaine injection may be helpful in determining the extent of weakness and the role of pain. Certain patients may have an improvement in their active motion and therefore considered for arthroscopic repair. Patients with pseudoparalysis, superior fixed-head migration, and inability to comply with a postoperative sling and require assistance of arms with ambulation are poor candidates for rotator cuff repair.

Arthroscopic Treatment: Surgical Technique

Patient Positioning

Patients undergoing surgical repair of a large or massive rotator cuff repair should have near normal passive motion preoperatively. The surgery begins with an anesthetized supine patient. The range of motion is tested, and any deficits are reversed with gentle manipulation. Emphasis on full elevation, abduction external rotation, cross-chest adduction, and internal rotation is performed. The patient can be positioned in either the lateral decubitus or beach chair position, depending on the surgeon's preference.

Portals

The standard diagnostic posterior portal is created after needle localization. This is generally placed two centimeters inferior to the angle of the acromion and the spine of the scapula. An anterior portal is developed anterior to the acromioclavicular joint, entering into the rotator interval. Irrigation and minor debridement is performed. The diagnostic exam begins with visualizing the biceps, labrum, and subscapularis. The scope is then reinserted in an anterior portal, and the posterior margin of the tear is visualized. Posteroinferior and inferior capsulotomy can be performed at this time. A brief bursal exam from the posterior portal can determine rotator cuff quality and retraction.

Step-by-Step Procedure

The initial articular repair begins with the subscapularis. A capsulotomy allows for tissue visualization and mobility. Open the soft tissue window superior to the upper border of the subscapularis. Soft tissue releases along the lateral margin of the coracoid can be performed, and in select patients, a coracoplasty can be performed to assist in tissue mobilization. Gentle shoulder internal rotation and posterior translation will open the working space during these steps. The scope can remain in the posterior articular portal or be switched to an anterolateral portal for a bursal approach. Anchors are placed from inferior to superior along the lesser tuberosity. A combination of simple and mattress sutures can be placed for a stable repair (Fig. 24.9). The superior border

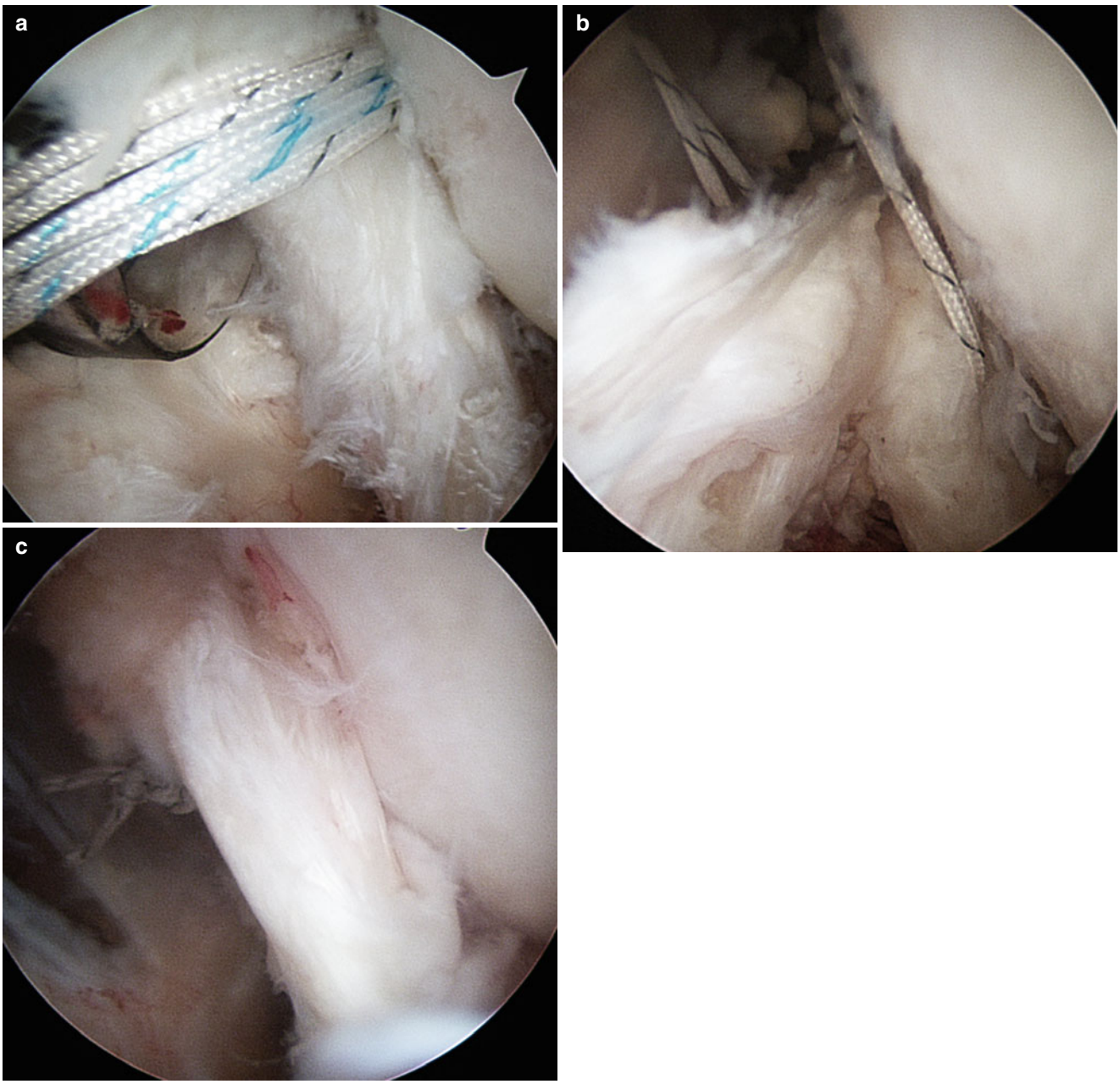

Fig. 24.9 The subscapularis repair. (**a**) Anchor placement on the lesser tuberosity. (**b**) Sutures retrieved through the detached tendon. (**c**) Completed single-row repair using mattress and simple sutures

of the subscapularis is comprised of tendon, and this provides secure fixation when repaired at the upper medial border of the lesser tuberosity.

The scope is switched to the subacromial bursa. A bursectomy is performed to improve visualization. It is common to place a suture in the long head of the diseased biceps and release it from the superior labrum. A lateral portal is created three centimeters lateral to the anterior portion of the acromion. Extensive debridement of the soft tissue below the acromion is performed and extends anteriorly to separate the deltoid, coracoacromial ligament, and the cuff, as well as the posterior interval between deltoid and the cuff. The greater tuberosity is gently debrided, and the scope is switched to the lateral viewing portal. The anatomy of the posterior cuff is visualized and the cuff is mobilized. The surgeon should determine the shape of the cuff tear (i.e., crescent, reverse L-shaped). Medial extensions of the tears are repaired with margin convergent sutures. The lateral convergence sutures are placed and can be left untied to allow for access to the tuberosity.

The infraspinatus is repaired with an anchor placed along the posterior margin of the greater tuberosity (Fig. 24.10).

Cuff delamination requiring multiple layers of repair is beneficial for return of function. A lateral view of the defect allows the posterior portal to be used for a piercing instrument to penetrate the superficial and deep layers and create a series of mattress and simple sutures. These sutures can be tied to secure the posterior pillar of the repair.

An anchor is placed along the medial edge of the tuberosity along the anterosuperior defect (Fig. 24.11a). Mattress sutures are passed through the supraspinatus. Patients with tendon shortening need to avoid over-tensioning of the repair. As sutures are tied, the supraspinatus and interval tissues are secured.

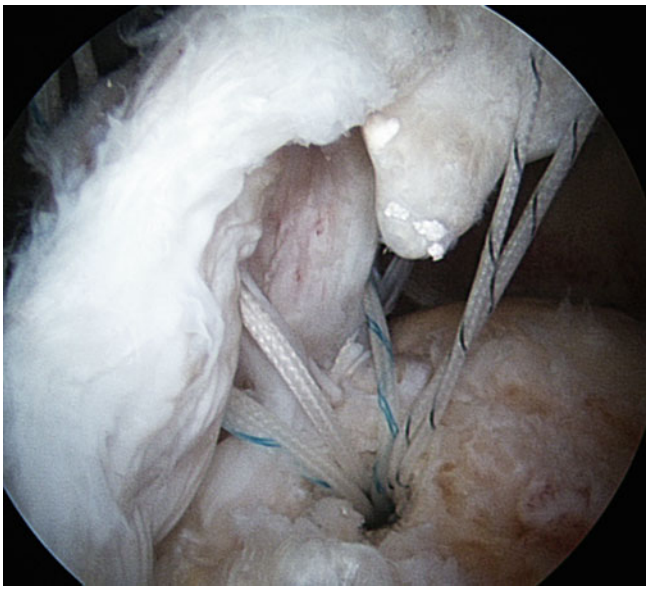

Fig. 24.10 The posterosuperior repair with suture anchor placed on the posterior margin of the greater tuberosity and multiple layers of infraspinatus tendon incorporated into the repair

The long head of the biceps is affixed to the posterior anchor. This additional tissue can be used to augment the supraspinatus repair and coverage of tuberosity and create additional fixation. Anchors combined with the transferred biceps may add fixation to the superior construct. The anterior greater tuberosity anchor can be reinforced with a lateral anchor, often a knotless design. This creates additional fixation, tissue compression, and support to a critical region of stress (Fig. 24.11b).

Subacromial arch debridement and minor decompression can be helpful to visualize and mobilize tissues. Preservation of the coracoacromial ligament is important to minimize the risk of anterosuperior escape. Decompression of the acromioclavicular joint is helpful for pain relief, anterosuperior tissue mobilization, and release of the coracohumeral ligament. Bone removal with preservation of capsular ligaments and coracoacromial ligament attachments are important to early results and potentially future surgery.

Suprascapular nerve decompression has become a topic of debate in patients with a painful, weak shoulder combined with atrophy and a cuff tear [20]. Preoperative evaluation including electromyography and nerve conduction velocity is helpful to discover compromise of the innervation to the infraspinatus or both the supraspinatus and infraspinatus. Using the lateral portal, the bursal view of the coracoid can be palpated from lateral to medial. A portal posterior to the

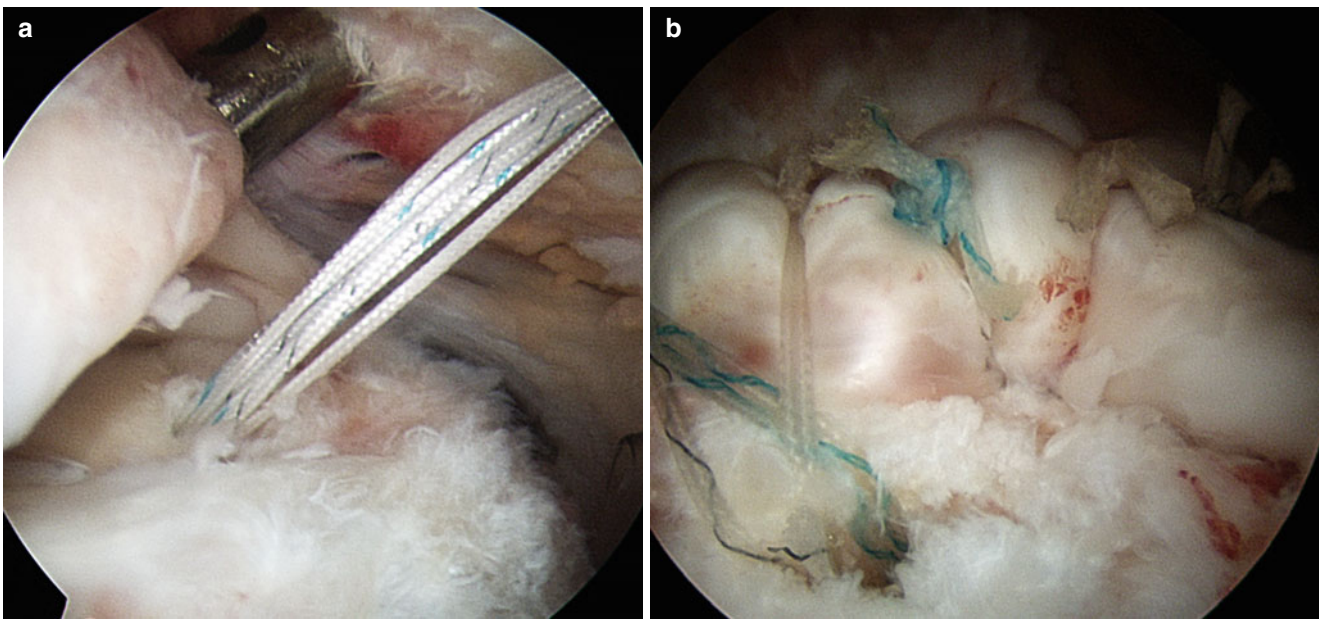

Fig. 24.11 The supraspinatus repair. (**a**) The tendon is fixed with an anchor adjacent to the articular surface. (**b**) A lateral anchor is added to compress the tendon and reduce the tension on the repair

acromioclavicular joint is created to pass instruments to retract the muscle belly of the supraspinatus. As the coracoid base is exposed, the coracoclavicular ligament attachments are visualized. Gentle elevation will expose the transverse ligament covering the suprascapular notch. After protection of the suprascapular artery and the nerve, this ligament can be carefully divided, freeing the nerve. Probing this passage will free any remaining bands of tissue.

Postoperative Care

Patients are placed in a sling with a small abduction pillow that is worn for 5–6 weeks. The initial exercises include hand grips, elbow flex and extend, and pendulum exercises. Shoulder retraction isometrics will tone scapular stabilizer muscles.

After five weeks, table slides, supine passive flexion, gentle assisted external rotation are performed with the assistance of physical therapy. The emphasis is on recovering pain-free range of motion. As this gradually improves, patients are repositioned upright and similar exercises continue. At 10–12 weeks, the active motion increases, and cross-chest stretching is added.

After 12 weeks, the gradual increase in active motion is followed by resistive strength training, both closed- and open-chain exercises. Strength gains are often not appreciated until 5 and 6 months following repairs. More advanced strength training to the cuff rotators and scapular stabilizers continues between 6 and 12 months. Resistive exercises emphasize daily moderate resistance with repetition and multiple sets. Core strength and scapular strength will assist in the patient's recovery. Return to sports and physical activity is based on patients' level of comfort, strength, and demands of their activities.

Literature Review

The large and massive rotator cuff tear has been a challenge to repair. Both open and arthroscopic surgeons have developed techniques to repair detached and retracted tendons. Overall, patient satisfaction following surgery has been encouraging. Follow-up imaging studies have demonstrated persistent defects in some repairs, which do not always reflect the level of satisfaction of the patient [14, 37, 41].

There are many citations describing patients' clinical outcomes following repair of large defects. O'Holleran et al. prospectively studied 311 patients in an analysis of satisfaction following surgery. They found a significant decrease in satisfaction in patients who had larger tears and in patients with massive irreparable tears [42]. Ito and Morioka compared patients treated with a McLaughlin procedure to patients treated with a patch graft and found a lower rate of re-tearing with a patch graft. Both groups had a significant increase in shoulder scores following surgery using the Japanese Orthopedic Association (JOA) score [43]. Oh et al. addressed the issue of the rising popularity of performing primary reverse total shoulder arthroplasty (RTSA) for large-to-massive cuff tears by looking at the success rate of rotator cuff repair in cases of painful pseudoparalysis. In their study of 195 complete repairs of large-to-massive tears, they found significant improvement in forward elevation, as well as multiple functional outcome scores when performing cuff repair. They concluded that primary cuff repair, rather than reverse total shoulder arthroplasty, should be attempted first in cases of large-to-massive tears with pseudoparalysis [44]. Hollinshead et al. recommended the use of the rotator cuff quality of life measure (RC-QOL) and the Functional Shoulder Elevation Test (FSET) as outcome measures for rotator cuff disease. Their study in 2000 showed that these two measures are able to distinguish between large and massive cuff tears, whereas the traditional SF-36 score is unable to make this distinction [45]. Gerber et al. reported that while repair of massive rotator cuff tears could be successful in terms of clinical outcome, this did not necessarily correlate with reversal of atrophy and fatty degeneration of the cuff musculature [5]. Lafosse et al. found that large and massive rotator cuff tears could be successfully repaired using an arthroscopic double-row suture anchor technique. They also found, however, that even with successful repair, patients with large-to-massive tears had more postoperative weakness than did patients with small tears [46]. Lo and Burkhart have described a technique of arthroscopic rotator cuff repair using single and double interval slides for successful management of massive, contracted, immobile rotator cuff tears [36]. Other authors have presented on arthroscopic techniques that can address massive tears with primary repair or reconstructive techniques using allograft to reinforce the repair [47, 48].

Patients presenting with large or massive cuff tears have a reasonable success rate when the glenohumeral joint has minimal degenerative changes, the tear is mobile, the muscular changes are mild to moderate, and the patient has a stable glenohumeral articulation [37]. Anterosuperior escape in a chronically deficient shoulder will present with pseudoparalysis. Shoulders with a fixed deformity will need to consider other options for treatment for predictable pain relief and arm elevation [45, 49].

Summary

Patients can develop large and massive rotator cuff tears acutely or as the result of an acute extension of a chronic, well-tolerated cuff tear. The presentation includes pain,

loss of strength, and decrease in function. Arthroscopic management of the torn cuff includes repair, treatment to associated biceps pathology, and decompression of the acromioclavicular joint in selected patients. Anticipated results include a significant improvement in pain relief, improved active motion, and return of function.

Biceps tenotomy can be helpful in pain management in the elderly patient or a patient that has acceptable active motion and cannot participate with postoperative restrictions and rehabilitation. Partial cuff repair of the subscapularis and infraspinatus can restore the force couple and improve the mechanics of the deltoid as an elevator. The biceps or other tissue graft can complete the closure if the supraspinatus is deficient and retracted. Suprascapular nerve release may be included to assist in pain relief and possibly improve function in patients with preoperative deficits. Arthroplasty alternatives should be selected in appropriate patients with arthritic changes of the glenohumeral joint, failed attempts at cuff repair, joint instability, and chronic pseudoparalysis.

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Disorders of the Rotator Interval: Coracohumeral Ligament and Biceps Tendon

25

Michael O'Malley, Knut Beitzel, and Augustus D. Mazzocca

Epidemiology

From the time Neer first described the anatomy of the rotator interval in 1970, our understanding of the biomechanical role of its contributing structures has continued to evolve [1]. This is in part due to a better understanding of the disability recognized in rotator interval pathology. Neer initially described the boundaries of the rotator interval as the space between the subscapularis and supraspinatus tendons. Neer and Foster followed this work with further studies providing evidence of the rotator intervals role in glenohumeral stability [2]. In 1981, Rowe and Zarins [3] described how instability at the shoulder may be in part secondary to various sizes of the rotator interval. The first large series of patients with rotator interval pathology was reported by Nobuhara and Ikeda in 1987, describing its role in both shoulder instability and adhesive capsulitis ("frozen shoulder") [4]. Slatis and Aalto described how the structures of the rotator interval play an important role in stabilizing the long head of the biceps tendon [5].

The prevalence of rotator interval pathology is difficult to quantify as it is thought to be related more to the function of its individual structures rather than the group of structures as a whole. However, Rowe and Zarins [3] found that in "normal" cadaver shoulders, 9 % demonstrated rotator interval lesions. This is in contrast to those with recurrent instability where 54 % demonstrated rotator interval lesions and required surgical stabilization. Similarly, Petersson found that 3.3 % of cadaver shoulders demonstrated medial biceps dislocation, most of which were associated with injury to the subscapularis [6].

Pathophysiology

The rotator interval is a triangular area in the anterosuperior aspect of the glenohumeral joint. Its boundaries are defined by multiple structures in the shoulder. The anterior margin of the supraspinatus (SSP) tendon forms its superior border, while the inferior border is defined by the superior margin of the subscapularis (SSC) tendon. The coracoid process forms the medial base of the rotator interval. Within this triangular area are the following structure: the superior glenohumeral ligament (SGHL), the middle glenohumeral ligament (MGHL), the coracohumeral ligament (CHL), the long head of the biceps tendon (LHB), and the anterior capsule.

To better understand the configuration of these structures around the shoulder, Jost et al. provide a most detailed description based on meticulous dissection of cadaver specimens [7]. The authors separated the rotator interval into medial and lateral regions, with the medial consisting of two layers and the lateral consisting of four layers. Medially, the CHL comprises the superficial layer, while the SGHL and anterior capsule form the deep layer. Laterally, the fan-shaped fibers of the superficial CHL follow the supraspinatus and subscapularis tendons to their humeral insertions to form the first layer. The second layer is composed of the supraspinatus and subscapularis fibers, with the subscapularis forming the roof of the bicipital groove. The deep fibers of the CHL form the third layer, and the fourth layer is comprised of the SGHL and the lateral joint capsule.

The SGHL originates from the anterosuperior labrum adjacent to the supraglenoid tubercle and crosses the floor of the rotator interval deep to the CHL before forming a U-shaped sling laterally underneath the LHB tendon and inserting into the proximal aspect of the lesser tuberosity known as the fovea capitis [8–10]. The SGHL, CHL, as well as contributions from both the supraspinatus and

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subscapularis tendon form what is known as the biceps reflection pulley. Although Walch initially described the SGHL and CHL as the primary stabilizers to the intra-articular aspect of the LHB tendon, more recent literature suggests that the subscapularis tendon is the primary stabilizer for LHB tendon [11].

The CHL has an irregular trapezoidal shape and serves as the major superficial layer of the rotator interval. It is a dense fibrous band originating medially from the lateral base of the coracoid process. Laterally it divides into two parts, one inserting into the greater tuberosity and the other inserting into the lesser tuberosity. Its insertion is often difficult to delineate as it blends with other structures laterally prior to its bony point of insertion [10]. Histologic studies have shown tissue more similar to capsule rather than ligament, and the controversy remains between those who feel the CHL is merely a capsular thickening or fold, and those who argue that it is a distinct ligament [10].

The LHB tendon originates with variable fiber insertions from the superior labrum and the supraglenoid tubercle. The majority of these fibers arise from the posterior aspect of the superior labrum [12]. Its overall length is approximately 9 cm with a width of 5–6 mm. The intra-articular portion is extrasynovial with a length measuring 34.5 ± 4.2 mm. In examining both neural and vascular supply to the LHB tendon, it was found that the first 3 cm of the intra-articular portion of the tendon has regions with significant hypovascularity, while the proximal 3 cm has a rich neural innervation [13]. The fasciculus obliquus, or the "longitudinal oblique system," is a structure not commonly mentioned as part of the rotator interval but has a close relationship with the structures of the rotator interval. It lies in the superficial layer of the anterior joint capsule and upon gross anatomic dissection resembles a spiral band. Its origin is from the infraglenoid tubercle and the long head of the triceps. It courses cranially before fusing with the MGHL, IGHL, and the posterior-superior aspect of the subscapularis. Its deep layer merges with parts of the SGHL and CHL at the bicipital groove. It then inserts on the lesser tuberosity of the humerus [14]. Its presence is worth mentioning as it serves as a reinforcing structure for the biceps pulley system, which consists of the CHL, SGHL, and fibers of the supraspinatus and subscapularis.

Recent studies have provided insight to the function of the rotator interval and its contributing structures. Nobuhara and Ikeda showed that by externally rotating the shoulder, the rotator interval is itself tightened, resulting in a decrease in posterior-inferior shoulder instability [4, 15, 16]. This evidence is supported by Harryman et al. [17] in their cadaveric study, in which the authors sectioned the structures of the rotator interval to derive conclusions about its exact function. Results of sectioning showed an increase range of shoulder flexion, extension, adduction, external rotation, as well as an increase in humeral head translation in the posteroinferior direction. Conversely, upon imbrication of the rotator interval, these motions were decreased. This was supported by Plausinis et al. [18] who showed that arthroscopic closure of the rotator interval resulted in an 11° loss of external rotation. Stated conclusions included the rotator interval serves as a "check rein" to excessive shoulder motion and stabilizes against inferior humeral head translation with the arm adducted and posterior translation with the arm flexed, abducted, and externally rotated.

The rotator interval also serves the ever important role of stabilization of the LHB tendon. As mentioned prior, structures of the rotator interval form a sling or reflection pulley that support the biceps tendon as its exist the glenohumeral joint. There are multiple studies showing the individual contributions of the structures forming this pulley system, including the SGHL, CHL, and fibers of the SSP and SSC. Petersson was the first to describe anteromedial dislocations of the LHB tendon in the setting of subscapularis tendon tears [6]. Walch et al. [11] describe "hidden" lesions of the lateral CHL, SGHL, and SSC tendon in association with SSP tendon tears resulting in LHB tendon dislocation. The label "hidden" was applied as many of the lesions were missed during both arthroscopic and open procedures. Additionally, defects in the capsular portion of the rotator interval may cause a loss in intra-articular pressure, which is associated with glenohumeral instability.

Multiple intra-articular lesions of the shoulder joint interact with lesions of the rotator interval and the LHB such as bursitis, rotator cuff tears, SLAP, and AC-joint disorders. For the LHB, primary tendinitis and ruptures have also been observed. Rupture almost always occurs near the insertion or at the proximal intertubercular groove. If the rupture is distal to the insertion, the stump can become incarcerated in the joint. Tendonitis of LHB, which has been recognized for over 50 years, has been increasingly reported as either an isolated source of shoulder pain or occurring in combination with one (or more) of the aforementioned disorders. An "hourglass biceps" in which a hypertrophic portion of the intra-articular segment of the LHB cannot slide during elevation of the arm, resulting in incarceration of the tendon, has also been described as a lesion of the biceps tendon complex [19].

History

A patient with rotator interval pathology may have various presentations. Rotator interval laxity can be seen in the setting of either acute traumatic injury or chronic overuse injury. Typical complaints consist of apprehension or gross instability in certain planes of motion, as well as early fatigue of the affected extremity.

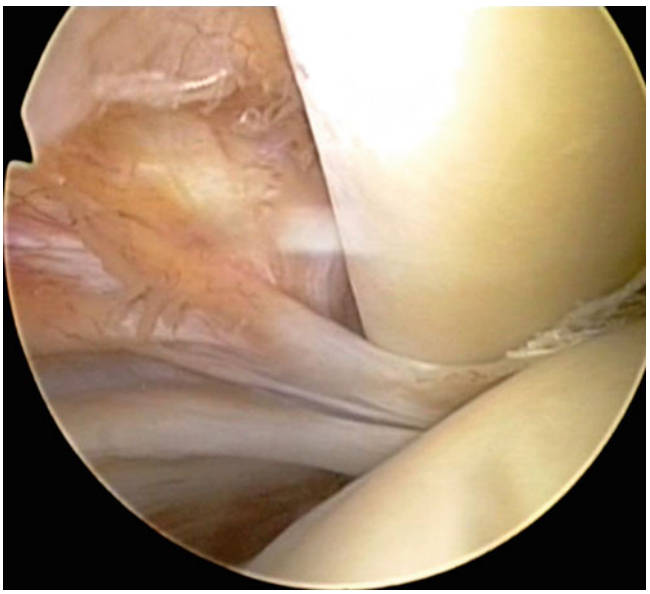

Fig. 25.1 Arthroscopic view of right shoulder with signs of synovial reaction of the rotator interval in a frozen shoulder

A spectrum of disease states can result from contractures of the rotator interval, from mild rotator cuff impingement to adhesive capsulitis [8]. Clinically, patients present with limited painful active and passive range of motion, with pain at rest and difficulty sleeping secondary to night pain. It is more common and responds less to both conservative and invasive measures in the diabetic population. It may occur with no particular inciting event or in the postoperative period (Fig. 25.1).

Patients with problems involving the LHB tendon typically present with anterior shoulder pain. At times this pain may radiate down the arm following the course of the biceps muscle. LHB tendinitis is typically seen in those >50 years of age presenting with the aforementioned complaints, in addition to pain with resisted elbow flexion or forearm supination. Rotator cuff lesions must be considered in the setting of biceps injury, especially if significant strain is placed on the arm. In overhead athletes and those reporting traction injuries to the shoulder, lesions of the superior labrum (SLAP) should always be considered. Patients may report a sensation of "snapping" in the anterior shoulder with abduction and external rotation of the arm or an audible painful "pop" upon sudden eccentric or concentric muscle contraction followed by the typical presentation of a "popeye" arm as the muscle retracts. In this setting patients often describe an onset of pain prior to the injury, followed by relief upon tendon rupture.

Coracoid impingement is a well-known but less common cause of anterior shoulder pain. The typical patient is one complaining of dull anterior shoulder pain with attempted forward flexion, adduction, and internal rotation of the shoulder. Pain is often recreated in the midrange of flexion. It is not uncommon for patients to complain of pain with attempting a push-up exercise as well. As described by Gerber et al., although this disease entity may be idiopathic in nature, it is also seen in the setting of prior trauma, instability, or iatrogenic injury. The source of the pain is thought to be secondary to impingement of the subscapularis between the coracoids process and the lesser tuberosity of the humerus [7].

Clinical Examination

Obtaining a thorough history is critical, allowing the examiner to tailor their exam to the suspected pathology. Adhesive capsulitis exhibits painful limited active and passive range of motion in all planes, especially in external rotation. Abnormal scapulothoracic motion may be seen, compensating for lack of motion at the glenohumeral joint. Discomfort is commonly localized to the point of deltoid insertion. Tenderness around the area of the coracoid may be seen as well.

Multiple studies have demonstrated the contribution of the rotator interval to humeral head translation and the overall stability of the shoulder [7, 17, 20]. The majority of patients on exam will exhibit inferior instability, oftentimes with associated anterior or posterior instability as well. A persistent inferior sulcus sign with the arm held in external rotation is strongly suggestive of rotator interval laxity. If the sulcus sign disappears with external rotation of the arm, the rotator interval is most likely intact [17]. Anterior or posterior apprehension signs may be present on exam and must be evaluated.

Tenderness along the superior aspect of the muscle belly is often seen in LHB tendon disorders. Speed's test is used to isolate the LHB tendon and the patient will report pain in the bicipital groove when the elbow is flexed against resistance [21]. Geaney and Mazzocca described the subpectoral biceps tendinitis test in which the examiner has the patient adduct and internally rotate the effected arm against resistance, localizes the inferior border of the pectoral tendon, and palpates the biceps in the axilla under the pectoralis tendon [21]. In those with biceps tendonitis, this test should recreate the pain. Similarly, Bennett described a provocative test aimed at causing subluxation or dislocation of the biceps tendon from the bicipital groove. With the arm not allowed to exceed 90° of elevation, the patient is asked to passively move the arm from an abducted-externally rotated position to one of cross-body adduction-internal rotation [22]. If the patient experiences a catching or snapping sensation at the area of the proximal biceps, the test is considered positive. Rotator cuff pathology also needs to be considered in the setting of bicep tendon injury, especially tears of the subscapularis and supraspinatus. This is particularly important in the setting of

proximal LHB tendon rupture in which the patient oftentimes presents with a "popeye" arm. Range of motion of the glenohumeral joint must always be assessed, in this case to rule out a mechanical block in motion secondary to an incarcerated biceps tendon stump.

In coracoid impingement, patients describe pain with forward flexion, adduction, and internal rotation, during which the lesser tuberosity is brought into contact with the coracoid. Tenderness is elicited upon palpation of the soft tissues surrounding the coracoid and lesser tuberosity.

Imaging

Various imaging modalities may be utilized to examine the structures of the rotator interval. In the setting of contracture, plain radiographs are often normal. Arthrography may show a decrease in overall capsular volume. In comparison to normal shoulders, Kim et al. [23] described magnetic resonance imaging (MRI) studies showing a disappearance of the normal subcoracoid fat plane and rotator interval height and area in comparison to normal shoulders. In their study utilizing noninvasive ultrasound imaging, Homsi et al. identified thickening of the CHL and joint capsule in comparison to normal age- and sex-matched controls [24]. These findings are confirmed on diagnostic arthroscopy, often demonstrating decreased capsular volume and a thickened, fibrotic capsule and CHL [25].

Plain radiographs are typically normal in the setting of rotator interval laxity, however subtle inferior humeral subluxation may be appreciated. Arthrography may show extravasation of contrast through the rotator interval in the setting of a defect or more commonly filling of the redundant capsule with the arm elevated in external rotation. MRI has gained popularity in that it may show increased interval size in those with instability, secondary to its enhanced soft tissue detail. In diagnostic arthroscopy, some authors have assessed capsular redundancy as the distance between the SSP and SSC in normal shoulders at baseline and with joint distention, such that any shoulder with measurements exceeding this are considered lax [26]. In addition to this capsular redundancy, Gartsman et al. reported other findings during arthroscopy suggestive of rotator interval lesions include biceps tendon fraying, tearing of the SGHL, and fraying of the superior edge of the subscapularis tendon [27].

There is no role for plain radiographs in the setting of LHB tendon pathology. Incidental findings of subtle cystic erosions of the lesser tuberosity may be seen; however, this modality is typically not helpful. MRI is considered the best modality for evaluating LHB tendon pathology. Buck and Zanetti recommended an intra-articular injection of 12 ml of 2 mmol of gadolinium to improve the accuracy for detecting lesions of the LHB tendon, rotator cuff, SLAP tears, etc. [28]. Some authors, including Bennett, feel that no study allows for clear evaluation of these lesions and that surgery alone is the only reliable means of identifying pathology.

Ultrasound is an easy and widely available method of imaging that has the advantage of a possible dynamic examination. The LHB tendon can be evaluated within the sulcus and the tendons of the rotator cuff as well as the tendon entering the pulley sling can be visualized. Tendinitis is often accompanied by fluid enhancement in the sulcus, although this cannot be seen as specific sign. However, the examiner needs to be skilled with the technique to achieve optimal results. If this is the case, especially lesions within the sulcus and medial subluxation of the biceps tendon towards the subscapularis muscle are reliable to detect. Ultrasound is regarded to be an optimal imaging for the detection of LHB subluxation and dislocation, but it is unreliable for intra-articular partial thickness lesions of the tendon [28–30].

Decision-Making Algorithm

Thus far, no widely accepted classification system has been established for rotator interval pathology. Fitzpatrick et al. described a system based on the specific anatomic structure involved, including lesions to the capsule, CHL, SGHL, anterior SSP, superior SSC, and the LHB tendon [31]. Mechanical strength of the lesion was used by Nobuhara and Ikeda as a means of classifying these lesions [4]. Type I injuries were contraction and inflammation in the superficial tissues of the interval. In type II lesions, there is glenohumeral instability secondary to laxity in the deeper tissues of the rotator interval. Regardless of classification scheme, rotator interval pathology is closely related to the dysfunction of its individual structures and the function of the LHB.

Based on the descriptions by Gaskill et al. [8], lesions of the rotator interval and LHB can be broadly divided into four groups: (1) contractures of the rotator interval, (2) laxity of the rotator interval, (3) tears of the biceps reflection pulley combined with LHB instability, and (4) primary lesions of the LHB.

If a contracture of the rotator interval is present, symptomatic treatment is sufficient in most of the cases. Although the pathology is not yet fully understood, it is generally accepted as a self-limiting disorder. Gentle motion exercises and analgesia are the most important factors for nonsurgical treatment. If necessary, local or systemic application of corticoids may accelerate the healing process. Arthroscopic interval release may be considered after conservative treatment has failed for at least 3–6 months.

Increased laxity of the rotator interval may be combined with instability of the shoulder joint. Such laxity does clinically present with an increased sulcus sign, which does not disappear in external rotation. Since the reason for clinically

symptomatic laxity of the shoulder joint is seldom the laxity of the rotator interval alone, surgical treatment should consider these additional pathologies. However, an arthroscopic closure of the interval may be added to other stabilizing procedures if these are indicated and the sulcus sign does not disappear in external rotation. Isolated closure of the rotator interval is only indicated in very seldom cases.

Habermeyer et al. [32] classified lesions of the biceps reflection pulley into four types. Lesions of the SGHL result in anterior instability of the LHB tendon and are classified as type 1. Type 2 is a combined lesion of the SGHL with a partial rupture of the anterior portion of the supraspinatus tendon. Type 3 is defined as a combination of a SGHL lesion and a partial rupture of the cranial portion of the subscapularis tendon. Type 4 combines a lesion of the anterior supraspinatus tendon and a lesion of the cranial portion of the subscapularis tendon and is thought to be due to anteroposterior instability. Lafosse's arthroscopic classification takes into account the direction and extent of LHB tendon instability, macroscopic lesions of LHB, and concomitant lesions of the subscapularis and/or supraspinatus tendon [33]. If such combined lesions of the reflection pulley are present, surgical treatment is based on the additional lesions and tenotomy or tenodesis of the LHB may be performed. Surgical techniques, which tried to reconstruct the biceps reflection pulley have not shown sufficient results.

Nonoperative treatment is typically sufficient for treatment of spontaneous ruptures of the LHB. Patients electing for nonoperative care will have residual cosmetic deformity and may complain of cramping with strenuous activity. Cramping often resolves but may persist in some cases. Although there is controversy regarding operative versus nonoperative treatment, little objective data exist [34, 35]. In our experience, patients who opt for nonsurgical treatment generally do well with a home exercise program and rarely have stiffness. We encourage full range of motion, including overhead activity, to allow evaluation for an incarcerated tendon stump.

Some physicians may recommend surgical treatment to more active individuals or those who require more supination strength. Patients who opt for surgical treatment are typically laborers with dominant extremity injuries. They will often complain of an aching pain and spasm with repeated activity. Other patients who opt for surgery are young athletes and middle-aged patients who are not satisfied with the biceps deformity. If a patient chooses operative treatment, we prefer to conduct the surgery within 3 months of injury.

Boileau et al. investigated the effectiveness of biceps tenotomy and tenodesis in the setting of both rotator cuff tear as well as SLAP tear. In the setting of a irreparable rotator cuff tear associated with a biceps lesion, biceps tenotomy and tenodesis are effective means of treating pain and dysfunction [36]. Similarly, over 80 % of patients with an isolated type II SLAP tear who underwent arthroscopic biceps tenodesis were able to return to their previous level of sport. This is compared to only 20 % of those returning to their previous level of sport following a SLAP repair. Additionally, in those with a failed attempt at SLAP repair, who subsequently underwent biceps tenodesis, were also able to return to their previous level of sport, making biceps tenodesis an acceptable alternative for a failed SLAP repair [37].

Arthroscopic Treatment: Surgical Technique

Surgery may be performed in beach chair or lateral decubitus position of the patient. We commonly place the patient in beach chair and use a posterior standard portal to arthroscopically inspect the glenohumeral joint. A thorough evaluation of the joint for any additional lesions of the intra-articular structures should always be performed because of the high incidence of combined lesions (e.g., rotator cuff, instability). The LHB is inspected for its entire intra-articular length. The origin of the tendon and the superior labrum is tested with a probe after establishing an additional anterior portal to evaluate the biceps tendon anchor for SLAP lesions. The reflection pulley is evaluated for any lesions (hidden lesions of the SSC) or signs of instability (Figs. 25.2 and 25.3). Finally, the tendon is pulled out of the sulcus and the pulley structures are tested for their stability (Fig. 25.4).

Both biceps tenotomy and biceps tenodesis have been shown to be effective in treating biceps symptoms [37].

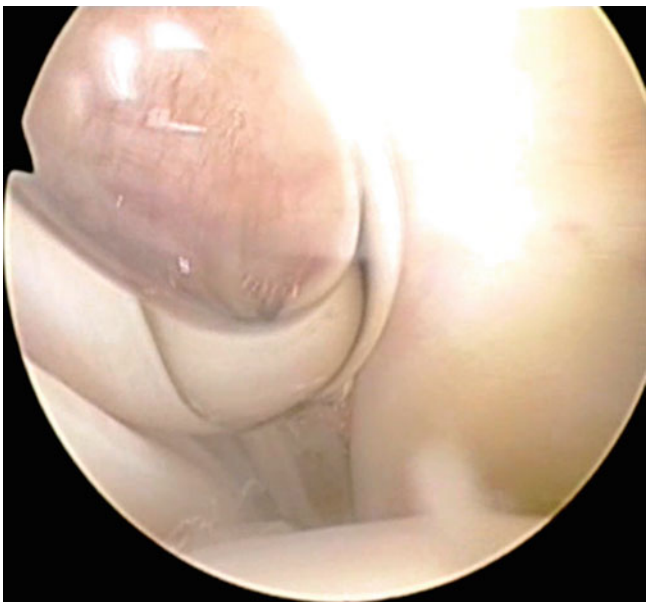

Fig. 25.2 Arthroscopic view of a left shoulder (posterior standard portal): note the laxity of the SGHL found in this type III lesion according to Habermeyer et al. [32]

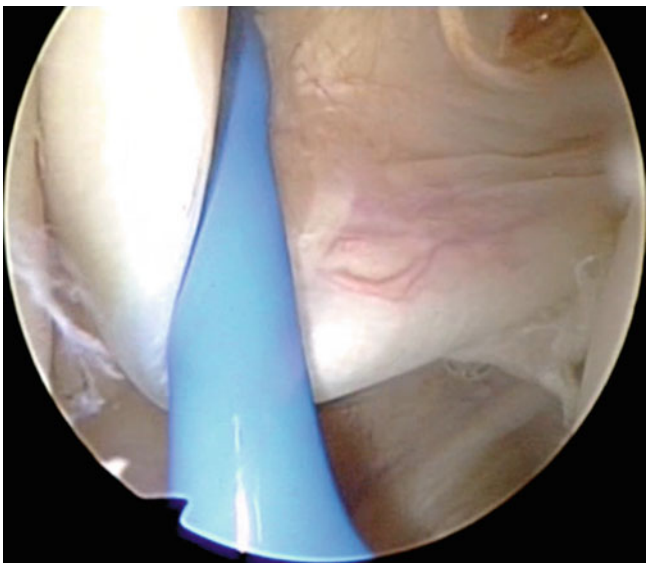

Fig. 25.3 Arthroscopic view of a left shoulder (posterior standard portal): note the posterior part of the pulley reflection system

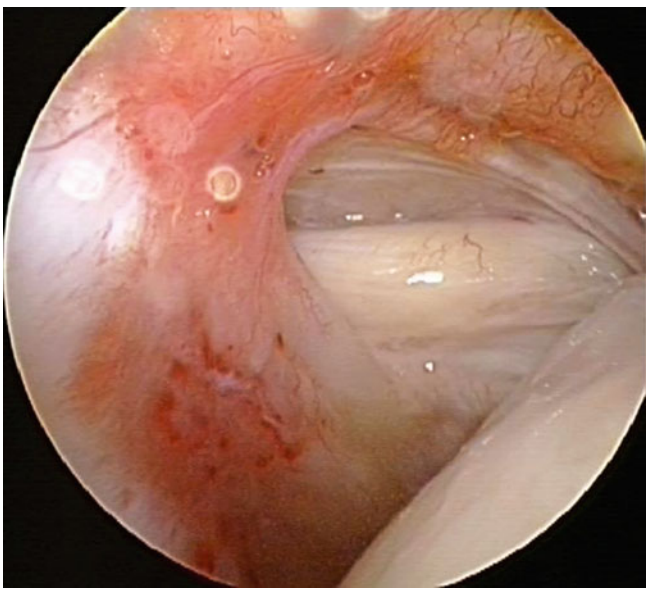

Fig. 25.4 Arthroscopic view of a left shoulder (posterior standard portal): The LHB tendon is pulled out of the sulcus to test for stability of the anterior reflection pulley

Today, tenotomy of the LHB is performed mostly arthroscopically [38]. A standard working portal is used to cut the biceps at the insertion with either an arthroscopic scissor or a duckbill cutting snip. The superior labrum should not be damaged to keep its function. After detaching the tendon, the surgeon should check that the tendon slipped into the sulcus. SLAP lesions in older adults may be debrided at this time as well. When opting for surgical treatment, biceps tenodesis remains the most common treatment in our experience. Multiple methods are available for tenodesis of the LHB tendon. These techniques can be distinguished according to the anatomic regions of fixation of the tendon stump.

The proximal tenodesis techniques are performed arthroscopically in nearly all cases and the tendon stump can be fixed with sutures, suture anchors, or tenodesis screws [21, 39]. Jayamoorthy et al. [40] compared repair techniques biomechanically. In comparison to interference screw fixation, the authors conclude that keyhole tenodesis was significantly stronger, while fixation with a bioabsorbable screw was not as strong. Keyhole tenodesis failed by tendon splitting and slippage, while interference screws failed exclusively by slippage. Mazzocca et al. [41] compared four different fixation techniques biomechanically: subpectoral bone tunnel, arthroscopic interference screw, subpectoral interference screw, and arthroscopic suture anchor. Bone tunnel showed significantly more cyclic displacement than the other three methods, and all three had favorable load to failure characteristics.

The advantages of these techniques are the preservation of the tendon length and the ability to perform the refixation without the need for an additional skin incision. Usually the proximal tenodesis is performed before concomitant procedures, such as RC repair. A standard posterior portal is used for initial evaluation of the joint and the LHB. A probe may be used to examine the intertubercular portion of the tendon by pulling the tendon into the joint. A needle is pierced through the tendon to place a shuttle suture. This suture can be used to shuttle a nonabsorbable suture through the tendon. At this point, various methods of fixation may be used. For soft tissue tenodesis, the tendon is fixed to the rotator interval. Tenodesis can also be performed using a suture anchor (e.g., 5.5-mm bioabsorbable Corkscrew FT Suture Anchor, Arthrex Inc., Naples, Fl) armed with 2 No. 2 FiberWire sutures or using a Bio-Tenodesis screw (e.g., 4.5-mm bioabsorbable SwiveLock anchor, Arthrex Inc., Naples, Fl). For these techniques, the anchor is placed in the entrance of the bicipital groove after decortication with a motorized bur. The suture anchor allows a fixation without pulling the tendon out of the anterior portal. In contrast, for fixation using a tenodesis screw, the tendon stump is pulled out of the anterior portal and loaded into the screw. In this case, transecting the tendon proximal to the sutures and close to its origin performs the tenotomy before fixation.

Our preferred arthroscopic technique utilizes a tenodesis screw to fix the tendon stump within the intertubercular groove (suprapectoral). The tendon is marked arthroscopically and a holding suture is placed through the tendon. The tendon is then cut close to its origin, without damaging the superior labral complex. The scope is moved to the subacromial space using the lateral portal. The falciform ligament of the pectoralis tendon is identified and the biceps tendon is found underneath. The accessory anterior portal is localized with a spinal needle. The proximal 20 mm of the tendon are

removed to eliminate diseased tendon from the tenodesis and recreate an anatomic fit. The proximal 15 mm of the remaining tendon stump is whip-stitched (alternative: Krakow stitches). The intertubercular groove is localized and a 2-mm guide wire is inserted. The guide wire is then reamed over with 7- or 8-mm cannulated reamer to a depth of 30 mm. The tendon is pulled out of accessory anterior portal and one suture is inserted through the tenodesis screw and tightened. Finally, the screw is inserted into the tunnel and an arthroscopic knot pusher is used to tie suture over the top of the tenodesis screw. New developments in screw design even allow for direct insertion of the tendon into the hole with the use of a forked tenodesis screw.

The author's preferred open technique is the subpectoral approach, which is close to the muscle belly of the biceps [42]. Even if initially ruptured, the biceps tendon generally will not retract beyond the point of fixation. Our procedure of choice is the subpectoral tenodesis with an interference screw. Prior to tenodesis, an arthroscopy is performed to identify any associated pathology and to tenotomize and debride the bicipital tendon stump.

With the arm abducted and internally rotated, the inferior border of the pectoralis major tendon is palpated. The incision is over the inferior border of the pectoralis tendon to 3 cm below the inferior border on the medial aspect of the arm. A scalpel is used to cut down through the subcuticular tissue. An electrocautery is used to control bleeding, a Gelpi or Weitlaner self-retaining retractor can be used for visualization. The fatty tissue is then cleared until the fascia overlaying the pectoralis major, coracobrachialis, and biceps is identified. If these anatomical landmarks are not seen, or if the cephalic vein is seen in the deltopectoral groove, the dissection may be too proximal and too lateral. Once the inferior border of the pectoralis major has been identified, the fascia overlaying the coracobrachialis and biceps is incised in a proximal to distal manner. A pointed Hohmann retractor is placed under the pectoralis major and on the proximal humerus to retract the muscle proximally and laterally. A blunt Chandler retractor is placed in the medial aspect of the humerus to retract the coracobrachialis and short head of the biceps (Fig. 25.5). Vigorous medial retraction should be avoided to prevent injury to the musculocutaneous nerve. The long head of the biceps musculotendinous junction should be visualized, and it is then withdrawn from the field. To ensure appropriate tensioning of the biceps tendon, the proximal portion of the tendon is resected to leave 20–25 mm of tendon proximal to the musculotendinous portion of the biceps (Fig. 25.6). One centimeter proximal to the pectoralis major tendon, the periosteum is reflected. A No. 2 nonabsorbable suture (e.g., FiberWire, Arthrex Inc., Naples, FL) is placed onto the tendon. Twelve millimeter of the tendon is secured to ensure that adequate fixation is maintained and that the musculotendinous portion of the biceps will sit

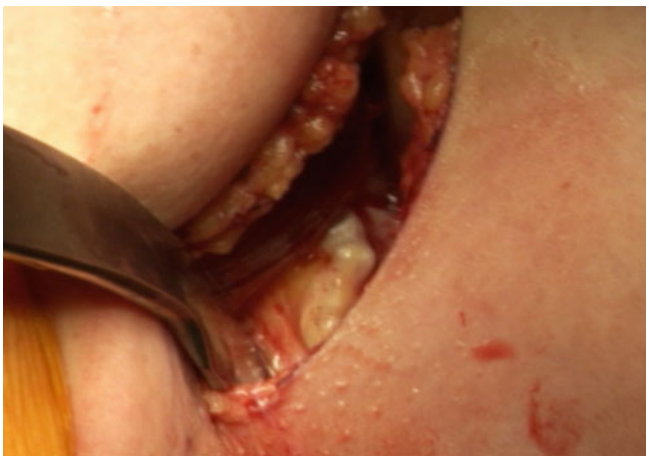

Fig. 25.5 Preparation of the subpectoral insertion for the subpectoral tenodesis. Note the LHB tendon in its place after it has been cut proximally

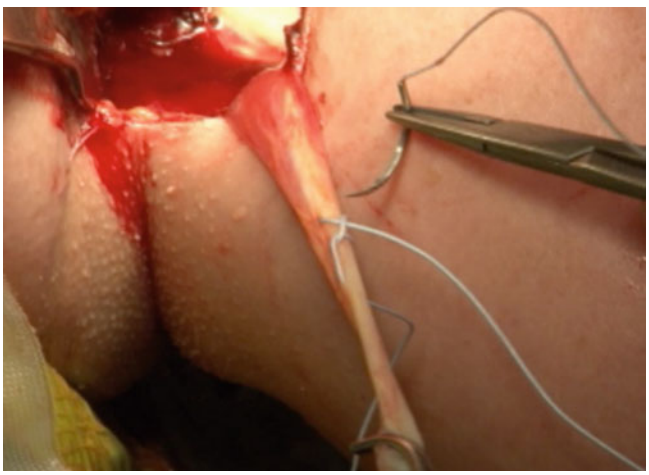

Fig. 25.6 Tendon is prepared for subpectoral tenodesis (2 cm proximal to the muscle)

underneath the inferior border of the pectoralis major tendon. This is critical for the proper tensioning of the muscle tendon unit as well as for cosmesis.

For most patients, an 8-mm cannulated reamer is of adequate size to allow placement of the tendon into the bone tunnel and secure fixation with an 8-mm bioabsorbable interference fit screw. The calibrated reamer is advanced over the guide pin to the 30-mm mark. Drilling beyond the posterior cortex of the humerus, which may increase the risk of complications during this surgical procedure is not necessary.

With a wire loop passed through the driver, one limb of the suture is pulled through the screw and the screwdriver handle (Fig. 25.7). The surgeon holds the other limb loosely. The limb that is passed through the driver is then pulled tightly, until the end of the tendon is securely placed against the tip of the driver. The tip of the driver is placed at the

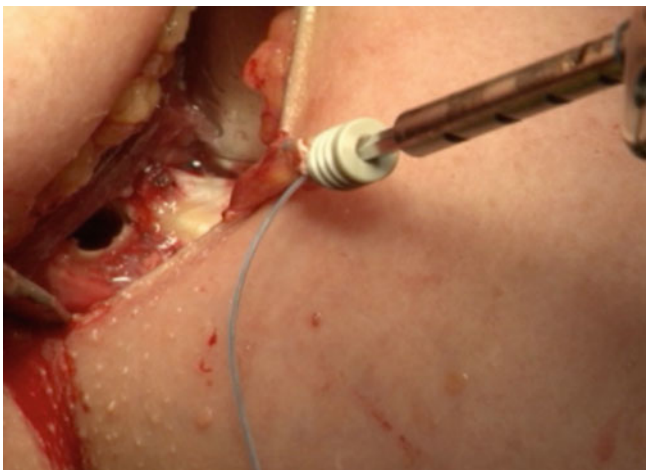

Fig. 25.7 Tendon stump is inserted for subpectoral tenodesis

superior aspect of the bone socket and manually inserted until the tendon reaches the base of the tunnel. The bioabsorbable interference screw is placed directly over the top of the tendon until the head of the screw is below the cortex.

Postoperative Care

The postoperative rehabilitation is mainly based on the additional lesions and surgical procedures, which are often combined to LHB surgery (e.g., rotator cuff repair). Following arthroscopic tenotomy without additional surgery, the patient should rest the arm for the first couple of days. Range of motion does not have to be restricted, but active loading of the biceps should be avoided for the first 4–6 weeks after tenotomy.

After tenodesis, patients begin with passive range of motion exercises but quickly progress to active-assisted and active range of motion. Full range of motion at the elbow and grip strengthening is allowed directly postoperative. Strengthening exercises involving elbow flexion or forward elevation of the arm should be restricted until 6 weeks after surgery.

Literature Review

Although no current consensus exists on the question whether the LHB tendon should be tenotomized or a tenodesis should be performed, both procedures have shown good clinical results as has been reported in the recent literature [35]. Gill et al. demonstrated significant reductions in pain and an improvement in function with a complication rate of 13.3 % after tenotomy [43]. A recent systematic review performed by Slenker et al. [35] showed comparable favorable results for both tenotomy and tenodesis. However, the only relevant difference shown may be more cosmetic deformity for the groups of biceps tenotomy. Osbahr et al. [44] found identical results in their study with 30 % of cosmetic changes with tenotomy. Additionally cramping and weakness with vigorous activities have also been reported [38]. There is no significant and clinical relevant difference in elbow flexion strength or strength of pronation and supination of the forearm [45]. Other authors have suggested tenotomy as effective for relief of pain related to LHB tendon lesions. In our opinion, indications for tenotomy are largely centered on older patients or patients demonstrating severe comorbidities and contraindications for tenodesis.

Summary

Lesions of the rotator interval and LHB can be broadly divided into four groups: (1) contractures of the rotator interval, (2) laxity of the rotator interval, (3) tears of the biceps reflection pulley combined with LHB instability, and (4) primary lesions of the LHB.

Contractures of the rotator interval are mainly treated nonsurgically and laxity of the interval is often combined with an anterior-inferior instability. Tendinitis and ruptures of the LHB tendon are well-documented phenomena that are not yet completely understood. Multiple surgical options for treatment patients with LHB tendon related pain have been described.

Literature reveals variable results comparing tenotomy and tenodesis with no technique demonstrating clear clinical superiority. Arthroscopic procedures include simple tenotomy of the tendon and multiple techniques of arthroscopic intra-articular and suprapectoral tenodesis. The tendon stump can be fixed using suture anchors or various types of tenodesis screws. Open treatment is more favored for subpectoral tenodesis.

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Subscapularis Tears

26

Ian K.Y. Lo

Epidemiology

The subscapularis is the largest of the rotator cuff tendons and provides over 50 % of the rotator cuff force [1]. Despite its conspicuous position in the anterior aspect of the glenohumeral joint, subscapularis tendon tears are likely both underreported and underappreciated. This is largely related to the difficulty in evaluating the subscapularis tendon both clinically and surgically (whether open or arthroscopic). The incidence of subscapularis tears, therefore, has varied in the literature but is estimated between 10 and 30 % of arthroscopic shoulder procedures [2–5]. In general, tears of the subscapularis are less common than tears of the supraspinatus and partial tears are more common than complete tears. While subscapularis tears do occur in isolation, they more commonly occur in combination with other rotator cuff tears.

Traditionally, the main function of the subscapularis has been described as an internal rotator and anterior stabilizer of the glenohumeral joint. However, its primary function is to assist in balancing the force couples about the shoulder in conjunction with the other muscles of the rotator cuff. Subscapularis tears lead to a disruption of the anterior moment, imbalance of the transverse plane force couple, and an unstable fulcrum of motion with abnormal biomechanics. An important principle of rotator cuff repair should include restoration of the anterior moment (i.e., subscapularis repair) in order to balance the force couples about the shoulder and restore function [1, 6–9].

Pathophysiology

Similar to tears of the supraspinatus tendon, subscapularis tears are likely caused by a number of etiologic factors, which in isolation or combination result in tendon disruption. Intrinsic factors including alterations in microscopic (e.g., decrease cellularity, fascicular thinning/disruption, granulation tissue), metabolic, and vascular anatomy can predispose to degenerative tearing. Extrinsic factors including single traumatic or repetitive traumatic events may lead to eventual fiber failure. Histologic evaluation of cadaveric subscapularis tendons suggest that intrinsic tendon degeneration may be an important etiologic factor in tears of the subscapularis [10, 11]. These studies have demonstrated that the incidence of histologic changes in the subscapularis tendon is similar to that of the supraspinatus tendon and is most commonly seen in the superior and deep portion of the tendon insertion.

Unique to the anatomy of the subscapularis tendon is its close proximity to the overlying coracoid. This has led some authors to hypothesize its role in the development of subscapularis tendon tearing similar to the relationship between the acromion and supraspinatus tears.

We define subcoracoid stenosis as narrowing of the subcoracoid space and subcoracoid impingement as direct contact of the coracoid against the subscapularis tendon or lesser tuberosity. The coracohumeral distance (a measurement of the subcoracoid space) is measured radiographically as the distance between the coracoid tip and the humerus/lesser tuberosity when viewed on axial CT or MRI images. The normal coracohumeral distance has been reported to measure 8.7–11.0 mm [12, 13]. Various studies have demonstrated narrowing of the coracohumeral distance (i.e., subcoracoid stenosis) in association with subscapularis tears. In one study, in 35 patients requiring subscapularis repair, the coracohumeral distance was measured as 5.0 ± 1.7 mm, compared to a control group ($N = 35$) that had no rotator cuff, subscapularis or subcoracoid pathology, which measured 10.0 ± 1.3 mm [14].

Subcoracoid impingement usually results from subcoracoid stenosis. Similar to tears of the supraspinatus tendon, subscapular tears have been theorized by some authors to result from abrasive and erosive wear by the overlying coracoid. A more likely scenario, however, is that the subscapularis tendon fails under tension as it arches or is "bowstrung" across a prominent coracoid (i.e., the roller-wringer effect) [15]. In this scenario,

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the prominent anterior coracoid causes tensile loads on the undersurface of the subscapularis tendon leading to fiber failure of the articular surface of the subscapularis insertion [6, 7, 15]. This is consistent with histologic changes of subscapularis tendon degeneration and that the most common subscapularis tear is a partial-thickness articular surface tear.

While these and other studies have suggested a relationship between both subcoracoid stenosis and subcoracoid impingement to subscapularis tears, the exact temporal or causal relationship between these two etiologic factors and actual tearing is still unclear [15, 16].

History and Clinical Examination

The primary symptoms of patients with subscapularis tears particularly those in combination with other tears of the rotator cuff include pain, loss of mobility, and loss of function. Patients will classically report a traumatic episode in forced external rotation or hyperextension. However, similar to patients with posterior-superior rotator cuff tears, many patients may not recall a particular traumatic event.

Physical exam may demonstrate a decrease in internal rotation strength and an increase in passive external rotation. However, a number of special tests have been described to specifically evaluate the integrity of the subscapularis tendon.

The *lift-off test* is performed by placing the back of the hand of the affected arm on the lower lumbar region [17]. The test is considered positive if the patient is unable to raise the hand posteriorly off the back by internally rotating the glenohumeral joint. This indicates a complete subscapularis tear. However, the lift-off test may be normal in patients with upper subscapularis tendon tears and is of limited value in patients who cannot bring the affected arm into position because of pain or restricted range of motion.

In contrast, the *belly-press test* is performed by positioning the palm on the abdomen [18, 19]. The patient presses on the abdomen keeping the elbow anterior to the mid-coronal plane of the body. In patients with subscapularis insufficiency, to press on the abdomen the posterior deltoid instead provides the force and the elbow drops posteriorly. A variation of this test is the *Napoleon test*, which is performed in the same fashion but also accounts for the position of the wrist [20]. In patients with subscapularis tendon tears, in addition to the elbow dropping posteriorly, the wrist palmar flexes against the abdomen. The amount of wrist flexion has been correlated to the size of subscapularis tendon tearing. Tears of less than 50 % of the subscapularis tendon can have negative Napoleon tests (i.e., wrist fully extended), tears involving more than 50 % but not the entire tendon have an intermediate result (i.e., wrist flexed 30–60°), while tears involving the entire subscapularis tendon had a positive Napoleon test (i.e., wrist flexed 90°)(20) (Fig. 26.1).

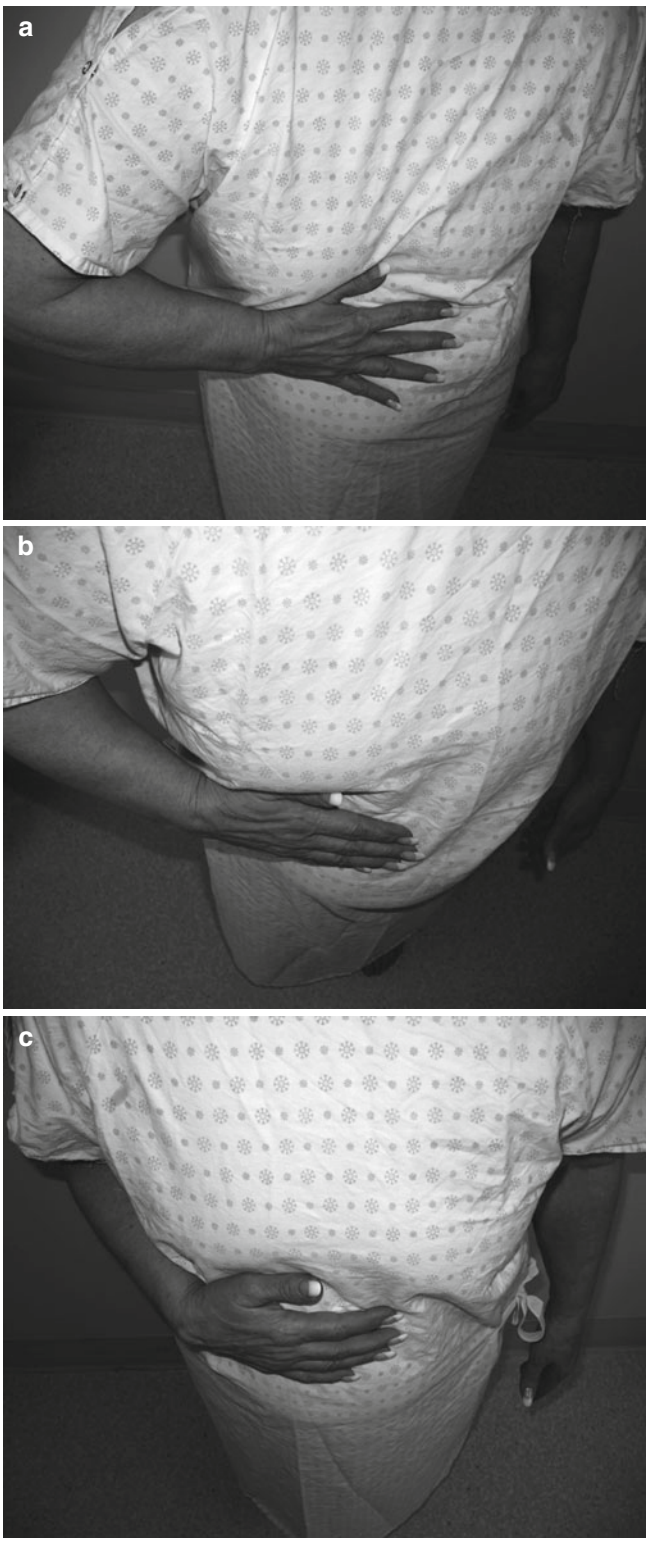

Fig. 26.1 Napoleon test: (**a**) Negative Napoleon test, the patient can press on the belly with the wrist at 0°, indicating normal subscapularis function. (**b**) Intermediate Napoleon test, as the patient presses on the belly, the wrist flexes 30–60°, indicating partial function of the subscapularis. (**c**) Positive Napoleon test, indicating a nonfunctional subscapularis, in which the patient can press on the belly only by flexing the wrist 90°, using the posterior deltoid rather than subscapularis function

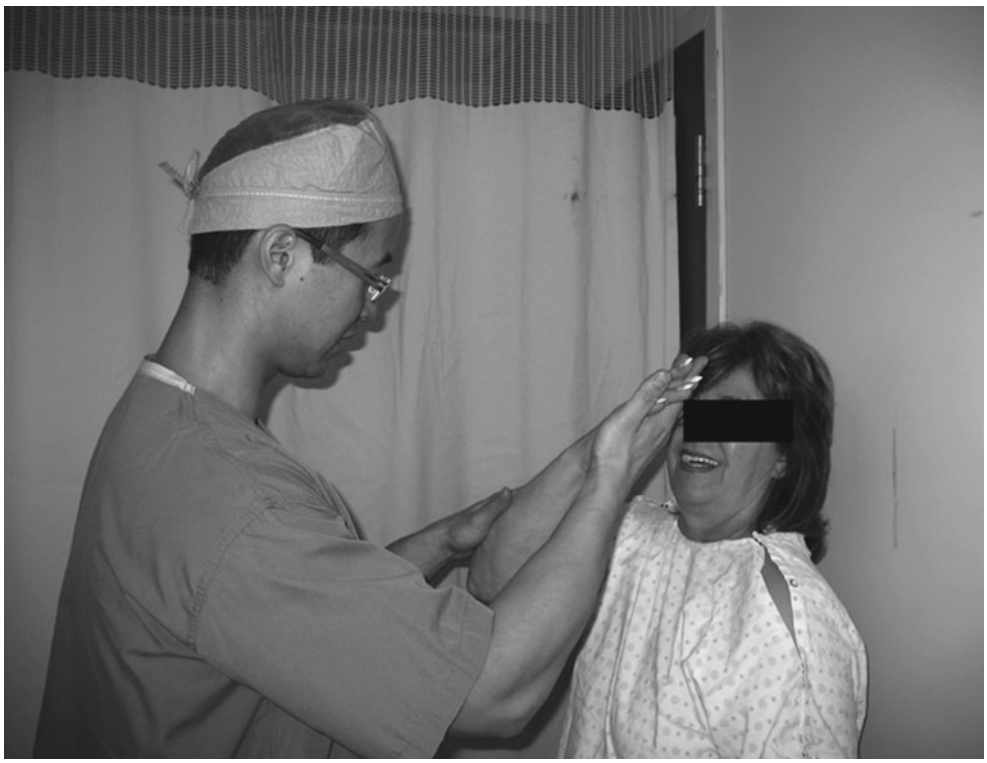

Fig. 26.2 The bear-hug test. A positive bear-hug test results when the patient cannot hold the hand against the shoulder as the examiner applies an external rotation force

The most sensitive test, particularly for partial or upper subscapularis tears, is the *bear-hug test*. In this test, the patient places the hand on the opposite shoulder with the fingers extended and the elbow into a forward position [3]. Using an external rotation force perpendicular to the plane of the forearm, the examiner pulls the patient's hand off of the shoulder. The test is considered positive if the examiner is able to lift the patient's hand off the shoulder (Fig. 26.2).

Imaging

Plain radiographs are usually nonspecific for subscapularis tendon tears but may demonstrate static anterior subluxation of the humeral head on axillary radiographs. In addition, when a subscapularis tear is associated with a massive posterior-superior tear, then proximal humeral head migration with narrowing of the acromial humeral interval may also be present.

Although ultrasound can provide a relatively inexpensive, noninvasive, dynamic assessment of the rotator cuff, it is highly observer dependent. Ultrasound has been reported to be 100 % sensitive and 85 % specific in the detection of all rotator cuff lesions [21]. In this study, ultrasound correctly identified six of seven tears of the subscapularis tendon. In a larger study of 17 isolated subscapularis tendon tears, ultrasonography correctly demonstrated 86 % of full-thickness tears of the subscapularis. However, ultrasonography is not as accurate in demonstrating partial-thickness tears or small upper subscapularis tendon tears [22, 23].

Currently, magnetic resonance imaging (MRI) is considered the modality of choice for evaluating rotator cuff pathology including tears of the subscapularis. Magnetic resonance imaging arthrography has been reported to be 91 % sensitive and 86 % specific for detecting tears of the subscapularis tendon [24]. However, others have reported less promising results, and subscapularis tendon tears particularly full thickness and upper subscapularis or partial-thickness tears are commonly missed on MRI. In one study, surgically confirmed subscapularis tendon tears were only identified in 31 % (5/16) of patients preoperatively on MRI [25].

In addition to direct pathology related to the subscapularis tendon, injury to adjacent structures may be suggestive of subscapularis tendon tearing. Tears of the subscapularis tendon commonly disrupt the stabilizing reflection pulley of the long head of the biceps tendon. This may be detected as extravasation of contrast material anterior to the superior border of the subscapularis tendon on axial images and usually results in medial subluxation or dislocation of the long head of the biceps tendon.

Recently, Adams et al. [26] reported on the use of MRI for the diagnosis of subscapularis tears in 202 patients with arthroscopic confirmation. In this study, the MRIs were solely evaluated by fellowship-trained orthopedic surgeons. Four MRI criteria were utilized in this study including the presence of a subscapularis tear on axial or sagittal oblique images, the presence of a dislocated long head of the biceps tendon, and atrophy of the subscapularis muscle belly (Fig. 26.3). Patients with two or more

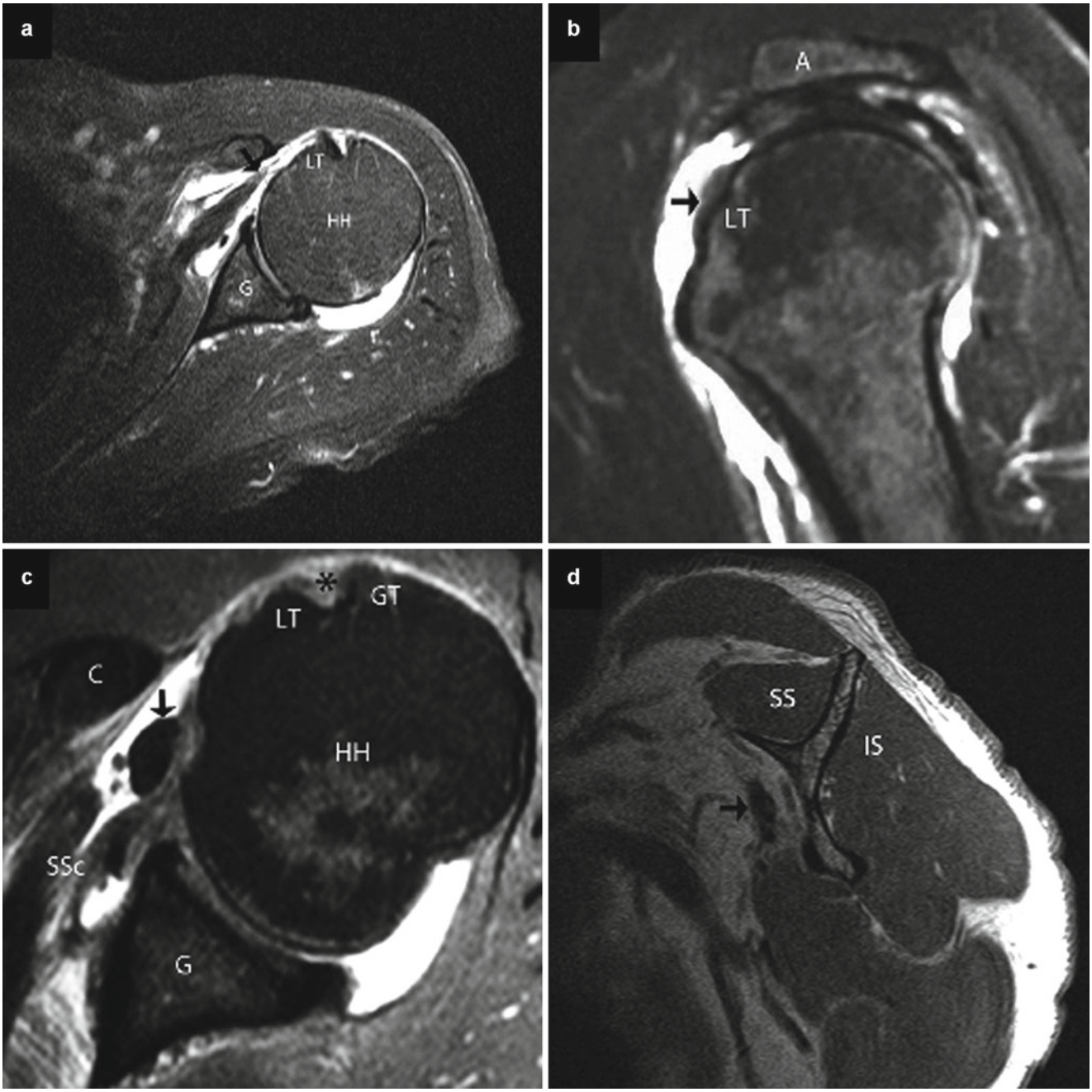

Fig. 26.3 MRI indicative of subscapularis tendon tearing. (**a**) Axial MRI view of a left shoulder with a torn and retracted subscapularis tendon (*arrow*) from the lesser tuberosity (*LT*). (**b**) Sagittal oblique MRI view of a left shoulder in a patient with a massive, retracted tear of the subscapularis tendon (*arrow*). (**c**) Axial MRI view of a left shoulder with a medial dislocation of the long head of the biceps tendon (*arrow*) into a split tear of the subscapularis tendon (*SSc*). (**d**) Sagittal oblique MRI view of a left shouder medial to the glenoid in a patient with a massive, retracted tear of the subscapularis tendon. One should note the significant atrophy of the subscapularis muscle belly (*arrow*). (*C*, coracoid; *G*, glenoid; *GT*, greater tuberosity; *HH*, humeral head; *IS*, infraspinatus muscle belly; *SS*, supraspinatus muscle belly). Adapted from Adams et al. [26]

positive criteria were considered diagnostic for a subscapularis tear. Eighty-two patients had subscapularis tears confirmed by arthroscopy and were correctly identified in 73 % of patients for a sensitivity of 73 % and specificity of 94 %.

In addition to tendon tearing, the chronicity of the tear and the quality of its muscle may be assessed on sagittal oblique images medial to the glenoid. Severe atrophy and fatty degeneration on preoperative MRI or CT generally correlate with poor intraoperative tendon quality, limited tendon excursion,

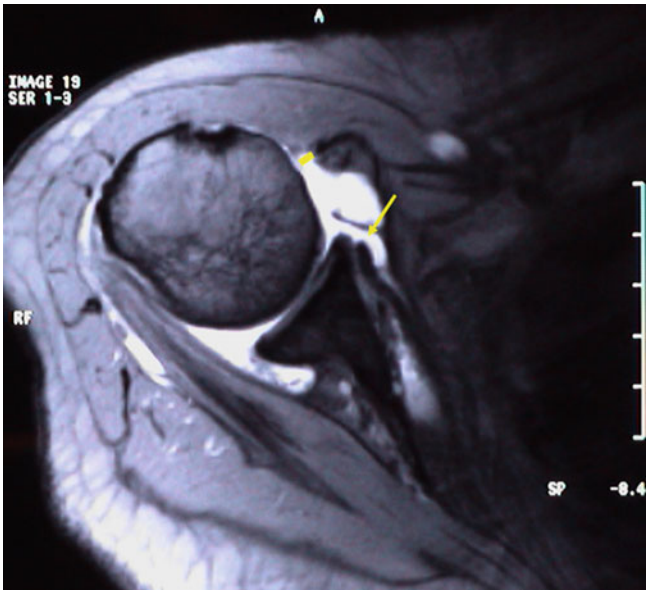

Fig. 26.4 Axial T2-weighted MRI demonstrating a complete subscapularis tendon tear (*arrow*) with a narrowed coracohumeral distance (*yellow line*)

and may be a negative prognostic sign on the ability to repair the subscapularis tendon tear [27–29]. However, we will still consider surgical repair even in patients with >50 % fatty infiltration. In many patients the subscapularis tendon is commonly reparable even in the presence of severe atrophy and fatty degeneration and may improve function through a tenodesis effect providing a more stable fulcrum of motion [30].

In addition to subscapularis pathology, radiographic evidence of subcoracoid impingement should be considered. Care should be taken when interpreting diagnostic studies since both proximal humeral migration and anterior subluxation of the glenohumeral joint can exacerbate narrowing of the subcoracoid space particularly when imaging studies are obtained supine (e.g., MRI).

The normal coracohumeral distance (i.e., the shortest distance between the coracoid tip and the humerus/lesser tuberosity), as measured on axial CT or MRI images, measures approximately 8.7–11.0 mm. We consider a coracohumeral distance of <6 mm as evidence of narrowing [13, 15, 31, 32] and should raise the suspicion of subcoracoid impingement (Fig. 26.4).

Treatment: Indications and Contraindications

The indications and contraindications for arthroscopic subscapularis repair are essentially the same as for arthroscopic rotator cuff repair. However, we will still consider surgical repair even in patients with >50 % fatty infiltration. In many patients, the subscapularis tendon is commonly reparable even in the presence of severe atrophy and fatty degeneration and may improve function through a tenodesis effect providing a more stable fulcrum of motion [30].

Decision-Making Algorithm

When performing an arthroscopic rotator cuff repair, we believe that the vast majority of subscapularis tears should be repaired, even upper subscapularis or partial tears. We believe that restoring the anterior moment of the subscapularis is important to provide a stable fulcrum of motion. The upper subscapularis is the thickest and most robust portion of the subscapularis insertion, and anatomically the anterior attachment of the rotator cable attaches to the superior aspect lesser tuberosity. Therefore, repair of the upper subscapularis not only restores the subscapularis moment arm but reconstructs the rotator cable [33].

Since the majority of subscapularis tears will involve disruption of the medial biceps sling, concomitant biceps instability will require treatment. In our experience, biceps tendon reduction and reconstruction of the medial sling is rarely successful, and therefore, in cases of concomitant biceps instability, definitive biceps treatment is required. Except in elderly low-demand patients, a biceps tenodesis is performed in the suprapectoral region [34].

The decision and choice of fixation method following releases is based on a number of factors including the normal footprint anatomy of the subscapularis tendon insertion and the mobility of the subscapularis tendon. The normal footprint of subscapularis has demonstrated an average superior to inferior height of 25.8 ± 3.2 mm, and a width measures 18.1 ± 1.6 mm [35]. Furthermore, the footprint is essentially parallel to the long axis of the humerus and is broad proximally and tapers distally [35, 36]. Therefore, in patients with significant tendon excursion with minimal tension following releases, a double-row subscapularis repair is performed to maximize footprint anatomy and fixation strength. In particular, reconstruction of the broad footprint anatomy is more critical in the superior subscapularis where the footprint is the widest and the tendon the strongest [37]. However, in patients with insufficient tendon excursion, a single row repair may be performed and the bone may be medialized 5–10 mm to maximize bone tendon contact [38].

Clinical Case Example

The illustrated case is of a 58-year-old male janitor who approximately 6 months ago slipped and fell at work leading to chronic pain and dysfunction. Clinical examination demonstrated a full passive range of motion but loss of active overhead elevation. Subscapularis testing demonstrated an

increase in passive external rotation, a positive belly-press sign, and a positive bear-hug sign. The patient was unable to perform a lift-off test due to pain.

Arthroscopic Treatment: Surgical Technique

Patient Positioning

We perform all shoulder arthroscopies with the patient in the lateral decubitus position. The torso is bolstered by a beanbag with all bony prominence padded and protected. The patient is prepped and draped, isolating the forequarter and allowing access circumferentially about the shoulder. The arm is supported by a Spider2 Limb Positioner (Smith & Nephew, Andover, MA) and the patient's arm is positioned initially in slight flexion and abduction.

Portals

We utilize a standard posterior portal for glenohumeral arthroscopy, created approximately 3–4 cm medial and 3–4 cm inferior to the posterior lateral corner of the acromion. To perform arthroscopic subscapularis repair, two other portals are generally required, an anterior portal and an anterosuperolateral portal. The key portal is the anterosuperolateral portal created approximately 2–3 cm lateral to the anterolateral corner of the acromion and oriented anterior to the supraspinatus tendon, tangential to footprint of the subscapularis tendon on the lesser tuberosity (Fig. 26.5). The anterosuperolateral portal is the primary working portal utilized during subscapularis repair for subcoracoid decompression, tendon releases, suture passage, and knot tying. The anterior portal is created approximately 3–4 cm anterior to the anterolateral corner of the acromion and is primarily used for anchor insertion and suture management. For this reason we commonly use percutaneous incisions and a cannula is rarely utilized.

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

A standard diagnostic arthroscopy is performed focusing on the anterior structures of the shoulder. Our preference is to perform arthroscopic subscapularis repair primarily visualizing through the posterior glenohumeral portal (the "glenohumeral" approach) using both 30° and 70° arthroscopes. In particular the 70° arthroscope is extremely valuable when looking "around the corner" of the humeral head (Fig. 26.6). To further improve visualization of the subscapularis insertion, the arm may be positioned in flexion, abduction, and internal rotation, relaxing the fibers of the subscapularis tendon and allowing visualization of the tendon insertion into bone. An assistant may apply a posterior translational force to the upper arm, again improving visualization and also increasing the anterior working space. Occasionally, the anterosuperolateral portal may be utilized for visualization (the "bursal" approach) while working through an anterior or accessory anterior portal. This portal can be useful when extensive dissection of the anterior subcoracoid space is

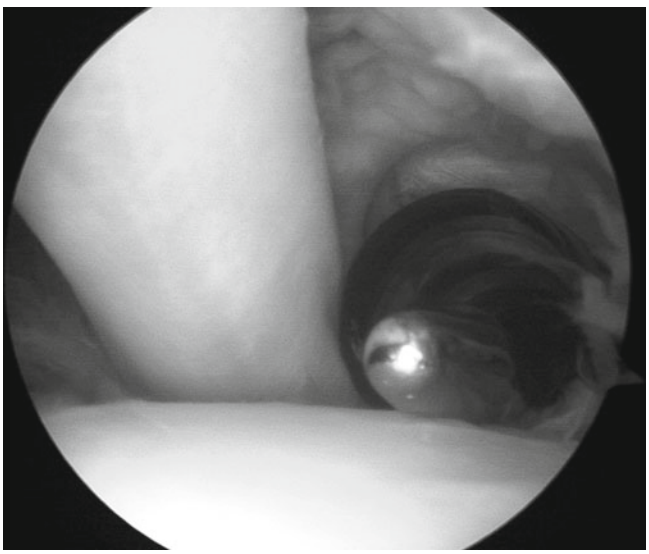

Fig. 26.5 Arthroscopic view of a right shoulder from the posterior portal, demonstrating the anterosuperolateral portal. The anterosuperolateral is created anterior to the supraspinatus tendon, tangential to the lesser tuberosity

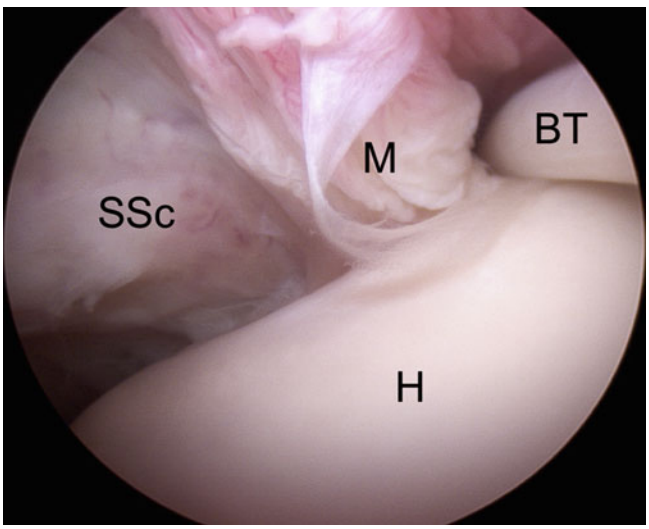

Fig. 26.6 Arthroscopic view of a right shoulder from the posterior glenohumeral portal using a 70° arthroscope. The 70° arthroscope provides top-down view of the subscapularis (*SSc*), biceps tendon (*BT*), and medial sling (*M*) (*H* humeral head)

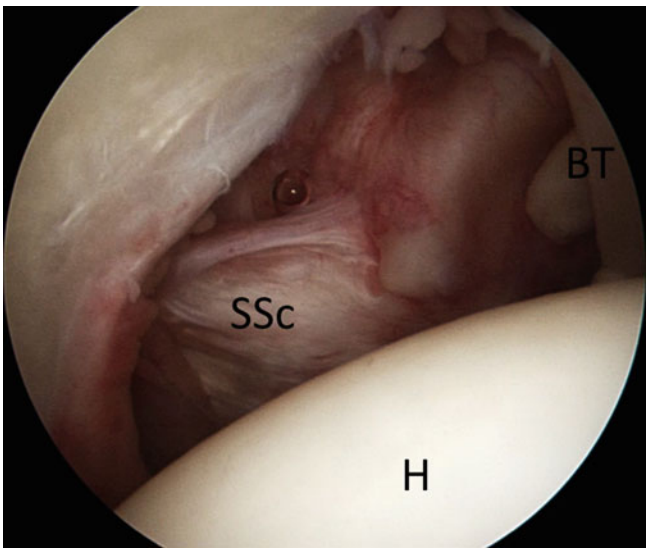

Fig. 26.7 Arthroscopic view of a right shoulder through a posterior glenohumeral portal demonstrating a subscapularis (*SSc*) tear (*BT* biceps tendon, *H*, humeral head)

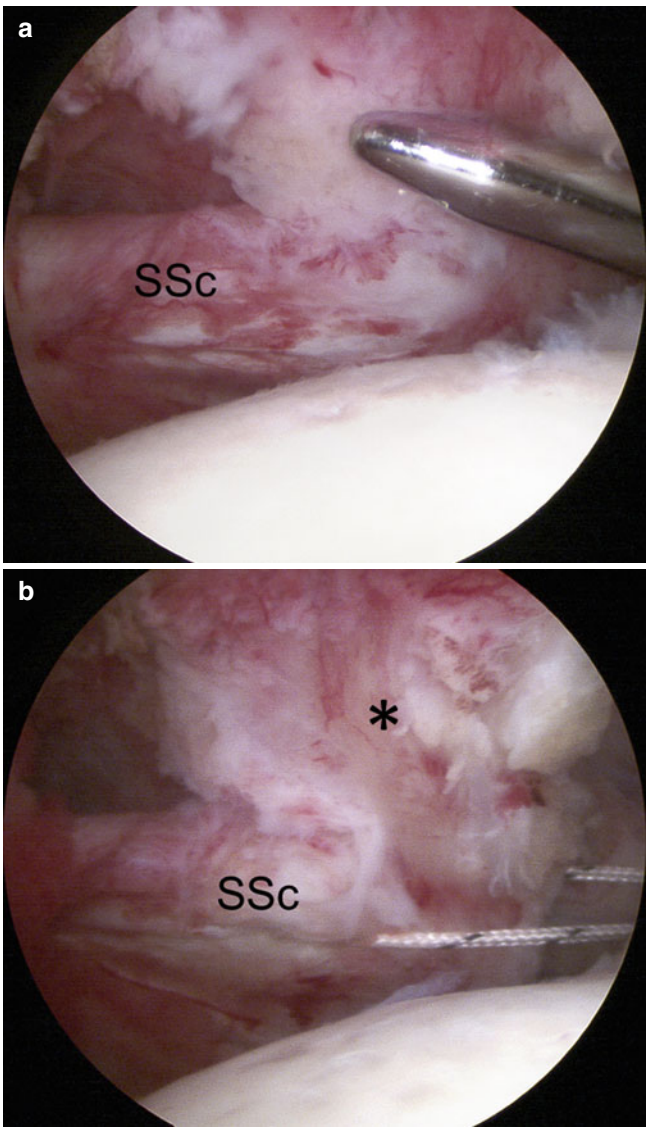

Fig. 26.8 Arthroscopic view of a right shoulder through a posterior glenohumeral portal demonstrating the comma sign. (**a**) The comma sign (as marked by the switching stick) is an arc of tissue arising from the superolateral corner of the torn subscapularis. (**b**) A traction stitch has been placed in the corner of the comma sign (*). Applied traction draws the superior and lateral border of the subscapularis laterally (*SSc* subscapularis)

required and when performing double-row subscapularis repairs during final tendon fixation to bone.

The key to recognizing subscapularis pathology is to appreciate the normal anatomy of the subscapularis insertion. In particular due to their close proximity, subscapularis tears involving the superior border commonly result in tearing of the medial biceps sling and subsequent instability of the long head of the biceps. In fact, identifying a subluxed biceps tendon medial and posterior to the subscapularis should raise the suspicion of an occult subscapularis tear.

Once a subscapularis tendon tear has been identified (Fig. 26.7), initial treatment of the biceps tendon is obligatory due to associated instability. A traction stitch is placed along the intra-articular portion of the long head of the biceps tendon and the tendon is released from the superior labrum. Our preference is to perform a suprapectoral biceps tenodesis in the majority of cases and a biceps tenotomy in elderly low-demand individuals. If a biceps tenodesis is to be performed, approximately 4–5 cm of tendon is removed (to maintain its anatomic length-tension relationship) and a suture is whip-stitched through the residual tendon [34]. In addition to improve visualization, tagging and releasing the biceps tendon can improve visualization of the subscapularis tendon tear.

In cases of chronic, full-thickness, complete subscapularis tendon tears, the borders of the subscapularis tendon may be difficult to identify due to scarring to the inner deltoid fascia and conjoint tendon. In this scenario, the key to recognizing the subscapularis tendon is to identify the "comma sign" [39]. The comma sign is a comma-shaped arc of tissue which is attached to superior lateral border of the subscapularis and is formed from the concomitantly avulsed medial biceps sling (i.e., medial coracohumeral ligament, superior glenohumeral ligament) (Fig. 26.8a). By placing traction on the "comma sign," the subscapularis is drawn laterally, exposing the superior and lateral borders of the subscapularis (Fig. 26.8b).

In most chronic, full-thickness subscapularis tears, advanced releases are required to improve tension-free excursion of the tendon to the lesser tuberosity. Acute or partial tears may not require releases. Our preference is to perform a stepwise three-sided release of the subscapularis tendon (i.e., superior, anterior, posterior) avoiding inferior dissection which risks the axillary nerve. Beginning with the superior release,

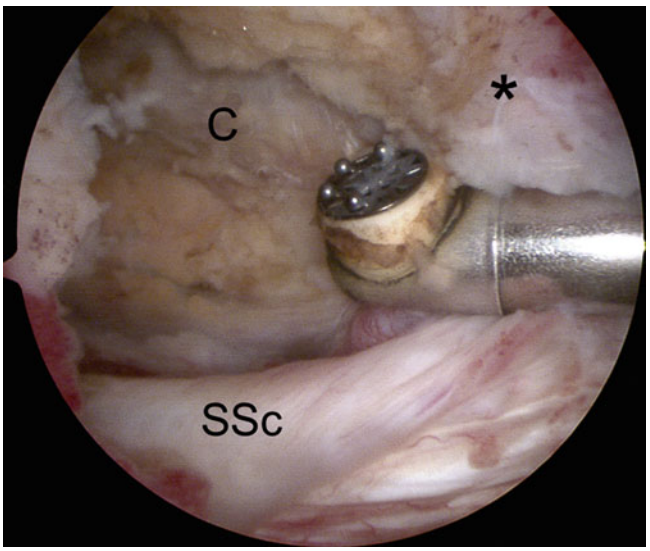

Fig. 26.9 Arthroscopic view of a right shoulder through a posterior glenohumeral portal demonstrating the superior release. A "window" has been created through the rotator interval exposing the coracoid (*C*) while maintaining the integrity of the comma sign (*) (*SSc* subscapularis)

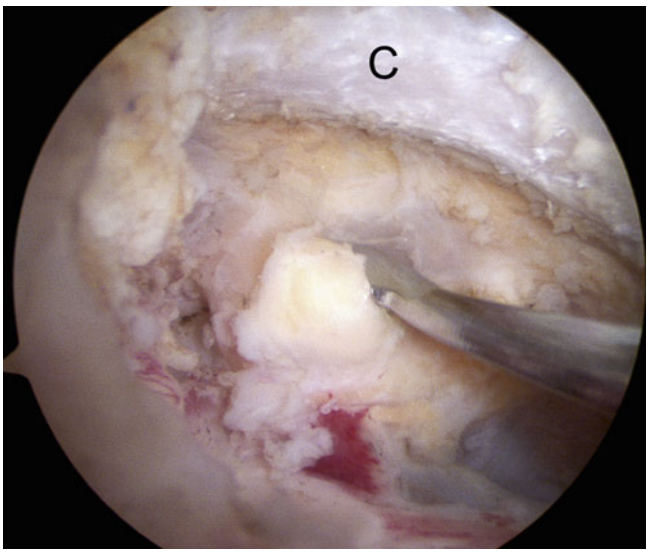

Fig. 26.10 Arthroscopic view of a right shoulder through a posterior glenohumeral portal using a 70° arthroscope demonstrating release of the coracohumeral ligament from the neck of the coracoid (*C*)

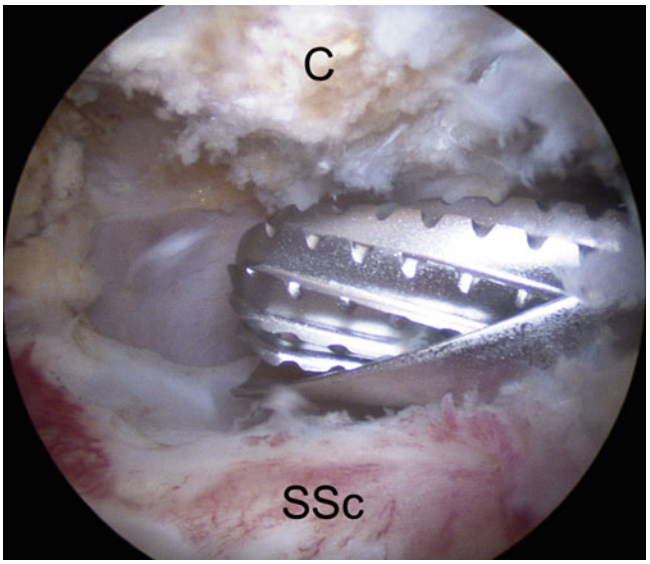

Fig. 26.11 Arthroscopic view of a right shoulder through a posterior glenohumeral portal using a 70° arthroscope demonstrating subcoracoid decompression. An oval burr is inserted through the anterosuperolateral portal anterior to the subscapularis (*SSc*), and the posterolateral tip of the coracoid (*C*) is removed

the comma sign is again identified and a traction stitch is placed in the corner of superolateral border of the subscapularis tendon. By pulling on the traction stitch, the subscapularis tendon is drawn laterally exposing its superior border and the rotator interval. A shaver or electrocautery (Super TurboVac 90, ArthroCare, Inc., Austin, TX) is introduced through the anterosuperolateral portal, and the rotator interval is excised, being careful to preserve the lateral border of the rotator interval (i.e., the comma sign) and the superior border of the subscapularis. As the release continues, essentially a "window" is created through the rotator interval exposing the coracoid tip, the coracoid attachment of the coracoacromial ligament, and the conjoint tendon (Fig. 26.9) [40].

With the rotator interval excised, the arthroscope is then positioned through the rotator interval revealing entire length of the coracoid and the subcoracoid space. The anterior release is then performed and instruments are introduced through the anterosuperolateral portal anterior to the subscapularis improving access to the subcoracoid structures. Furthermore, a 70° arthroscope is routinely used which provides an "aerial" view of the subcoracoid space. The posterolateral surface of the coracoid is exposed including its neck and base excising any superior or anterior adhesions including the coracohumeral ligament (Fig. 26.10).

The subcoracoid space is then assessed. In cases of chronic subscapularis tearing, particularly in conjunction with massive posterior-superior rotator cuff tearing, narrowing of the subcoracoid space may occur both primarily or secondarily. The subcoracoid space assessed measuring the distance between the tip of the coracoid and the lesser tuberosity or subscapularis tendon (i.e., coracohumeral distance). In cases of severe narrowing, the arm may be placed in forward flexion, adduction, and internal rotation to document the presence of subcoracoid impingement. In patients with a subcoracoid space less than 6 mm, a subcoracoid decompression is performed [41]. An oval burr is introduced through the anterosuperolateral portal, and the posterior lateral tip of the coracoid is excised in line with the subscapularis tendon and lesser tuberosity (Fig. 26.11). A space is

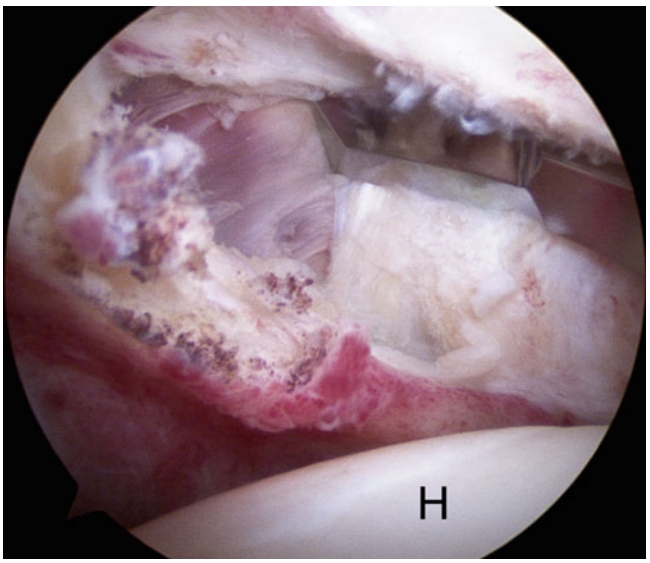

Fig. 26.12 Arthroscopic view of a right shoulder through the posterior glenohumeral portal demonstrating a completed posterior release of the subscapularis (*H* humeral head)

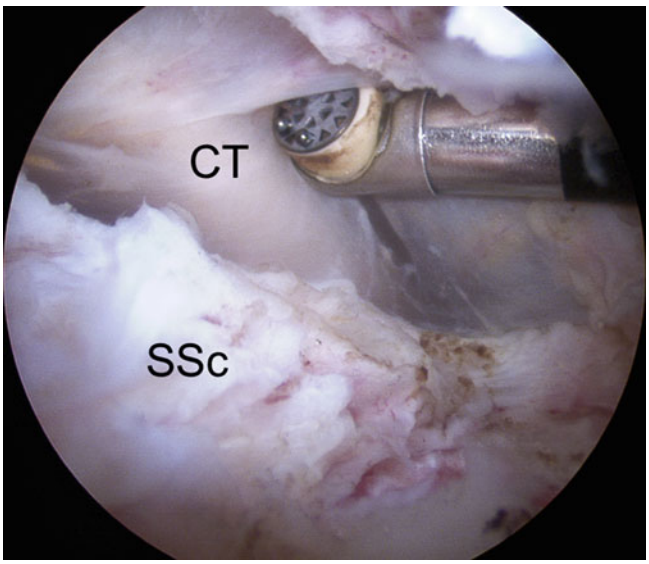

Fig. 26.13 Arthroscopic view of a right shoulder through the posterior glenohumeral portal demonstrating lateral release of the subscapularis tendon (*SSc*) off the conjoint tendon (*CT*)

created of approximately 10–11 mm. Care is taken during decompression to maintain the integrity of the coracoacromial ligament and conjoint tendon.

The posterior release is performed next. The arthroscope is retracted into the glenohumeral joint and instruments are introduced through the anterosuperolateral portal posterior to the subscapularis tendon. Traction is applied to the subscapularis tendon, and using a combination of electrocautery and an arthroscopic elevator, the middle glenohumeral ligament and capsule are released from the posterior subscapularis (Fig. 26.12).

In some chronic subscapularis tears, the lateral border of the subscapularis tendon may become obscured due to extensive scarring of the lateral subscapularis border to the inner deltoid fascia and conjoint tendon. In addition, in patients with partial subscapularis tears where the subscapularis is split into superficial and deep layers, the superficial layer commonly extends out laterally over the lesser tuberosity and bicipital groove as a continuation to the transverse ligament. In each case the lateral border of the subscapularis must be defined. Traction is applied to the comma sign and the superior and lateral borders of the subscapularis tendon are revealed. Instruments (e.g., electrocautery, shaver) are introduced through the anterosuperolateral portal and the lateral border is carefully dissected from the lateral extensions (e.g., inner deltoid fascia, transverse ligament). In severe cases, dissection of the subscapularis off the conjoint tendon is required improving excursion (Fig. 26.13) and reconstituting the subcoracoid space.

Once the releases have been completed, the excursion of the subscapularis tendon is reassessed. In the vast majority of cases, sufficient mobility is obtained to allow tension-free repair to bone. However, to improve tendon contact or reduce tension of the repair, the bone bed may be medialized. The bone bed is prepared in the usual fashion using a high-speed burr, being careful to expose bleeding bone without compromising implant fixation.

Prior to subscapularis tendon fixation to bone, a suprapectoral biceps tenodesis is performed. A separate low anterior portal is created perpendicular to the bicipital groove, and tendon is secured into the prepared bone tunnel using interference screw fixation (Bio-Tenodesis Screw, Arthrex, Inc., Naples, FL) [42]. Biceps tenodesis is performed prior to subscapularis tendon fixation, since early subscapularis repair obscures visualization of the inferior bicipital groove.

Standard or advanced techniques are then utilized for subscapularis tendon fixation to bone. If sufficient excursion is obtained, a linked double-row construct is utilized and is our preference. In this particular patient, a double-row subscapularis repair is achievable. Two anchors (4.5 mm CrossFT BC, ConMed-Linvatec, Largo FL) are placed along the medial aspect of the footprint starting inferiorly (Fig. 26.14) and progressing superiorly using separate anterior percutaneous portals. The sutures are then passed through the medial aspect of the tendon using an antegrade suture passing device (FirstPass, ArthroCare, Inc., Austin, TX) (Fig. 26.15a). When passing sutures through the subscapularis tendon, a self-retrieving antegrade suture passing device is desirable since the anterior aspect of the subscapularis is not visualized (i.e., blind suture passage). All sutures are passed through the medial aspect of the tendon in a mattress fashion (Fig. 26.15b). Suture are then tied reducing the medial aspect

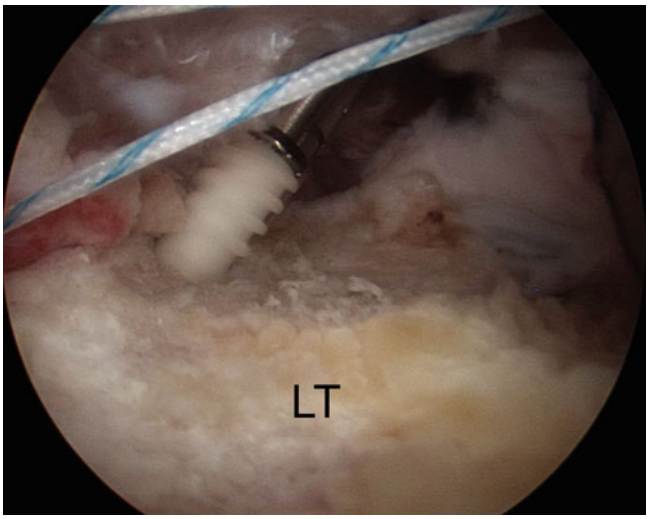

Fig. 26.14 Arthroscopic view of a right shoulder through the posterior glenohumeral portal using a 70° arthroscope. A medial row of anchors is placed along the medial aspect of the footprint adjacent to the articular cartilage starting inferiorly (*LT* lesser tuberosity)

of the subscapularis tendon insertion to bone. The suture ends are then brought out to the lateral aspect of the footprint and secured using a lateral row of knotless anchors (5.5 mm SpeedScrew, ArthroCare, Inc., Austin, TX) (Fig. 26.16). This reduces the lateral aspect of the tendon to bone, improving fixation and reconstructing the anatomic footprint of the subscapularis insertion (Fig. 26.17). Usually two anchors are used for lateral row fixation.

Once the subscapularis tendon is repaired, repair of concomitant rotator cuff tears is performed. Repair of the subscapularis tendon draws anterior margin of supraspinatus tendon laterally facilitating repair [43]. It is important also to maintain the integrity of the "comma sign" with the subscapularis tendon as this tissue can be also utilized during repair of the posterior-superior rotator cuff (Fig. 26.18).

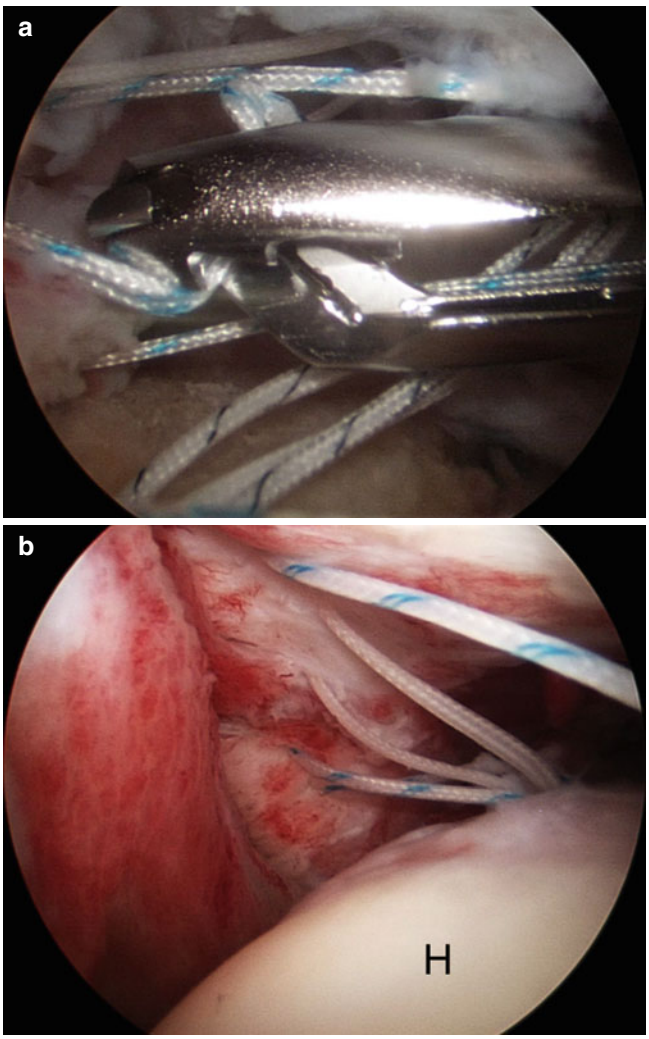

Fig. 26.15 Arthroscopic view of a right shoulder through the posterior glenohumeral portal using a 70° arthroscope. (**a**) A self-retrieving antegrade suture passing device (FirstPass, ArthroCare, Inc., Austin, TX) is utilized to pass mattress suture along the medial aspect of the tendon. (**b**) Completed medial mattress suture passage (*H* humeral head)

Postoperative Care

Postoperatively, patients are immobilized in a sling for 6 weeks. Immediate hand, wrist, and elbow motion is permitted. In patients with complete subscapularis tendon tears, passive external rotation with the arm at the side is restricted to 0° (i.e., arm pointed straight ahead). In patients with partial or upper subscapularis tears, external rotation may be allowed to approximately 30°. If a biceps tenodesis has been performed, only passive range of motion of the elbow is allowed until 6 weeks postoperatively. Forward elevation is begun at 6 weeks postoperatively. Active assisted to active range of motion is progressed from 6 to 12 weeks with strengthening beginning approximately 12 weeks postoperatively.

Literature Review

The clinical results of arthroscopic repair of subscapularis tears have recently been clarified, as a number of studies have been published which specifically address the subscapularis. Burkhart et al. first reported on arthroscopic repair of full-thickness subscapularis tears in 25 isolated or combined subscapularis tears [20]. By modified UCLA criteria, excellent and good results were obtained in 92 % of patients, with one fair and one poor result. Eight isolated subscapularis tears improved in their UCLA score from on average from 10.0 to 32.8. The authors found that multi-tendinous tears that involve at least half of the subscapularis in association with tears of the supraspinatus and infraspinatus cause

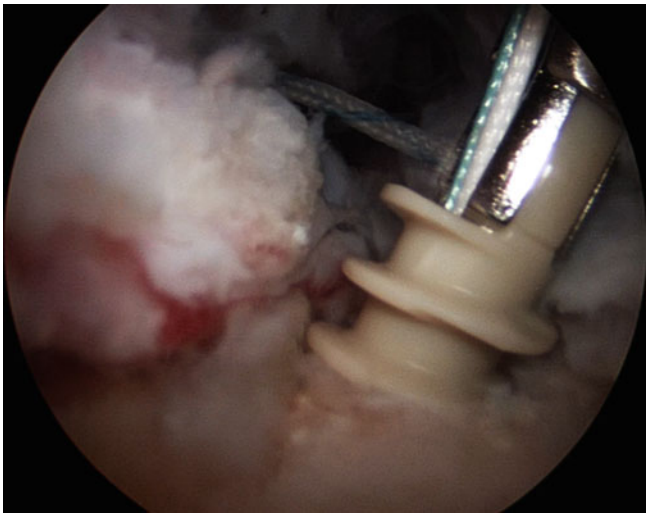

Fig. 26.16 Arthroscopic view of a right shoulder through the posterior glenohumeral joint using a 70° arthroscope. Residual suture limbs from the medial anchors are secured through a lateral row of knotless anchors (5.5 mm SpeedScrew, ArthroCare Inc., Austin, TX) reconstructing the anatomic footprint of the subscapularis insertion

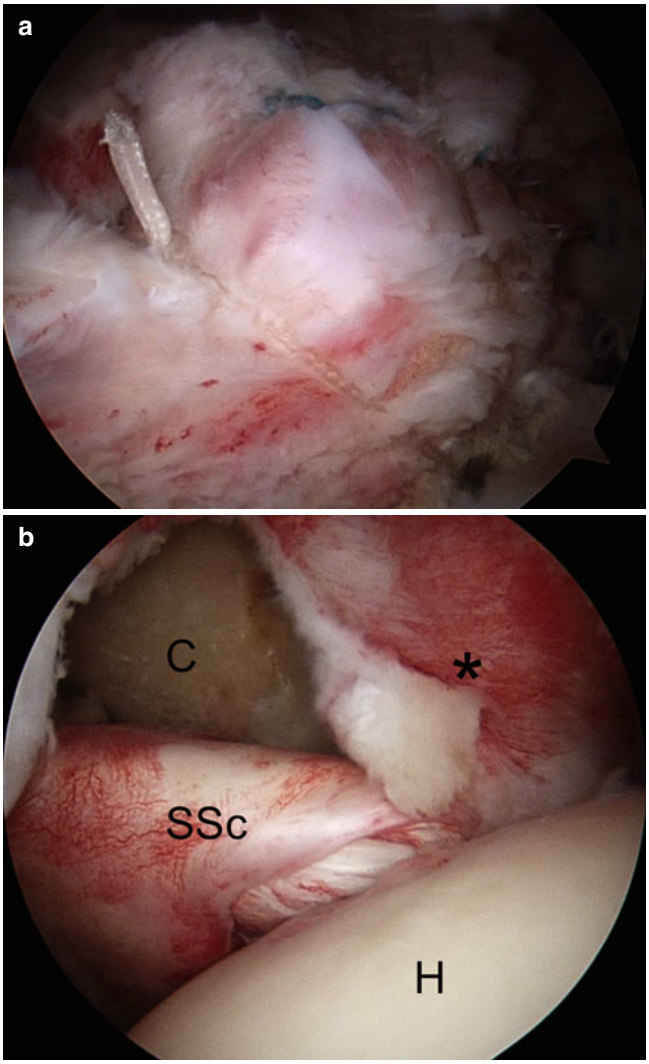

Fig. 26.17 Arthroscopic view of a right shoulder demonstrating the completed double-row repair. (**a**) View through the anterosuperolateral portal. (**b**) View through a posterior portal (*C* coracoid, *H* humerus, *SSc* subscapularis, * comma sign)

profound functional deficits, particularly when they displayed proximal humeral head migration on radiographs. Out of the 17 combined tears, 10 patients had proximal migration of the humeral head, and all had complete loss of overhead function preoperatively. Eight of these ten patients had durable reversal of proximal humeral migration and restoration of overhead function following surgery. The two patients with recurrence of proximal migration had the poor and fair results, with little or no change in range of motion. Subsequent to this, a number of authors have reported excellent short and midterm clinical results following arthroscopic repair of the subscapularis [4, 5, 44–47].

Recently, Denard et al. reported on the long-term clinical results of arthroscopic repair of the subscapularis [48]. Although the follow-up rate was low (34 % of patients), the authors did report on 79 patients with a mean follow-up of almost 9 years. These authors demonstrated durable improvement in UCLA scores and ASES scores with 92.4 % of patients satisfied, and subjectively, patients rated their operated shoulder as 89.8 % of normal.

The anatomic results of arthroscopic subscapularis repair have also been reported. Lafosse et al. [5] reported on 17 isolated subscapularis tears repaired arthroscopically. He noted a significant improvement in the average relative Constant score from 58 to 96 % and UCLA score from 16 to 32 points. Postoperatively, all patients had CT arthrography for assessment of subscapularis repair integrity. They noted complete healing in 15 patients and partial re-rupture in 2 patients. The authors reported no significant increase in progression of fatty infiltration.

Nové-Josserand et al. [49] have reported on the clinical and anatomic results of 22 patients following arthroscopic repair of the isolated subscapularis tears. At a mean of 36 months postoperatively, the Constant score increased from 66 to 85 points. Using MRI or CT arthrography, they reported partial retearing in 14 % of patients and healed tendons in 86 % of patients. However, in 36 % of patients, while the subscapularis appeared intact, the tendon quality appeared thin. Furthermore, 55 % of patients demonstrated some progression of fatty infiltration of the subscapularis muscle belly. With the numbers available, there was no significant correlation between the quality of the tendon repair, fatty infiltration, and clinical outcome or subscapularis testing.

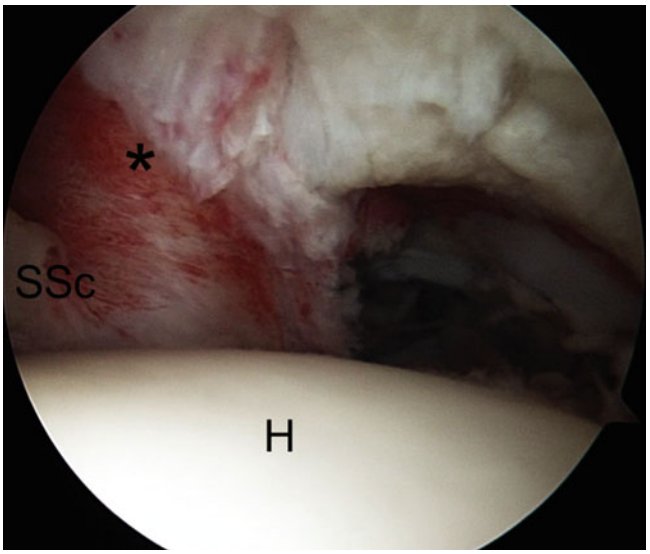

Fig. 26.18 Arthroscopic view of a right shoulder demonstrating the intact comma sign leading to the residual rotator cuff defect (*H* humerus, *SSc* subscapularis, * comma sign)

Summary

In conclusion, subscapularis tears are increasingly becoming identified as common isolated and combined lesions of the rotator cuff and can lead to significant dysfunction and disability of the shoulder. Careful evaluation both clinically and with advanced imaging can identify the majority of subscapularis tears. Using a stepwise approach to tear margin identification and releases, arthroscopic subscapularis repair is routinely achievable with excellent clinical results.

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Treatment Options for Irreparable Rotator Cuff Tears

27

Carmine Latte, Matteo Salvatore, Paolo Avanzi, and Andrea Grasso

The treatment of massive rotator cuff tears still remains a challenge for the orthopedic surgeon, their operative repair is technically difficult and associated with a higher recurrence rate than that of smaller tears [1–5].

There is no consensus in the literature regarding the definition of a massive rotator cuff tear. Patte [6] categorized rotator cuff tears based on its extent, topography in the sagittal and frontal plane, quality of the muscle, and state of the long head of the biceps tendon. Massive tears involve more than one tendon and at least 4 cm long in the sagittal plane, without (group III) or with (IV) humeral head osteoarthritis.

More recently, some authors have classified massive rotator cuff tears as a tear with a width of >5 cm [7] or a tear in which there is complete detachment of two or more tendons [8, 9]; others considered the tear patterns and the mobility of their margins [10].

Because of variations in patient's size and techniques in measurement, Gerber et al. [11] emphasize that there is no agreement on the definition of a massive tear and proposed a classification system based upon the amount of tendon detached from the tuberosities.

Elhassan et al. [12] supported the Gerber's functional definition concluding that "a massive tear is not necessarily irreparable and an irreparable tear is not always massive."

In another paper, Warner and Parsons [13] defined as irreparable the rotator cuff tear that, although soft tissue release performed to mobilize the remaining tendon fibers, it is impossible to repair it directly on its insertion on the humeral head. These tears usually involve both tendon and muscle fibers, which appear to be, respectively, severely degenerated and deeply. Usually, these changes are the result of a chronic pathology, while tears larger than 4–5 cm, but resulting from an acute rupture as the one caused by a traumatic injury, can often be completely repair to their anatomical footprint.

The prevalence of massive tears reported in the literature has ranged from 10 to 40 % of all rotator cuff tears [14–16].

Signs of irreparability include static superior migration of the humeral head, a narrowed (<5 mm) or absent acromiohumeral interval, and fatty infiltration affecting more than 50 % of the rotator cuff musculature [15–18]. An acromiohumeral distance less than 5 mm and a magnetic resonance imaging that shows fatty infiltration of the supraspinatus fosse muscle indicate an irreparable tears with an involvement of at least two or more tendons (Fig. 27.1) [12]. Therefore, an irreparable rotator cuff tears can be described as the one that should not be repaired due to the lack of potential healing (Fig. 27.2).

There are several treatment options for irreparable cuff tears, but deciding the correct one for each patient can be difficult. They include nonoperative management, debridement of the cuff with subacromial decompression, direct partial repair, muscle-tendon unit transfers, shoulder hemiarthroplasty, reverse shoulder arthroplasty, tissue interposition, and allograft augmentation.

Low-demanding patients, for example, can be treated nonoperatively or with debridement and acromioplasty, open or arthroscopically [19, 20]. Tenotomy of the long head of the biceps tendon, if present, determines a significant pain reduction of the shoulder treated [21].

Patients with severe pain, weakness, and no glenohumeral osteoarthritis, if affected by a massive rotator cuff tear, are good candidates for a tendon transfer. If the tear involves the posterosuperior part of the cuff, the latissimus dorsi transfer, as described by Gerber [22], could be an option; if the subscapular tendon is completely torn, the pectoralis major has been used as substitution [23].

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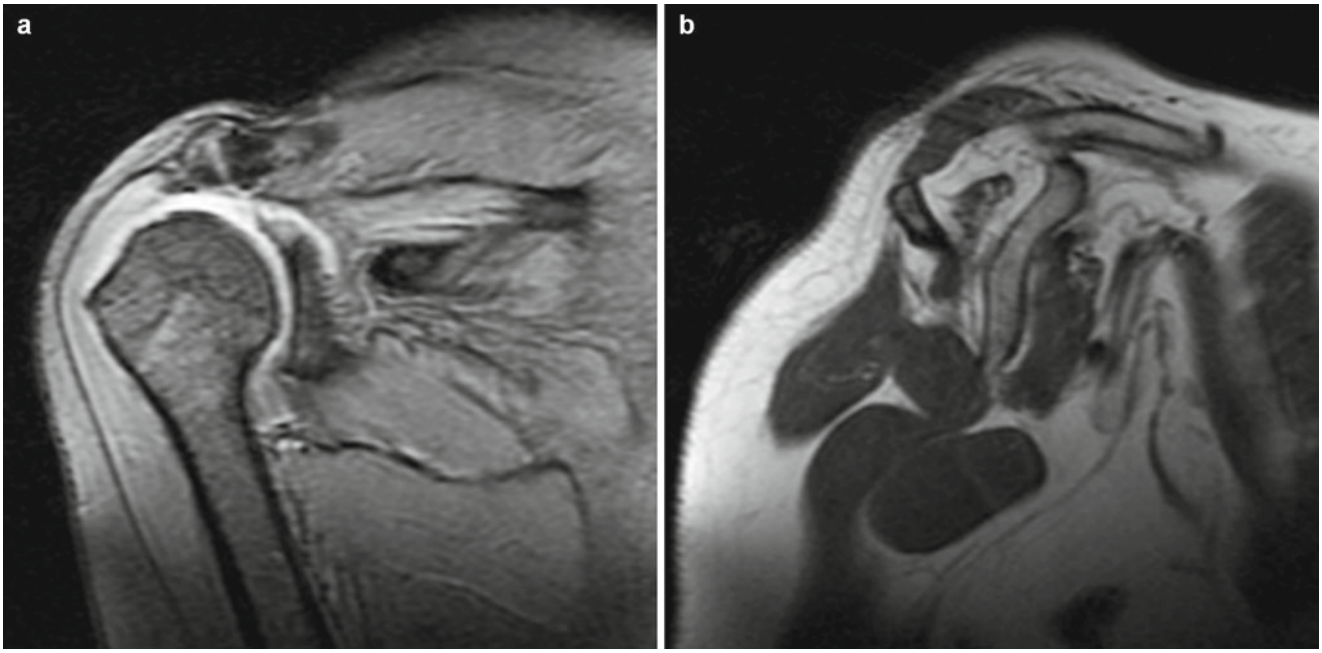

Fig. 27.1 MRI showing massive rotator cuff tear in T2-weighted sequence in coronal plane (**a**) and fatty infiltration of the supraspinatus in sagittal T1-weighted sequence (**b**)

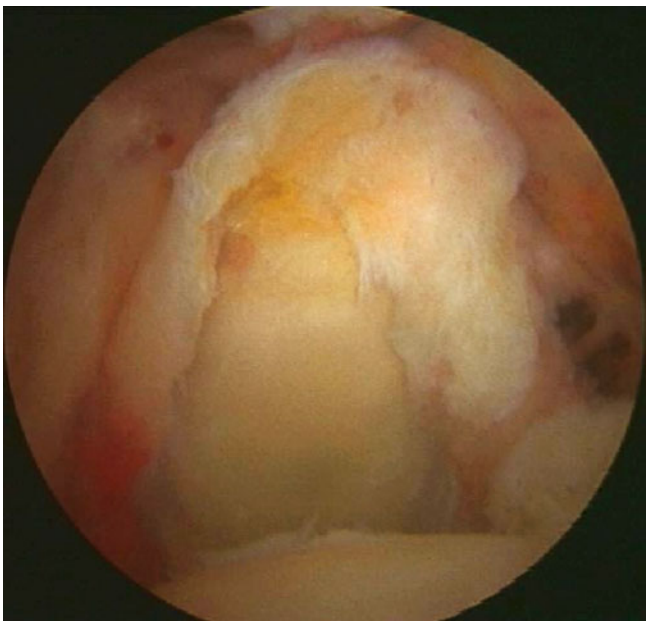

Fig. 27.2 Arthroscopic view of a massive irreparable rotator cuff tear

Rotator cuff augmentation with allografts or extracellular matrix scaffolds have shown nonunivocal results, and level I studies are absent [24–26].

An understanding of the patient's chief complaint and their functional ability to elevate their arm above horizontal plane should guide the treatment [27], even because of the lack of level I evidence papers, that are in some cases unethical if performed correctly to analyze the best treatment options in shoulders with massive rotator cuff tears.

Nonoperative Treatment

Because of high recurrence rate of irreparable tears, some authors have suggested that it may be preferable to treat such tears nonoperatively or with simple debridement [19, 28, 29].

Several studies have shown that patients with a massive tear have good motion and the ability to perform activities of daily living [10, 30, 31].

Hansen et al. [32] using a cadaver shoulder model demonstrated that stable glenohumeral abduction without excessive superior humeral head translation could be maintained in the setting of a massive tear but requires the generation of higher forces in the deltoid and the remaining intact portion of the rotator cuff. Also the subscapularis muscle forces were increased 30–85 % depending on the tear size. Some individuals with a massive tear, even those with a tear that involves the inferior infraspinatus and teres minor tendons, can maintain active shoulder abduction and good function with low-demand daily activities. One hypothesis is that contraction of the deltoid muscle superiorly translates the humeral head underneath the coracoacromial arch and allows pivoting around this abnormally superior center of rotation.

Bokor et al. [33] noted also a correlation between the duration of symptoms and the long-term success of nonoperative management; patients with symptoms for longer than

6 months shown inferior outcomes with respect to patients who start the conservative treatment earlier.

Physical Therapy and Rehabilitation

Recently, Levy et al. [34] described an effective system for rehabilitation and "reeducation" of the anterior deltoid muscle to compensate a deficient rotator cuff. In his work, a 12-week anterior deltoid rehabilitation program led to an improvement of the mean Constant score from 26 to 60 points and the mean forward flexion improved from 40° to 160° after completion of the program. This approach is supported by recent biomechanical studies that have demonstrated an important role for the anterior deltoid muscle in preventing superior humeral head migration and compressing the glenohumeral joint in the presence of a large cuff tear [35].

Other studies have also shown good results with physical therapy for patients with irreparable rotator cuff tears.

Ainsworth [36] evaluated a multimodal physical therapy program that emphasized patient education, posture correction, reeducation of muscle recruitment, strengthening, stretching, improved proprioception, and adaptation.

The long-term outcomes of patients with irreparable tears that treated conservatively were evaluated by Zingg et al. [37]. They studied retrospectively 19 patients at a mean follow-up of 48 months. Over this time, glenohumeral osteoarthritis progressed, as did the size of the tendon tears and the measured fatty infiltration. Despite this, the authors found that functional outcome scores remained acceptable, with a mean absolute Constant score to be 69, a relative Constant score to be 83, and a mean subjective shoulder value of 68. Pain averaged 11.5 on a 0–15point visual analog scale, where a score of 15 meant no pain. The authors concluded that nonoperatively treated massive rotator cuff tears in moderately symptomatic patients could maintain satisfactory shoulder function despite the progression of degenerative pathology.

The risk of rotator cuff arthropathy and irreversible fatty infiltration of muscle may limit future treatment options and must be considered when counseling patients.

Intra-articular Injections

Patients may benefit from fluid aspiration and corticosteroid injection. Repeated intra-articular injections of corticosteroids are discouraged, as they are largely ineffective [38, 39].

Thus, aspiration and corticosteroid administration may be a useful adjunct to physical therapy for patients who are unable or unwilling to undergo surgery [40].

Many surgeons are hesitant to give repeated cortisone injections into the shoulder with cuff tear arthropathy because of the risk of infection in a joint with a persistently large and often hemorrhagic effusion; however, it remains an excellent tool at the surgeon's discretion [41].

Hyaluronan injections are safer and may be repeated as necessary and, although of benefit for early and late osteoarthritis of the shoulder, have not yet been investigated for cuff arthropathy [42, 43].

Hyaluronans act by blocking pain receptors and stimulating endogenous hyaluronan production and have a direct anti-inflammatory effect by inhibiting leukocyte action [44].

Operative Treatment

There is some controversy over the role of arthroscopy in the management of irreparable rotator cuff tears. Arthroscopic debridement, partial repair with margin convergence, biceps tenotomy or tenodesis, and, more recently, suprascapular nerve release have all been described as potential treatments.

Debridement and Subacromial Decompression

Arthroscopic debridement has been reported to have satisfactory short-term outcomes in patients with a massive rotator cuff tear [10, 29]. This procedure is primarily indicated for elderly, low-demanding patients with pain but good preservation of active motion and an intact coronal and transverse force couple about the glenohumeral joint. Although shoulder strength does not improve after this intervention, function is usually enhanced because of relief from pain caused by mechanical impingement.

Preoperative relief from a subacromial injection is a favorable prognostic finding for improvement after this operation. If operative debridement and subacromial decompression is performed, it is critical that the coracoacromial ligament be preserved in the setting of a massive rotator cuff tear because of its function as important static stabilizer against anterosuperior escape of the humeral head [45].

In literature, there is no consensus about the results of this procedures that can be performed both open or arthroscopically [15, 16, 20, 29, 46]. Some authors reported poor long-term results after open subacromial decompression and resection of the coracoacromial ligament [46].

Gartsman [29] reported modest results of open debridement and subacromial decompression, with decreased pain relief and improved function but decreased strength as compared with the preoperative condition [35].

Zvijac et al. [47] reported deterioration of function and strength over time, and Kempf et al. [48] reported only modest improvement in the overall Constant score after the treatment of massive tears with arthroscopic debridement and long-term postoperative rehabilitation.

Rockwood et al. [20] reported decreased pain and improvement of function and strength in 44 (88 %) of 50 patients after tear debridement and decompression.

In order to maintain the integrity of the coracoacromial arch in the setting of a massive rotator cuff tear, alternative decompressive procedures have been described. Some authors described open debridement and tuberoplasty for massive irreparable rotator cuff tears to reshape the greater tuberosity for smooth articulation with the acromion [49, 50].

Fenlin et al. [49], in their study on a cohort of 20 patients followed for 27 months, reported satisfactory results in 95 % of the cases, and the improvement in the mean UCLA score was from 9.3 to 27.7 points [49].

Scheibel et al. [50] described a so-called reversed arthroscopic subacromial decompression, a procedure that includes arthroscopic debridement of the subacromial space and glenohumeral joint and an arthroscopic tuberoplasty with or without biceps tenotomy. In their series, 23 patients were followed for a mean of 40 months, and the age-adjusted Constant score increased from 66 to 91 points. The average acromiohumeral distance only decreased from 5.1 to 4.5 mm, and the integrity of the coracoacromial arch was preserved.

Partial Rotator Cuff Repair

Burkhart [51] described the side-to-side closure of massive, U-shaped rotator cuff tears introducing the term "margin convergence."

Based on the assumption that most massive rotator cuff tears are not retracted but are L-shaped tears with a vertical split from medial to lateral, which assume a U shape because of the elasticity of the muscle-tendon unit, Burkhart [51] stated that mobilization of these tears leads to failure of repair because of tension overload at the apex of the tear. He supposed that a side-to-side closure gives a mechanical advantage because of a biomechanical principle called margin convergence. In the technique of margin convergence, the free margin of the tear converges toward the greater tuberosity as side-to-side repair progresses. As the margin converges, the strain at the free edge of the cuff is reduced significantly, leaving an almost tension-free converged cuff margin overlying the humeral bone bed for repair. Side-to-side closure of two-thirds of a U-shaped tear reduces the strain at the cuff margin to one-sixth of the one that existed at the pre-converged cuff margin. This strategy gives a lower probability of failure of fixation to bone, either by anchors or transosseous tunnels. The principles of margin convergence and force couples must be followed when attempting repair of a massive rotator cuff tear.

Partial repair, in which there is a defect remaining in the superior portion of the cuff after margin convergence, can still be effective, if at least half of the infraspinatus can be repaired to bone. Burkhart recommended partial repair whenever complete closure of the defect is not possible and advised against local transfers of rotator cuff tendons [49].

In truly immobile tears, an interval slide as described by Tauro [52] sometimes allows an additional 1–2 cm of lateral excursion of the supraspinatus tendon and therefore permits a greater degree of partial repair. The results of this technique are variable.

Mazzocca et al. [53] in a cadaveric study supported the hypothesis that margin convergence decreases the size of the tear gap and reduces strain on the remaining tendons with minimal effect on glenohumeral translation. Moreover, the investigator found less tension and stress in the rotator cuff during knot tying after placement of the margin convergence sutures.

Biceps Tenotomy

The function of the long head of the biceps (LHB) tendon, particularly in the setting of a massive rotator cuff tear, is controversial. Some studies have shown the biceps to function as both a dynamic and a static humeral head depressor and shoulder stabilizer [54, 55].

Yamaguchi et al. [56] in their electromyographic studies have shown the LHB to be quiescent in patients with a massive cuff tear during active abduction, suggesting that its stabilizing role is likely more passive than active.

There is evidence suggesting that the LHB tendon may be a source of pain and contribute to the discomfort associated with symptomatic massive cuff tears.

Walch et al. [21] reported good outcomes after biceps tenotomy in patients with an irreparable massive rotator cuff tear. They performed 307 biceps tenotomies in patients with full-thickness rotator cuff tears that were considered irreparable. At a mean of 57 months postoperatively, the average Constant score increased from 48 points preoperatively to 68 points postoperatively. Concomitant acromioplasty was associated with better subjective and objective results in patients with a preserved acromiohumeral distance of 6 mm or more. They reported that 87 % of the patients were satisfied with the results of the surgery. Fatty infiltration of the rotator cuff, however, had a negative influence on both functional and radiographic outcomes.

Boileau et al. [57] in a retrospective study of 68 consecutive patients in whom a total of 72 irreparable rotator cuff tears had been treated arthroscopically with biceps tenotomy or tenodesis reported a 78 % of satisfactory results. The mean Constant score improved from 46.3 ± 11.9 points preoperatively to 66.5 ± 16.3 points postoperatively (P <0.001). The results did not differ between the tenotomy and tenodesis groups (mean Constant score, 61.2 ± 18 points and 72.8 ± 12 points, respectively). They concluded that both

arthroscopic biceps tenotomy and tenodesis can effectively treat severe pain or dysfunction caused by an irreparable rotator cuff tear associated with a biceps lesion. Shoulder function is significantly inferior if the teres minor is atrophic or absent. Pseudoparalysis of the shoulder and severe rotator cuff arthropathy are contraindications to this procedure.

Klinger et al. [58] compared the results of arthroscopic debridement in massive, irreparable rotator cuff tears with and without tenotomy of the LHB in a cohort of 41 patients. The average Constant score for the group without LHB tenotomy improved from a mean of 39 points (range, 19–54 points) preoperatively to a mean of 67 points (range, 41–87 points) and for the group with additional LHB tenotomy from a mean of 41 points (range, 16–54 points) preoperatively to a mean of 69 points (range, 49–87 points) at the time of follow-up. No statistical significance ($P > .05$) was found between the two groups. However, patients with additional LHB tenotomy had a longer duration of postoperative pain relief, but final pain score difference was not statistically significant. They concluded that additional LHB tenotomy did not significantly influence the postoperative results at the latest follow-up after arthroscopic debridement of massive, irreparable rotator cuff tears.

In a recent study, Kim et al. [59] noted that for patients with concomitant type II SLAP lesions and large to massive rotator cuff tears, the outcomes of simultaneous arthroscopic SLAP and rotator cuff repair were inferior to those of arthroscopic biceps tenotomy and cuff repair in terms of functional shoulder scores and range of motion. Biceps tenotomy and rotator cuff repair may be a more reliable method to address concomitant type II SLAP lesions and large to massive rotator cuff tears in patients, although a randomized controlled trial is needed to confirm the results. The arthroscopic tenotomy, while helping the functional outcomes and patients' pain, does not influence the course of glenohumeral osteoarthritis.

Suprascapular Nerve Block

The role that neuropathy of the suprascapular nerve (SSN) plays in the pain and weakness associated with massive rotator cuff tears remains unclear. It has been postulated that massive posterosuperior cuff tears can place traction on the SSN as the rotator cuff muscles retract. Several authors, using electrodiagnostic studies, have documented SSN neuropathy in patients with massive cuff tears [60–62]. Some of these studies also documented partial or complete recovery following partial or complete repair. Mallon et al. [60] prospectively studied eight patients presenting with massive rotator cuff tears that showed retraction and fatty infiltration of the supraspinatus muscle on MRI. All patients have suprascapular neuropathy as diagnosed by electromyography (EMG). Four of the eight patients underwent partial repair. Two of them underwent a new EMG 6 months postoperatively. In both cases, the EMG showed that the SSN has significant reinnervation potential.

Vad et al. [61] found in 7 of 25 patients with full-thickness rotator cuff tears abnormal EMG studies. The axillary nerve was affected in 4 of the 7 patients, while the SSN was affected in 2 of 7.

Costouros et al. [62] identified 26 patients with massive rotator cuff tears. Using EMG and nerve conduction velocity (NCV) studies, 14 of those patients with massive cuff tears were found to have a concomitant peripheral neuropathy. Seven of the 14 were found to have an isolated suprascapular neuropathy. Four were found to have an axillary neuropathy, 2 had an upper trunk brachial plexus injury, and 1 had a cervical radiculopathy. Six of the 7 patients with the isolated SSN were treated with either complete or partial repair. Of these patients, 6 months later, repeat EMG/NCV study showed partial or full recovery of the SSN.

Scaffolds

In recent years, there has been increasing interest in the potential role of human-, bovine-, and porcine-derived implants, of either small intestinal or dermal origin as augmentation, or even bridging devices when direct repair is impossible [63].

The ideal implant in the context of rotator cuff repair would be a biologically inert material that does not degrade or lose its tensile strength inside the body or one that allows sufficient ingrowth of host cells to allow healing of the cuff tendon before it loses its strength. Various grafts can be used in rotator cuff tears to supplement or augment a repair [63–66].

Soler et al. [24] in their original study reported some early complications using porcine dermal collagen implants as bridging constructs in irreparable cuff tears. In their series, 6 months after surgery, all patients reported a worsening of clinical picture due to resorption of the graft caused by an inflammatory response.

In another similar report about porcine small intestine submucosal implants in rotator cuff repairs, Malcarney et al. [67] stated an early failure of the repair caused by nonspecific inflammatory reactions. Other authors reported better results using human dermal allograft (see Chap. 40).

Gupta et al. [25] in their prospective observational study of 24 patients who underwent interposition repair of massive rotator cuff tears using human dermal allograft reported a significant improvement in pain, range of motion, and strength. Subjective outcome measures, including mean ASES and SF-12 scores, also demonstrated significant improvement at an average 3-year follow-up.

Bond et al. [68] recently described the results of a technique of staged arthroscopic reconstruction with the use of human dermal allograft that demonstrated improved overall functional outcomes at a mean 26-month follow-up.

Venouziou et al. [26] in their retrospective study evaluated the effectiveness of the acellular human dermal allograft as a bridging device for reconstruction of massive irreparable rotator cuff tears. They noted a significant improvement of pain and range of motion (ROM) and a high patient satisfaction. The mean American Shoulder and Elbow Surgeons (ASES) score improved from 23.8 points preoperatively to 72.3 postoperatively ($P = .001$). Also a significant correlation was found between the size of the tendon gap, which was bridged with the allograft and pain, ROM and ASES score. Patients with less than 2 cm tendon gap had a better outcome than those with greater tendon defects. Even if the literature about human dermal tissue matrix allograft seems to be encouraging a cost-effective analysis of extracellular tissue matrix versus arthroscopic repair, while taking into consideration long-term outcomes and quality-adjusted life years, would also help guide treatment intervention in the new healthcare environment.

Tendon Transfer

Nowadays, tendon transfers have gained acceptance as a treatment option for irreparable cuff tears. Local tendon transposition, distant tendon transfer, and deltoid flap transposition have all been proposed.

The local tendon transposition of a portion of subscapularis and teres major to cover superior cuff defects has been used with limited success; among the distant tendon transfers, the latissimus dorsi transfer for massive posterosuperior tears and pectoralis major transfers for the anterosuperior tears are described with more reproducible and long-term success [69].

Latissimus Dorsi Transfer

Latissimus dorsi (LD) tendon transfer represents often the best choice for treating massive and irreparable posterosuperior rotator cuff tears in young and active patients that are not suitable candidates for reverse shoulder arthroplasty. In these patients, the loss of active external rotation and inability to stabilize the arm in the space are very invalidating [70–72], and often, this condition is associated with chronic disabling pain that does not respond to conservative treatment. The LD tendon transfer may improve active range of motion and strength and reduce pain in these selected patients [73, 74].

Gerber et al. [75] introduced the idea of a LD tendon transfer for the treatment of irreparable posterosuperior cuff tears on the basis of the good results obtained by l'Episcopo [76] in children with brachial plexus birth palsies with this technique (Fig. 27.3).

The transfer of LD to the superolateral humeral head converts the transferred tendon to a humeral head depressor by virtue of its almost vertical orientation and into an external rotator by virtue of its insertion relative to the humeral head.

Gerber et al. [72] in a study evaluating the long-term result of LD tendon transfer noted that patients with deficient subscapularis preoperatively did not achieve the improvements in function and pain seen in those with an intact subscapularis. He concluded that LD transfer durably and substantially improves chronically painful, dysfunctional shoulders with irreparable rotator cuff tears, especially if the subscapularis is intact, but that if subscapularis function is deficient, the procedure is of questionable benefit and probably should not be used [72].

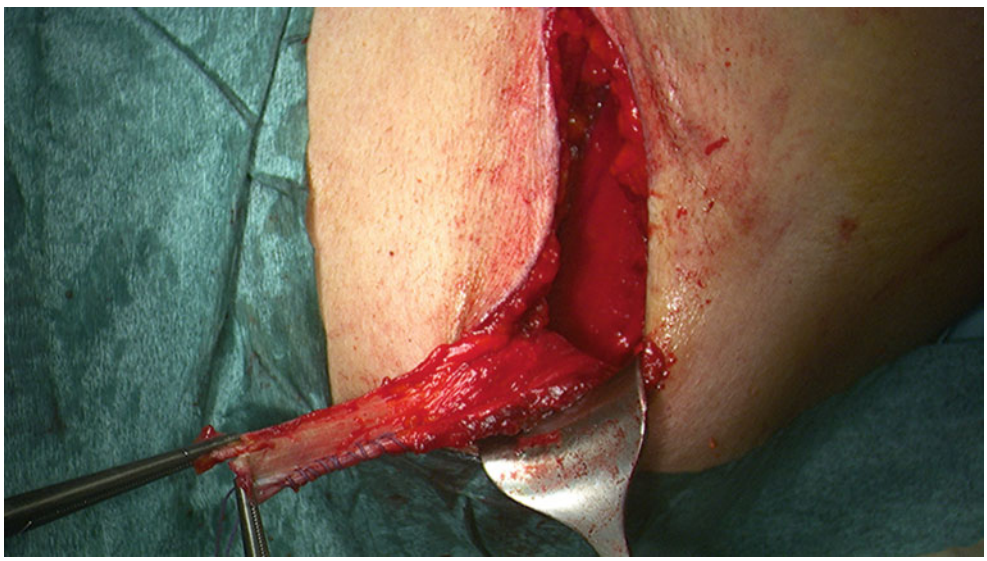

Fig. 27.3 Latissimus dorsi tendon transfer: tendon harvesting

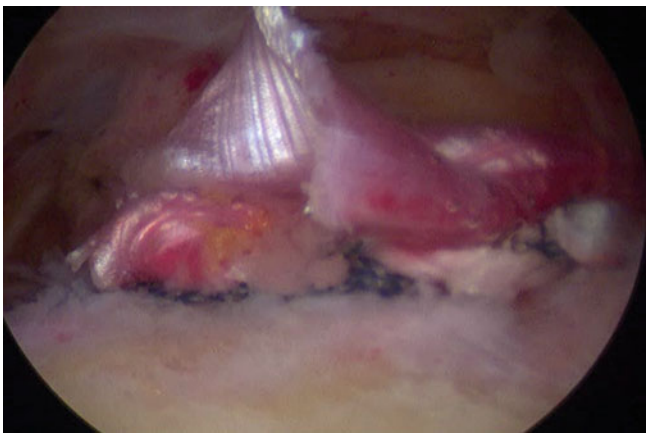

Fig. 27.4 Arthroscopically assisted latissimus dorsi tendon transfer: arthroscopic final view

LD transfer as a salvage procedure after failed rotator cuff repair has been shown to be effective. Miniaci and MacLeod [73] in their study could not detect any significant differences, either preoperatively or postoperatively, between patients with intact deltoid and those with deltoid compromise by a previous open rotator cuff repair with regard to pain, function, range of motion, UCLA shoulder score, or the overall satisfaction with the shoulder.

Warner and Parsons [13] further demonstrated that an intact deltoid is mandatory for restoration of shoulder function. Birmingham and Neviaser [77] also found that deltoid function was linked to the degree of improvement after LD transfer for failed rotator cuff repair.

Therefore, subscapularis and deltoid integrity, the absence of glenohumeral osteoarthritis, less than 70 years of age, and a contained humeral head with an intact coracoacromial arch are fundamental factors linked to the success of surgery [13]. Although LD tendon transfer can successfully restore shoulder function, it has not been shown to halt progression of cuff tear arthropathy [78].

An arthroscopically assisted technique for the LD tendon transfer has also been described by Gervasi et al. [79] (see Chap. 42) (Fig. 27.4).

Deltoid Flap Reconstruction

For the treatment of posterosuperior tears, some surgeons proposed and used a deltoid flap with variable results. Lu et al. [2] reported satisfactory medium-term results in terms of pain relief and improvement in shoulder function with this technique; but after a mean follow-up of 13.9 years, 50 % of the deltoid flaps had ruptured, and 70 % of shoulders had stage 2 or 3 osteoarthritis. No predictive factor for deltoid flap rupture was identified. Glanzmann et al. [80] reported minor functional gains but acceptable pain relief and patient satisfaction after deltoid flap. However, in both cases, the investigators did not recommend further use of this procedure.

Pectoralis Major Transfer

Pectoralis major (PM) transfer is a treatment option for the massive anterosuperior rotator cuff tears. Some authors stated that repair of chronic subscapularis ruptures can be challenging and has not led to favorable results [81, 82].

Resch et al. [83] reported good results in older patients (mean age, 65 years) with irreparable subscapularis tears treated using the superior half to two-thirds of PM to replace the subscapularis tendon. The PM tendon was routed behind the conjoined tendon (coracobrachialis and short head of the biceps) to lesser tuberosity to adapt the orientation of the pectoralis to that of the subscapularis. Of the 12 patient treated, at a mean follow-up of 28 months, there were 5 excellent outcomes, 4 good, 3 fair, and no poor outcomes.

Elhassan et al. [23] performed a study in which they evaluated patients with a subscapular lesion treated with the split transfer of the sternal head of the PM passed under the clavicular head; the authors assumed that this technique allows the clavicular head to act as a fulcrum for the transferred sternal head when it contracts. In this way, the axis of pull of the sternal head of the PM is more in line with the vector of the subscapularis. They noted that in patients with irreparable subscapularis tear after shoulder arthroplasty, there was a high risk of failure of transferred PM, particularly if there was preoperative anterior subluxation of the humeral head. In patients with isolated subscapularis insufficiency after a failed stabilization procedure, improvement in pain and function can be expected in those who have a concentric glenohumeral joint preoperatively. This is probably because a feature of all PM transfers is that the vector of pull of the transferred PM is still anterior to the chest wall, in contrast to the vector of the subscapularis, which is posterior to the chest wall. The investigators concluded that not all patients with irreparable subscapular tear will benefit by this procedure [23].

Biodegradable Spacer

A novel surgical technique to address irreparable rotator cuff tears uses a biodegradable subacromial spacer, the InSpace balloon (OrthoSpace, Kfar Saba, Israel), implanted between the acromion and the humeral head that permits smooth, frictionless gliding, restoring shoulder biomechanics.

The rotator cuff normally provides stability by compression of the humeral head into the glenoid, whereas rotator cuff disruption compromises concavity compression and

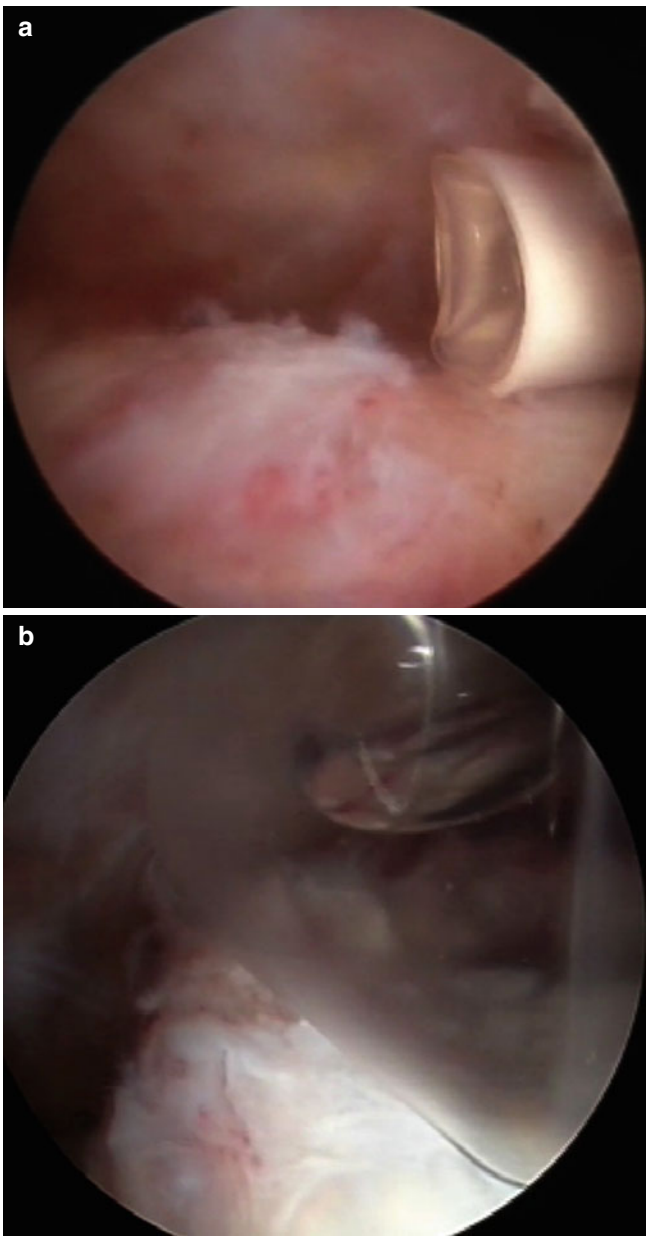

Fig. 27.5 Biodegradable spacer. (**a**) The spacer is introduced through the system deployer from the lateral portal. (**b**) The spacer is inflated to its maximal volume depending on the spacer size

alters glenohumeral load structure and direction. The deployment of a balloon spacer should reduce subacromial friction during shoulder abduction by lowering the head of the humerus and facilitating humeral gliding against the acromion during movement.

The InSpace system contains an introducer and a preshaped spacer made of poly(L-lactide-co-capro-lactone), which is a copolymer of poly-lactide and caprolactone. To enable insertion, the balloon is folded into a cylinder-shaped insertion tube, which is removed once the spacer is inserted into the subacromial space (Fig. 27.5).

The spacer degrades within 12 months, which is a period that conforms well to the rehabilitation time frame after any arthroscopic procedure performed on the rotator cuff. It is unclear, however, how long the spacer remains inflated, and it is not understood why pain and functional scores continue to improve beyond the period of spacer disintegration [84–86].

The device is contraindicated for patients with known allergy to device material or patients having active or latent infection or signs of tissue necrosis in the subacromial area.

Reverse Shoulder Arthroplasty

Reverse shoulder arthroplasty (RSA) represents the best way to treat an irreparable cuff tear associated to a severe glenohumeral osteoarthritis.

Grammont [87] introduced the semi-constrained reverse ball-and-socket design in 1985. Grammont's device shifts the center of rotation medially to the glenoid fossa to reduce the effective lever arm and distally to tension the deltoid and improve its mechanics.

There are three biomechanical advantages of using the reverse prosthesis design: the large glenosphere allows greater stability and range of motion; the glenosphere makes contact with the glenoid surface, placing the center of rotation of the shoulder within the glenoid, thereby reducing the torque on the baseplate bone interface; and the medialized center of rotation increases the number of deltoid muscle fibers recruited for abduction and lowering of the humerus places increased tension on the deltoid muscle. Furthermore, the resultant force applied to the neck of the scapula limits the shear forces which are responsible for loosening of the glenoid [87, 88].

This implant should be used only to alleviate pain and improves function in patients with cuff tear arthropathy and with an intact deltoid muscle.

There must be adequate glenoid bone stock available to implant the glenoid component securely. Contraindications include deltoid dysfunction (neurologic or structural), glenoid wear, or destruction that does not allow secure implantation of the glenoid component and active infection. Relative contraindications include younger age, rheumatoid arthritis, and surgeon inexperience with shoulder arthroplasty. Rheumatoid arthritis would at first evaluation seem to be an indication for the RSA.

The prosthesis can be implanted through a superior or a deltopectoral approach. The superior approach can be utilized in primary cases. In revision and primary surgery, the deltopectoral approach may allow improved exposure of the inferior glenoid, with better inferior placement and inferior tilt to the glenoid component. In revision cases with an exist-

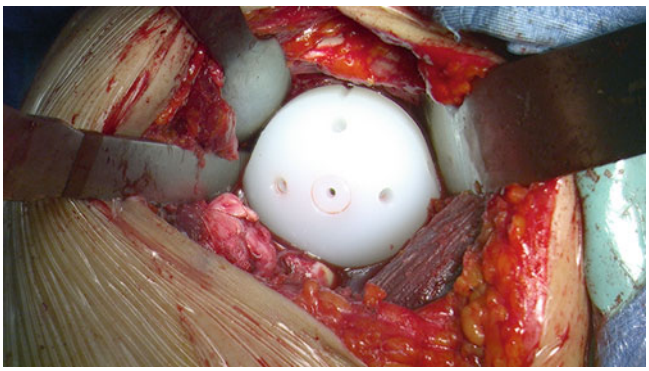

Fig. 27.6 Reverse shoulder arthroplasty: glenosphere implanted

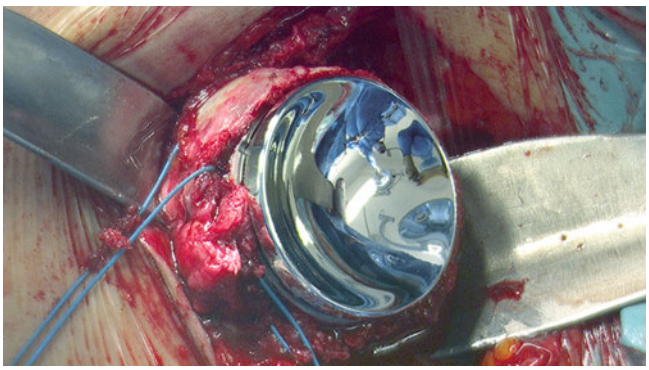

Fig. 27.7 Reverse shoulder arthroplasty: humeral component implanted

ing implant in place, the deltopectoral approach is more extensile and recommended [89].

The humeral neck cut is made at 155° (depending also from the implant used); after the humeral head and diaphysis preparation, it is possible to place a small cup that covers less than half of the glenosphere. This has the advantage of lowering the humerus resulting in overtensioning the deltoid. It allows a greater range of movement to occur before component bone impingement. Sufficient exposure of the glenoid is necessary for placement of the metaglene. The glenoid component is third of a sphere with a large diameter of 36 or 42 mm and no neck (Fig. 27.6). The posterior aspect of the glenosphere is in direct contact with the prepared glenoid surface. This design has the advantage of placing the center of rotation of the joint in contact with the center of the humeral head and provides a fixed center of rotation (Fig. 27.7). Furthermore, the large diameter allows greater range of movement before impingement of the components occurs and provides more stability.

The frequency of scapular notching, likely related to mechanical impingement by the medial rim of the humeral cup against the scapular neck in adduction, is of concern and has been suggested as a cause of glenoid loosening. Glenoid erosion by impingement of the humeral component on the inferior glenoid is often seen. It usually is not progressive but

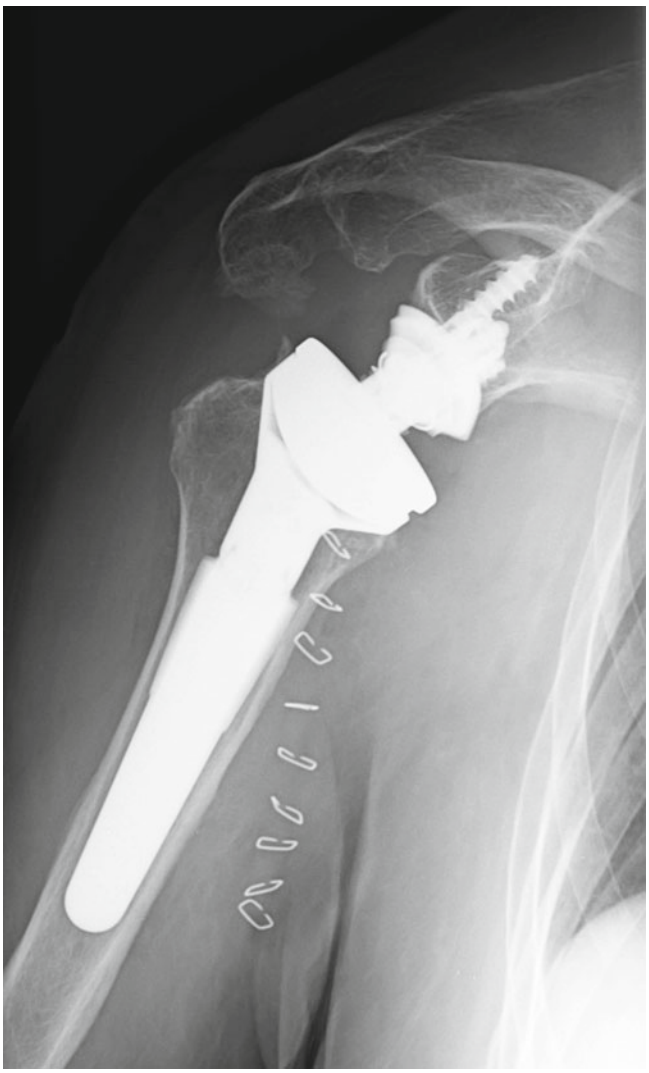

Fig. 27.8 Reverse shoulder arthroplasty: postoperative radiography

needs to be observed and revised if severe. The low positioning of the glenosphere is probably the most important factor and should avoid most of the notches [90] (Fig. 27.8).

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Calcific Tendonitis

28

Mustafa Karahan, Umut Akgun, and Baris Kocaoglu

Calcific tendonitis of the rotator cuff is a pathological process defined by reactive calcification of the tendon with an unknown etiology. Painter first mentioned calcifications in 1907. Since then, this pathology is referred to variously as calcifying tendinitis, peritendinitis calcarea, calcified peritendinitis, calcific periarthritis, and hydroxyapatite deposition disease.

Epidemiology

Various authors reported different incidence rates of calcific tendonitis. Bosworth found 2.7 % incidence of radiologically evident calcific deposits in a group of 6,061 workers [1]. Of this large group, only 35 % was symptomatic. Welfling et al. studied a group of patients with shoulder pain and reported the incidence of calcific tendonitis as 6.8 % [2]. Mostly, authors agreed that women are more commonly affected than men [3]. Peak age of incidence ranges between 30 and 50 years. Patients with diabetes are prone to develop asymptomatic calcifications [4].

Pathoanatomy

Even though the exact cause is still unknown, calcific deposits accumulate in or around the rotator cuff tendons. Two different theories, degenerative and reactive, were proposed as the cause of calcific tendonitis [5, 6]. Codman described the theory of degeneration by necrosis of the fibers of the rotator cuff followed by dystrophic calcification [5]. This theory was accepted by many authors but some argued this. Uhthoff et al. discussed that the age of the patients, course of the disease, and histology of the calcific tendonitis cannot be explained by degeneration theory [6]. Uhthoff and Loehr proposed a reactive mechanism of calcification that is actively mediated by cells [7]. According to this theory, persistent hypoxia causes fibrocartilage transformation in the tendon. He divided calcific tendonitis into three distinct stages: pre-calcific, calcific, and post-calcific (Fig. 28.1). Pre-calcific stage is characterized by metaplasia of tenocytes into chondrocytes. Calcific stage is subdivided into formative, resting, and resorptive phases. In formative phase, calcium crystals are deposited in matrix vesicles; later on these will cause larger calcific foci. In the resting phase, calcific foci are bordered by fibrous tissue. Resorptive phase is initiated by formation of thin vascular channels around the calcium deposits. Macrophages and multinucleated giant cells begin to remove the calcium. Post-calcific stage is a remodeling period of the tendon. Space occupied by calcium deposits is replaced by granulation tissues. This granulation tissue forms mature collagen fibers along the tendon axis.

Although histopathologic studies can demonstrate this pathologic sequence, the initiator of the process is still not known. Codman's theory of hypoxia is favored by many authors [8, 9]. Human leukocyte antigen serotype class A1 (HLA-A1), murine progressive ankylosis gene (ANKH), and tissue nonspecific alkaline phosphatase gene (TNAP) are some examples of molecular researches on calcific tendonitis [10].

The most common anatomical location of calcific tendonitis is the distal 1.5–2 cm of the supraspinatus tendon. This location is coherent with the theory of hypoxia because this region of the tendon is also known as the hypovascular "critical zone." This zone also exists in the infraspinatus and subscapularis tendons. Other possible sites of calcification can be listed as subscapularis tendon, infraspinatus tendon, biceps-labral complex, and subchondral bone (intraosseous) [11–13].

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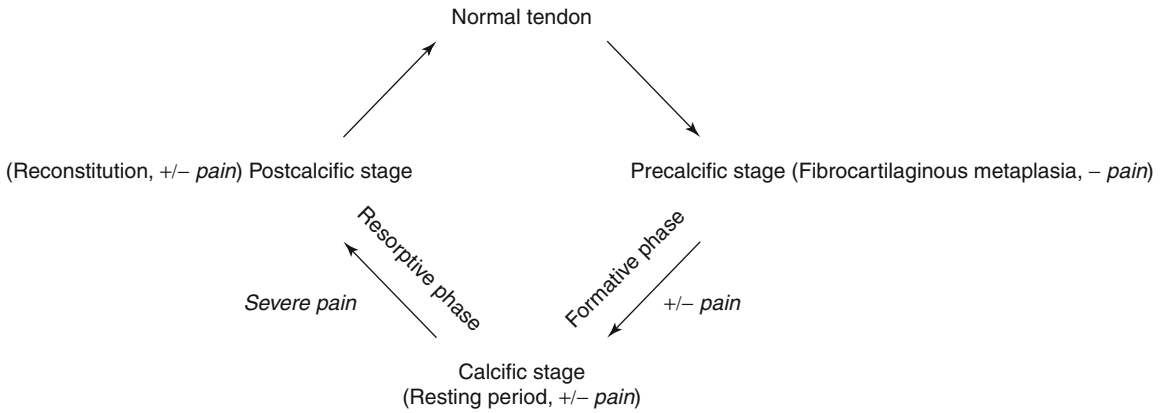

Fig. 28.1 Pathologic course and staging of calcific tendonitis according to Uhthoff and Loehr [7]

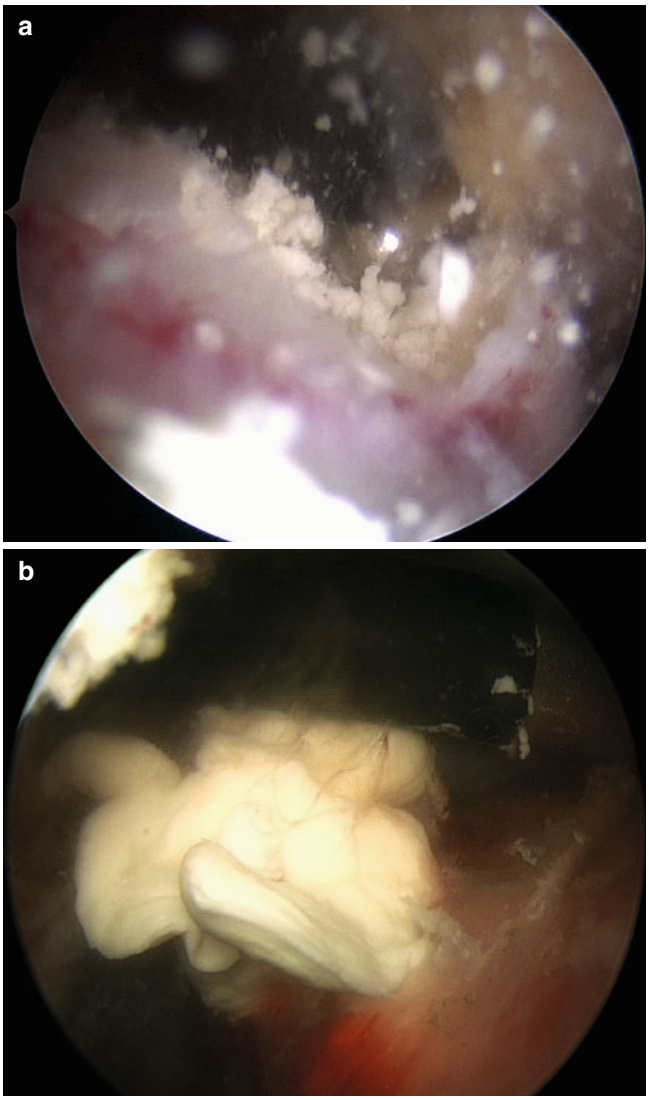

Fig. 28.2 (**a**) Subacromial view of a right shoulder subacromial view showing chalklike calcific deposits. Suction may cause a snowstorm view in the subacromial space. (**b**) Subacromial view of a left shoulder showing creamy-toothpaste calcific deposits

The nature of the calcific deposition changes in different phases of the disease. If the patient is operated in the formation phase, the calcific deposits are like chalk (Fig. 28.2a). When the patient progresses to the resorptive phase, deposits transformed to a creamy-toothpaste-like nature [14] (Fig. 28.2b).

Jim et al. showed that 25 % of patients with calcific tendonitis may have concomitant rotator cuff tears [15].

History

Uhthoff et al. showed that calcific tendinitis follows a definite progression in most patients and that resolution is inevitable, with the length of time required being the only true variable [6]. History of patients with calcific tendonitis can be best described by the three phases described by Uhthoff et al. [6]. This three-phase chronology can be useful in planning treatment:

Phase I (Pre-calcification Stage)

In the pre-calcification stage, patients generally are asymptomatic, but the process has begun. There might be slight pain with extreme range of motion.

Phase II (Calcification Stage)

This resting phase is of variable length and ends with the beginning of the resorptive phase. This stage can be exceedingly painful, and many patients seek treatment at this time. Patient can have catching, crepitus, and also intermittent pain similar to impingement. In most of the cases, there is mechanical block with acute episodes of pain.

Phase III (Post-calcification Phase)

Pain subsides markedly during this phase, but the patients are unable to reach that phase without medical intervention.

Imaging

Standard Radiology

Standard radiographs must be obtained whenever calcification of the cuff is suspected. Radiographic evaluation is also important during follow-up examinations because it permits assessment of changes in density and extent of calcification [4]. Initial radiographs should include anterior-posterior views with the shoulder in the neutral position and in internal and external rotation [16]. Deposits in the supraspinatus are readily visible on films obtained in neutral rotation (Fig. 28.3a), whereas deposits in the infraspinatus and teres minor are best seen on internal rotation views [4, 16]. Calcifications in the subscapularis occur only in rare instances; a radiograph obtained with external rotation will show them well. Scapular views will help to determine whether a calcification is causing impingement. Calcium deposits are often visible on radiographs, particularly in the acute or resorptive phase [4, 16]. Most authors agree that radiographic evidence of degenerative joint disease is usually lacking in patients with calcific tendonitis. This is true for patients in the fourth and fifth decades of life, when calcifying tendinitis peaks. Calcifications seen in arthropathies have a quite different appearance. They are stippled and overlie the bone insertion and are always accompanied by degenerative osseous or articular changes. These calcium deposits must be clearly distinguished from reactive intratendinous calcifications [7, 17].

Computerized Tomography (CT)

Calcific tendonitis could be seen on CT as an incidental finding [18]. On CT, deposits that appear well defined on radiographs generally appear homogeneous. CT is the modality best suited for evaluating osseous involvement, particularly if there are aggressive changes like osseous erosion [19]. These changes are most often seen in the femur and humerus, with cortical erosion [20, 21]. CT may also be helpful for evaluating calcification in unusual locations, thereby localizing the disease to a tendon and confirming calcific tendonitis [16]. CT is the most accurate modality to predict the consistency of calcific deposits. This could be important when planning intervention, for example, needle aspiration. Soft or semiliquid

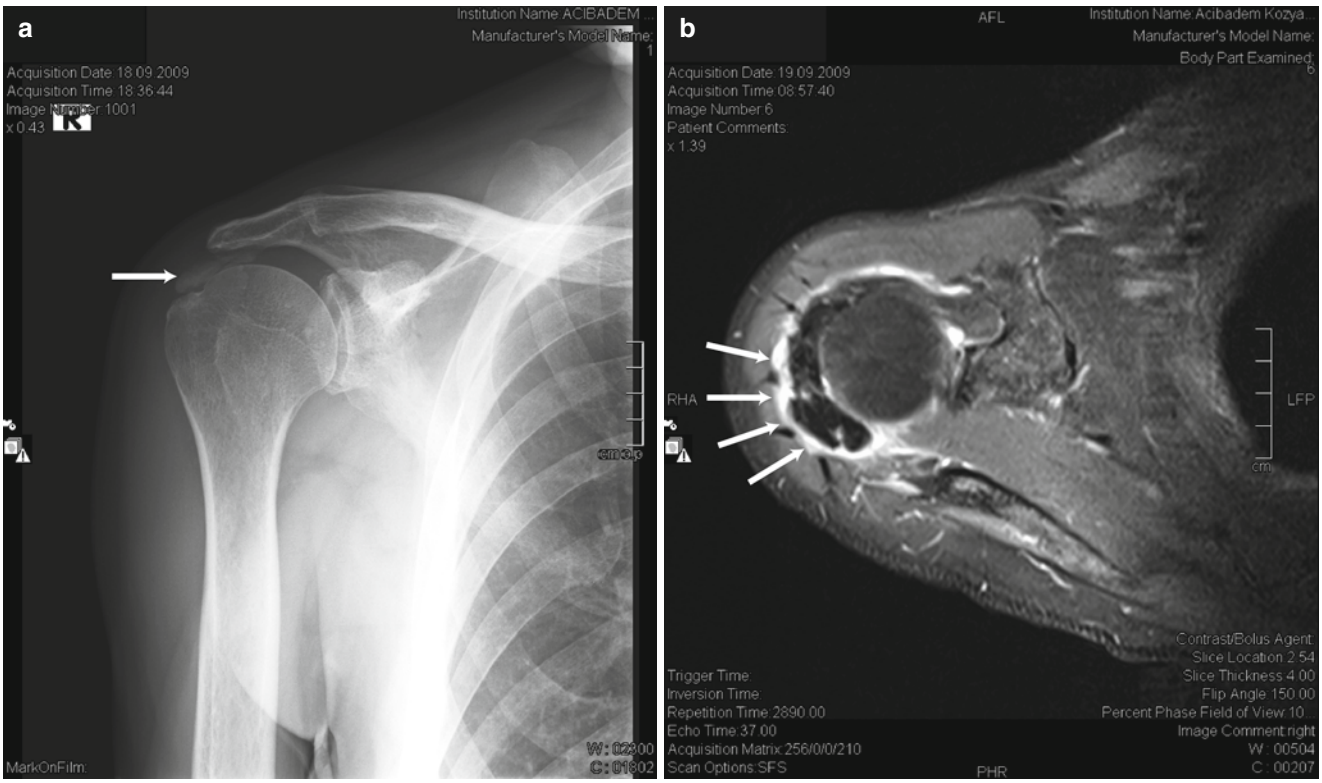

Fig. 28.3 (**a**) Anterior-posterior view of a right shoulder; *white arrow* pointing a huge calcific deposit located in the subacromial space. In neutral rotation, this deposit is presumably located in the supraspinatus tendon. (**b**) T2-weighted axial MR image of the same patient; *white arrows* pointing the huge calcific deposit located in the supraspinatus and infraspinatus tendons

calcifications can have a heterogeneous appearance on CT; on the other hand, hard or solid calcifications appear more homogeneous and have a higher density [16].

Ultrasound (US)

US is useful in evaluation of calcific tendonitis, particularly in the shoulder [22]. US could be used in both diagnostic and therapeutic procedures [16, 22]. Tendon calcification is seen as a hyper-echoic focus, with or without posterior acoustic shadowing [16]. US is reliable in detection and localization of rotator cuff calcifications but is unable to classify the pathologic phase. Because of this limitation and the possibility of other pathologic conditions of bone, radiographs should be obtained in conjunction with US [16]. In a study of 217 patients, Hartig and Huth found US more sensitive than radiography in detecting calcium deposits [23]. The deposit was visualized by US in 100 % of cases but was shown radiographically in only 90 %. In addition, US permits more exact localization of the deposit without subjecting the patient to radiation.

Magnetic Resonance Imaging (MRI)

MRI may be indicated in rarely. On T1-weighted images, calcifications appear as areas of decreased signal intensity. T2-weighted images frequently show a band of increased signal intensity compatible with edema (Fig. 28.3b). Direct MR arthrography (MRA) for calcific tendinitis of the shoulder was recently evaluated by Zubler et al. and was reported to be insufficient in the diagnosis of calcific tendonitis, because small calcific deposits may be difficult to visualize and lead to false-negative results and normal hypo-intense areas within tendons may lead to false-positive results [24].

Treatment: Indications and Contraindications

Pain in calcific tendonitis can be chronic or acute. Pain can be generated by 4 different mechanisms: chemical irritation of calcium, pressure within the tissue by edema, impingement-like bursal thickening, and chronic stiffening of the glenohumeral joint [25]. In chronic cases, natural progress of the disease is usually paused at formative phase. On the other hand, resorptive phase is usually characterized by acute pain. Widely accepted first-line treatment of calcific tendonitis is a conservative measure. Physical therapy, nonsteroidal anti-inflammatory drugs (NSAIDs), subacromial injections, ultrasound, extracorporeal shock wave lithotripsy (ESWL), needling, and lavage are some examples of conservative options.

Surgery is only needed when conservative measures are failed. Gschwend et al. listed three indications for surgery, progressive symptoms, pain interfering with daily living, and resistance to conservative therapy [26].

Decision-Making Algorithm

Patients with calcified tendonitis of the shoulder usually present with acute shoulder pain irrelevant to physical findings. Simple shoulder X-ray will reveal the calcification. Skipping X-rays and proceeding to MRI may result in false diagnosis.

Patients should be treated conservatively initially. Conservative treatment should be aimed at relieving pain, reducing inflammation caused by the protuberance of the deposit, resorption of the deposit, and preservation of musculature. Simple analgesic medication, sling, NSAIDs, and local cortisone injections will reduce pain and inflammation. Percutaneous needling and ESWL may dissolve the deposit. Physical therapy and rehabilitation will help the patient to restore normal biomechanics of the shoulder.

A small number of the patients will require surgery. Arthroscopic debridement of the calcified deposit is successful in most of the patients. Surgeon should be alert towards development of postoperative stiffness.

Arthroscopic Treatment: Surgical Technique

Patient Positioning

Author prefers beach chair position in all subacromial procedures. Interscalene regional anesthesia may be helpful for postoperative pain management. Patient-controlled subacromial analgesia can be combined with general anesthesia in selected patients.

Portals

Classic posterior and anterior portals are used for glenohumeral assessment. Lateral portals are prepared according to the position of the calcific deposits.

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

Although the preoperative imaging may guide us towards the lesion site, a stepwise procedure would be easier to find the calcific deposits. Glenohumeral arthroscopy is the first step. If calcific deposits are localized in the biceps or subscapularis tendons, it would cause a bulginess in the tendon easy to find

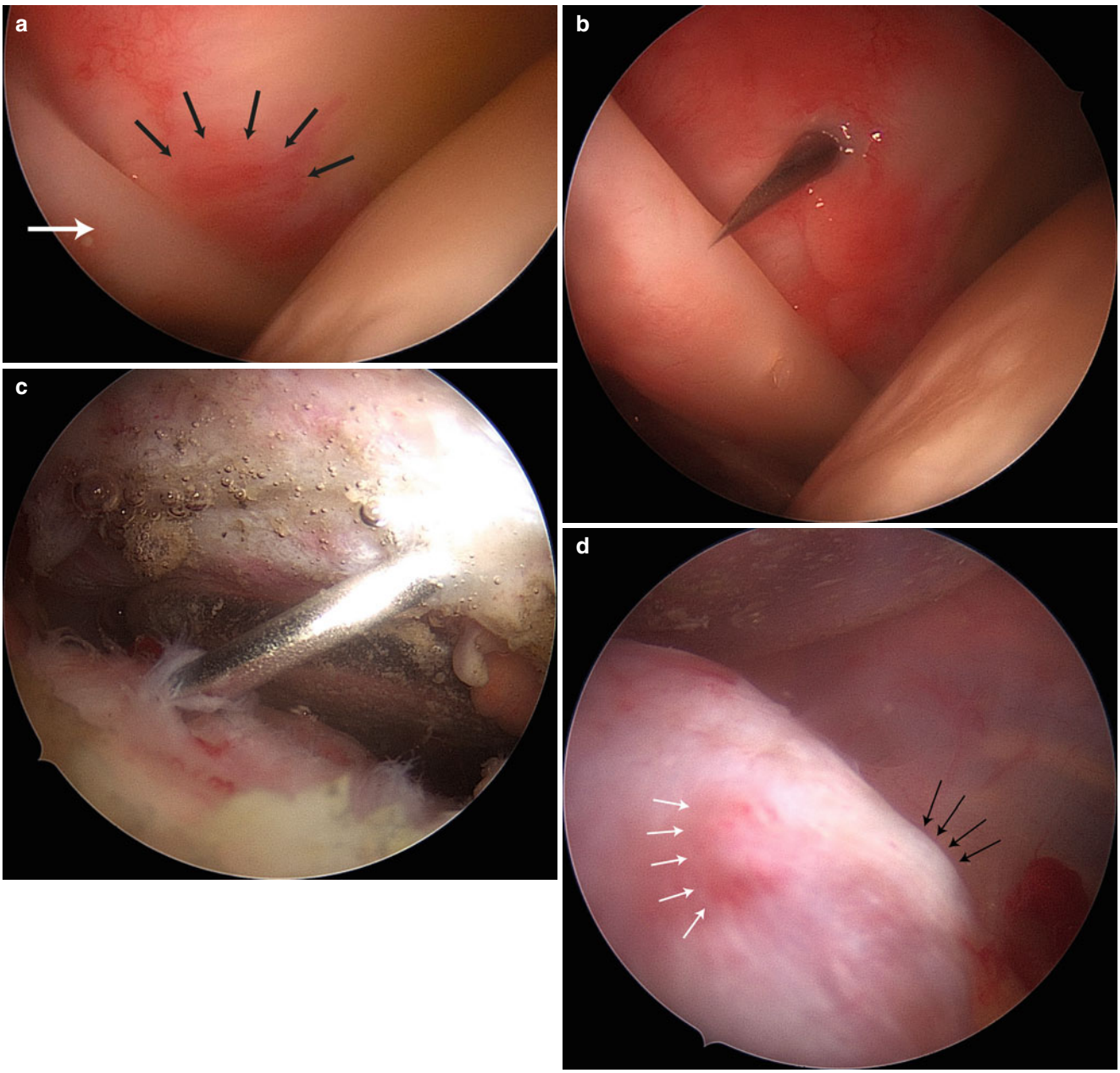

Fig. 28.4 (**a**) Arthroscopic view of a right glenohumeral joint from the posterior portal; *black arrows* pointing a hypervascular area in the rotator cuff just posterior to the long head of biceps (*white arrow*). (**b**) Hypervascular area is marked with a needle which will be used as a guide in the subacromial space. (**c**) Arthroscopic view of the subacromial space from the posterior portal showing the guide needle previously placed to mark the hypervascular area. (**d**) Arthroscopic view of the subacromial space from the lateral portal. The needle was removed and subacromial bursae was cleaned. *White arrows* pointing a hypervascular area and *black arrows* pointing puffiness within the rotator cuff. This area is the location of the calcific deposits

out during glenohumeral arthroscopy. Most of the time, calcific tendonitis affects the supraspinatus tendon and this does not cause an evident bulginess on the articular side of the tendon. Articular side of the supraspinatus, infraspinatus, and teres minor tendons should be observed from both posterior and anterior view. A local reactive hypervascular area is a good sign for localizing the deposits (Fig. 28.4a). Once this hypervascular area on the articular side of the tendon is found, this zone is marked by a spinal needle or a suture from the subacromial space (Fig. 28.4b). On the next step, scope should be placed in the subacromial space. Subacromial lateral portal can be prepared in line with the spinal needle used to mark the possible site of calcific deposit. In chronic cases, there would be a hypertrophic subacromial bursa. In order to examine the subacromial side of the rotator cuff tendons, subacromial bursa should be debrided. As bursal inflammation may increase

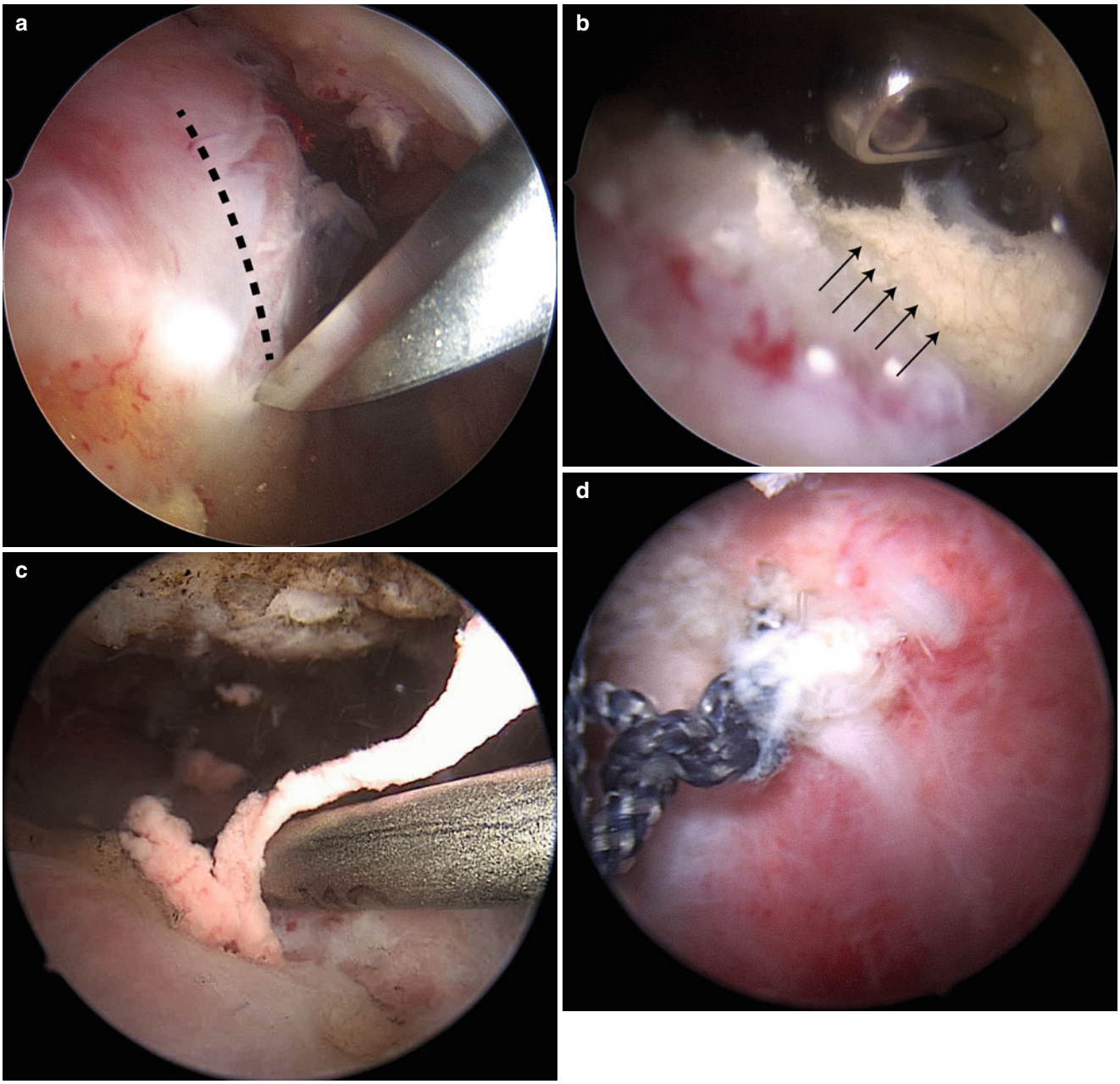

Fig. 28.5 (**a**) Arthroscopic view of the subacromial space of a right shoulder from the posterior portal; N-11 blade is introduced from the lateral portal, a superficial cut over the calcific deposits will be done in line with the tendon fibers (*black dashed line*). (**b**) Shaver is used to clean out the calcific remnants within the cuff. *Black arrows* pointing chalklike deposits within the cuff. (**c**) A probe is used to remove milk creamy deposits through the superficial cut on the cuff. (**d**) Defect within the tendon is repaired by a simple side-to-side suture

bleeding, bursectomy can be done by a radiofrequency probe to prevent bleeding. When the subacromial bursectomy is completed, previously placed needle or suture on the tendon should be found (Fig. 28.4c). Calcific deposits may cause a prominent bulginess or a swelling on the bursal side of the cuff (Fig. 28.4d). If not, gentle probing of the tendons and rotating the shoulder may be helpful to localize the deposits. A hypervascular area may be evident over the effected tendon. If the location cannot be found arthroscopically, fluoroscopy can be used to mark the calcification. The suspected area of the tendon can be needled cautiously; when the needle hits the calcific deposit, a leakage of calcium may be seen in the subacromial space.

Step-by-Step Procedure (Box 28.1)

Once the location of the calcific deposit is identified, a needle or a N-11 scalpel blade can be used to make a

superficial cut on the surface of the tendon. This cut should be parallel to the longitudinal fibers of the tendon in order to cause the least possible damage to the tendon (Fig. 28.5a). This will also simplify the later repair of the tendon if needed. Through this cut, calcific deposits can be debrided. Chalklike deposits can be sucked by using a small-sized shaver (Fig. 28.5b) whereas creamy deposits can be milked by using an arthroscopic probe (Fig. 28.5c). A small smooth curette can be used to remove the possible calcific remnants within the tendon, but this should be done with care in order to prevent damage to the healthy tendon tissues. Debridement of the calcifications should be done as much as possible. At this point, adequate irrigation is very important to remove calcium remnants within the subacromial space. In the end, if the resultant defect within the tendon is more than 10 mm or a full-thickness lesion was made in the cuff, author prefers a side-to-side repair with non-sliding knots (Fig. 28.5d). Sliding knots should be avoided because they may damage the tendon while sliding. Knots should not be placed in a firm manner, because an increased pressure within the tendon may prevent healing of the tendons and cause prolonged postoperative symptoms.

In some cases, calcific deposits may be found within the footprint of the cuff tendons. In these unusual types, resultant defect cannot be repaired by side-to-side sutures and suture anchors are needed.

Author does not prefer to perform a routine acromioplasty. In selected cases with signs of radiographic or arthroscopic acromial spurs and/or impingement symptoms, acromioplasty can be added to this procedure.

Box 28.1: Tips and Tricks

- Determining the exact location of the calcific deposit prior to surgery will reduce the operation time. On an anteroposterior X-ray, deposits in the supraspinatus tendon move medially when the arm is internally rotated whereas lesions in the infraspinatus move laterally as the arm is moved into internal rotation.
- Scattering of the deposit into the subacromial space may cause extensive inflammatory response in the acute postoperative period which should be addressed with proper anti-inflammatory medication that may include cortisone.
- Finding the exact location of the deposit from the subacromial space is not always precise. Visualization of increased vascularity in the rotator cuff through glenohumeral approach will enable to pinpoint the exact location.
- Insertion of a percutaneous needle through the deposit under glenohumeral arthroscopic vision will allow the surgeon to locate the deposit precisely during subacromial arthroscopy. A monofilament suture may be passed through the needle since the needle may displace while maneuvering the shoulder.
- Supraspinatus insertion into the greater tuberosity may be confused with calcific deposit. Most of the time, the deposit is recognized by the tagging or direct visualization. If not recognized, repetitive multiple needling of the rotator cuff will cause the calcium deposit to leak. The surroundings of the deposit should thoroughly be cleared of bursitis avoiding any rotator cuff injury.
- If the deposit is soft, usually shaving the lesion will be sufficient, a N-11 blade may be required to cut away the deposit into pieces. Retaining cuff integrity throughout the procedure is of utmost importance.
- It is not necessary to repair the cuff unless it is a total rupture. Acromioplasty or cuff repair increases risk of postoperative stiffness.
- The patients should start full passive range of motion as soon as possible.

Postoperative Care

Rehabilitation program can be changed according to surgical procedure (debridement ± cuff repair). Rehabilitation could be initiated immediately after arthroscopic debridement surgery. Arm sling after the surgery could be used when outside the home. For the treatment of the upper extremity edema, hand-squeezing exercises should be prescribed. One week after surgery, active elbow and shoulder pendulum exercises could be initiated. After the first week, shoulder passive anterior elevation and external rotation exercises could be started to increase the range of motion. Thus, hot and cold application and active scapula stretching exercises could be performed. As tolerated, shoulder stretching and strengthening exercises could be started. Internal rotation and adduction stretching and also scapular strengthening exercises can be added.

Two months after surgery, stretching exercises at all plans and strengthening against variable load with arm at 90° could be started. Initiating daily living exercises including load carrying and handling also makes the patient cope with social life.

Literature Review

Although this chapter is dedicated to the arthroscopic treatment of calcific tendonitis, most of the cases can be solved by conservative measures. Main problem in calcific tendonitis is pain. Throughout the years, rest (immobilization), heat, nonsteroidal medication, physical therapy, ultrasound, and extracorporeal shock wave lithotripsy (ESWL) have been used to decrease pain. There have been varying reports on the success of these measures. NSAIDs are the initial treatment line. A formal physical therapy program or gentle exercises may help maintain range of motion. Ultrasound is an adjunctive therapy mostly done for pain management. There is mixed evidence that active therapeutic ultrasound is more effective than placebo ultrasound [27]. In a well-designed randomized double-blind comparative study on ultrasonography and sham insonation in patients with symptomatic calcific tendinitis, ultrasound treatment resulted in greater decreases in pain and greater improvements in quality of life in addition to radiographic decrease in calcium deposit size [28]. Extracorporeal shock wave lithotripsy (ESWL) has originated from Europe. In ESWL, low energy is used to decrease pain, whereas high energy is used to break up calcium deposits. Loew et al. [29] randomly assigned patients to control groups, low-energy groups, high-energy groups, and high-energy groups that received either one or two sessions. The results showed energy-dependent success, with relief of pain ranging from 5 % in the control group to 58 % after two high-energy sessions. Daecke et al. [30] determined long-term effects and complications of ESWL. They concluded that the level of success was energy dependent and that there were significant differences in radiologic changes between the groups in a prospective study evaluating 115 patients at 4-year follow-up. At the end of the 4 years, 20 % of the entire patient population had undergone surgery on the involved shoulder.

Those who have not benefited from the conservative measures above may need invasive interventions to treat calcific tendonitis. Invasive interventions include steroid/anesthetic injection, "barbotage" (multiple needle punctures), aspiration, and irrigation. Subacromial steroid injection may be helpful if some of the symptoms come from impingement [31]. Needle lavage technique is best used in patients with an acutely painful shoulder in the resorptive phase, and it will help to decrease the intratendinous pressure that results in pain. Treatment with modified ultrasound-guided fine-needle technique has been shown to be an effective therapy with a significant clinical response and perhaps greater precision [22]. Using ultrasound-guided needle puncture, Farin et al. [32] found favorable results in more than 70 % of patients.

Progressive symptoms, pain interfering with daily living, and resistance to conservative therapy are summarized as surgery indications by Gschwend et al. [26]. Open surgical removal of calcific deposits was first performed by Harrington and Codman in 1902 [5]. Different authors reported good results with this procedure. Best results were obtained in patients with chronic, progressive symptoms continued more than 1 year before surgery [33]. In 1987, Ellman described arthroscopic technique for removing calcific deposits [34]. Later, many authors reported good clinical results with arthroscopic techniques [35, 36]. Main advantages of the arthroscopic surgery can be summarized as shorter hospitalization and recovery time with better cosmesis.

Summary

Calcific tendonitis of the rotator cuff is a pathological process defined by reactive calcification of the tendon with an unknown etiology. Over the years, the treatment focused on decreasing the pain that involved rest, heat, nonsteroidal medication, physical therapy, "needling," and localized injections of anesthetic and corticosteroids. More than 90 % of patients can be treated by conservative measures. Failure to respond to nonoperative treatment may necessitate surgical intervention. Harrington and Codman performed the first reported operative removal of a calcific deposit [5]. Later, arthroscopic treatment for these deposits has been described with successful results [35, 36]. This method continues to be the trend in surgical treatment where the conservative treatments are failed.

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Adhesive Capsulitis

29

Matteo Salvatore, Carmine Latte, Giuseppe Milano, and Andrea Grasso

Introduction

Despite extensive research, the adhesive capsulitis of the shoulder is pathology of uncertain etiology and with poor consensus regarding the optimal method of treatment.

The first appearance of a description of a pathologic condition of the shoulder generically defined as scapulohumeral periarthritis that leads to stiffness hails in the nineteenth century in France [1] and in the USA [2]. In 1934, Codman [3] first introduced the term "frozen shoulder" to identify a clinical entity difficult to define, to treat, and to explain. Neviaser [4] identified as "adhesive capsulitis" a "chronic inflammatory process involving the capsule of the shoulder causing a thickening and contracture of this structure which secondarily becomes adherent to the humeral head." Zuckerman and Cuomo [5] defined the condition as a condition characterized by "significant restriction of both active and passive shoulder motion that occurs in the absence of a known intrinsic shoulder disorder."

Epidemiology

The true incidence of adhesive capsulitis in general population varies from 2 to 5 % [6–8]. Women aged between 40 and 60 years are most commonly affected [7, 8] often bilaterally even if the pathology affects the contralateral side years after onset of symptoms in the first shoulder [9, 10]. Recurrence in the same shoulder is unusual, and the condition has not been reported to have a predilection for ethnicity.

Pathophysiology

Classification

Nevertheless the numerous schemes of classification existing, the one preferred by the authors classifies adhesive capsulitis as primary and secondary. The first group, also known as idiopathic, includes all the forms in which no findings on history (although diabetes mellitus does concur) or examination explain the onset of disease. Secondary or acquired group depends from known causes: among these, we can identify intrinsic and extrinsic cause (Fig. 29.1). Rotator cuff

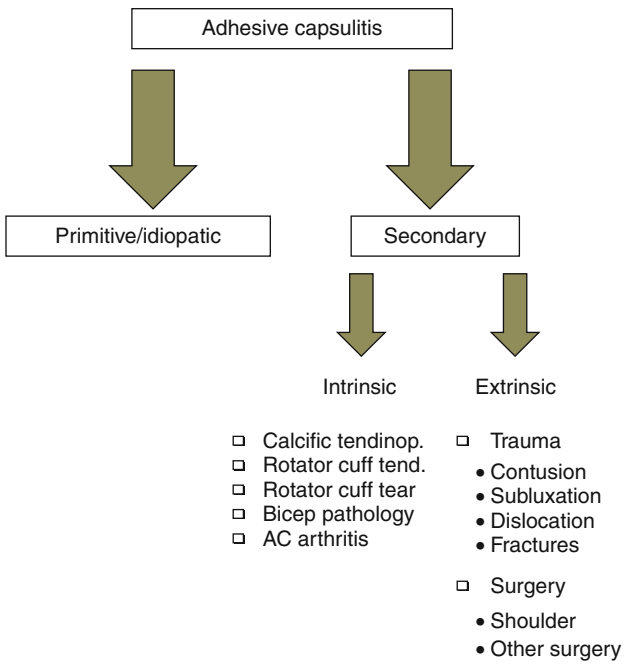

Fig. 29.1 Etiology of adhesive capsulitis

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tendonitis and rotator cuff tears, biceps pathology, calcific tendonitis, and acromioclavicular arthritis are the most common intrinsic cause, while a shoulder trauma or surgery performed on the shoulder or "around" it represents the extrinsic cause.

The most common traumatic event of the upper humerus such as contusion, subluxation, dislocation, and fracture may result in a restriction of shoulder motion, while rotator cuff surgery and anterior or posterior capsulorrhaphy, performed both arthroscopically or open, are possible causes of shoulder stiffness. Some authors reported a higher incidence of capsulitis even if the surgery is performed near the shoulder region, i.e., mastectomy performed for breast cancer, especially when associated with axillary lymph nodes dissection [11] or neck dissection [12].

Comorbidities

Many comorbid medical factors have been related with increased pain and dysfunction of the shoulder [6] and can be present in either primary or secondary adhesive capsulitis. In particular hormonal dysfunction, cardiac diseases, neurologic pathology, and many others like malignancy, Dupuytren disease, hyperlipidemia, and drugs-related diseases can be associated to this pathology.

The strong association between diabetes mellitus and adhesive capsulitis is well known. Bridgman [7] first described it after observing a five-time higher incidence among 800 diabetic patients with respect to 600 nondiabetic. Arkkila et al. [13] reported an overall incidence of 10.3 % in patients with type I diabetes and 22.4 % in patients with type II diabetes. The rate of bilateral frozen shoulder was higher among diabetic patients with respect to general population with long-lasting symptoms and more severe stiffness [14]. There was no association found between glycemic control and the prevalence of frozen shoulder in the diabetic population studied by Yian et al. [15].

Less well-known hormonal dysfunctions related with capsulitis are thyroid disease and ACTH deficiency; in particular, Cakir et al. [16] reported a 10.9 % incidence of capsulitis in patients with thyroid disease.

Tuten et al. [17] verify a 3.3 % incidence of capsulitis in male cardiac surgery patients (7 of 214). Also Parkinson's disease and neurosurgery seem to be related with a relative higher incidence of shoulder stiffness [18, 19]. Gheita et al. [20] in their series had 9 patients of 60 with malignant disease with adhesive capsulitis.

Few studies reported that adhesive capsulitis occurs two to three times more frequently in twins than in normal population, even if this result may be related to individual-specific environmental factors rather than a true genetic component [21]. In fact, still controversial is the existing genetic predisposition for frozen shoulder. Instead, more authors reported a strong association with the Dupuytren's disease: patients affected have eight times more chances to develop a frozen shoulder compared with general population [22, 23]. Finally Hand et al. [8] in their series found a 17% of capsulitis associated with hypercholesterolemia, moreover vaccination of anti-influenza and pneumococcal infection [24], use of fluoroquinolones [25], and highly active anti-retroviral therapy for HIV infections seem to be related with frozen shoulder [26].

Etiologic Theories

The etiology and pathophysiology of adhesive capsulitis, although the improvement in basic science research, remain poorly understood [27]. The role of both immunologic factors, cell signaling, and inflammatory mediators has been supported [22, 28–31]. Most information comes from resistant cases requiring surgical treatment, performed open or arthroscopically.

To have a shoulder movement restriction is necessary, anatomically, a contracture of the rotator interval, coracohumeral ligament, and anterior capsule. In fact, release of the coracohumeral ligament, such as shown by Neer et al. [32], restores external rotation of the shoulder. Shoulders with and without adhesive capsulitis show significantly different rotator interval dimensions [33] and thickening of the axillary pouch [34].

Neviaser [4] during open surgery found capsular and synovial inflammation that lead to adherence of the axillary pouch. Subsequent studies have shown evidence of thickening and contracture of the inferior capsule rather than adherence of the axillary fold [35].

The histologic examination shows perivascular infiltration and capsular fibrosis such as usual pattern of the adhesive capsulitis. The synovial hyperplasia and capsular fibrosis are derived from the activity of cytokines such as transforming growth factor β (beta) (TGFβ), platelet-derived growth factor (PDGF), interleukin 1b, and tumor necrosis factor (TNF), as demonstrated from the biopsy samples of patients with adhesive capsulitis [30].

Hannafin et al. [36], studying biopsy specimens of patients with adhesive capsulitis, attempted to correlate clinical examination and arthroscopy with the three histopathologic phases of fibroplasia, previously described by Neviaser. The capsular fibroplasia and contracture seem to depend to the hypervascular synovitis that determines a progressive fibroblastic response in the capsular tissue. The authors proposed a cellular pathway, which finally might result in the clinical scenario of an adhesive capsulitis.

The hypervascular and highly cellular collagenous tissue, composed primarily of fibroblasts and myofibroblasts, represents the typical histologic and immunohistochemical finding.

It has also been postulated that the active fibroblastic process that occurs in adhesive capsulitis is similar to that which occurs in Dupuytren's disease [22]. The explanation of this intuition comes from a family of naturally occurring proteinases that control collagen matrix remodeling, namely, the matrix metalloproteinases (MMPs) [28]. Hutchinson et al. [37] reported 12 cases of frozen shoulder in patients affected by gastric carcinoma after treatment with MMPs inhibitor.

Bunker et al. [28] examined the capsular tissue from patients with frozen shoulder, Dupuytren's contracture, and from normal controls. They analyzed the tissue for various factors, including MMPs, and found overexpression of messenger RNA (mRNA) for MMPS and natural MMP inhibitors in the capsule of patients with frozen shoulder with respect to the tissue coming from the patients with Dupuytren's disease and with normal capsule. They concluded the study assuming that the expression of the MMP is a possible factor involved in frozen shoulder, even if other studies need to be performed to establish the causal relationship.

Finally, well known is the high incidence of adhesive capsulitis in patients with diabetes mellitus; however, a true explanation of this relationship is not been yet published. One of the hypotheses postulated says that excessive glucose concentration can determine a faster rate of collagen cross-linking and glycosylation in the shoulder capsule [38, 39]. This process over time can determine a restriction in the shoulder movement possibly responsible of an adhesive capsulitis. The higher incidence of this cross-linking in the collagen may be also the reason of the contracture in the Dupuytren's disease.

Natural History

Reeves [9] distinguished three sequential stages: the painful stage, the stiff stage, and the recovery stage. Hannafin and Chiaia [40], based on the clinical and histological findings described by Neviaser, [41] identified four stages:

- *Stage 1*, the pre-adhesive stage. The patients present mild end-range pain and are often misdiagnosed as having rotator cuff impingement. In this stage, arthroscopic findings demonstrate mild erythematous synovitis.
- *Stage 2*, the acute adhesive or "freezing" stage. The patients frequently have a high discomfort and pain near end range of movement. Even if it seems impossible to move also passively the arm, examination under anesthesia reveals connective tissue changes resulting in a relatively loss of motion; arthroscopic features are characterized by a thickened red synovitis.
- *Stage 3*, the fibrotic or "frozen" stage. The patients note an improvement of pain but significant stiffness. Examination under anesthesia reveals equal passive motion compared to when awake; arthroscopic features are represented by less synovitis but more mature adhesions.
- *Stage 4*, the "thawing" phase. The patients present painless stiffness and motion that typically improves by remodeling. Severe capsular restriction without apparent synovitis is a common arthroscopic finding [42].

History

Even if specific diagnostic criteria do not exist, patients with primary adhesive capsulitis often present a consistent history and clinical examination. An insidious onset, a progressive increase in pain, and gradual loss of motion are typical of idiopathic adhesive capsulitis and some secondary ones (e.g., secondary to diabetes mellitus). Often a minor traumatic event may coincide with the patient's first recognition of symptoms. Pain, specifically sleep disturbing night pain, frequently motivates the patient to seek medical advice. Most patients are comfortable with the arm at the side or with mid-range activities but often describe a sudden, transient, excruciating pain with abrupt or end range of movements.

With the only history taking, we can presume the stage or irritability level of the patient's condition. If the patient is able to sleep through the night, it indicates less irritability. It also indicates that the painful synovitis/angiogenesis is resolving as consistent with stage 3.

It is important to distinguish if pain or stiffness is the predominant symptom. The patient experiencing more stiffness than pain likely has less symptomatic synovitis/angiogenesis and more fibrosis. It is also important to investigate if the symptoms have been improving or worsening over the last 3 weeks. Improving symptoms may indicate that the patient is advancing from stage 2 to stage 3 and that the irritability level is decreasing. Recognizing the extent of tissue irritability has a direct influence on the plan of care.

Clinical Examination

A full upper-quarter examination should be performed to rule out cervical spine and neurological pathologies. The challenges in diagnosing adhesive capsulitis are differentiating true glenohumeral loss of motion from pain-related guarding and identifying any concomitant conditions.

Observing patient since her/his entrance in medical office can give useful information about her/his shoulder problem. Patients with advanced adhesive capsulitis may have lost the natural arm swing that occurs with walking. Moreover, muscle atrophy of the shoulder girdle may be present. As a result of impaired motion in the glenohumeral joint, abnormal scapular movement may be observed with active forward flexion of the affected shoulder. It is important to remember that a

physical examination of a patient with adhesive capsulitis can be uncomfortable, and the patient may need to briefly rest or gently "shake out" the shoulder between maneuvers.

A vague, diffuse tenderness over the anterior and posterior shoulder regions could be yielded by palpation. Some authors noted that in adhesive capsulitis, the digital pressure on the area of the coracoid process elicits local pain (coracoid pain test) and could be considered as a pathognomonic sign of adhesive capsulitis [43].

Focal tenderness over other specific structures is rare; its presence suggests another diagnosis or concomitant pathology, such as rotator cuff or biceps tendinopathy.

Loss of motion with forward flexion, abduction, and external and internal rotation should raise suspicion for adhesive capsulitis. With frozen shoulder, the examination of the shoulder typically reveals significant limitation of both active and passive elevation, usually less than 120° [22], but loss of motion is stage dependent. Scapular substitution frequently accompanies active shoulder motion [44].

Passive motion should be assessed supine to appreciate the quality of the resistance to motion at the end range of movement (end feel). It is important to compare these maneuvers on the affected and unaffected sides to accurately assess deficits. The patient should initially be asked to actively test the limits of motion; if loss of motion is observed, the physician may assist passively, with scapular stabilization to ensure an accurate measurement of movement.

The Apley scratch test is the most widely accepted method for measuring internal rotation; it evaluate the highest vertebral level reached.

A greater than 50 % reduction in passive external rotation or less than 30° of external rotation, when measured with the arm at the side, is a common finding in individuals with frozen shoulder [22, 45, 46].

Full range of motion in any plane suggests another diagnosis. Frequently, passive glenohumeral motion is very restricted due to pain at or before end range, and muscle guarding can often be appreciated at end range. Partial improvement in motion related to diminished pain and muscle guarding has been reported after local or regional anesthetic [47].

In adhesive capsulitis, strength should theoretically be preserved in all planes because it does not affect the dynamic stabilizers of the shoulder (i.e., rotator cuff, biceps tendon, and deltoid muscle); however, patients with adhesive capsulitis may not have enough range of motion to perform strength testing. Special tests, such as impingement signs and Jobe's test, are not helpful in differentiating frozen shoulder from rotator cuff tendinopathy because they require painful end-range positioning.

Resisted strength testing can result in pain-related "breakaway" weakness that mimics true weakness. Patients with advanced adhesive capsulitis may also have muscular atrophy that can cause weakness. Leggin et al. [48], using handheld dynamometry, revealed significant weakness of the shoulder internal rotators and elevators in these patients. The shoulder internal rotators were significantly weaker in patients with frozen shoulder compared to patients with rotator cuff tendinopathy; however, significant weakness of the external rotators and abductors was also found relative to the uninvolved side.

Summarizing adhesive capsulitis differentiates from other pathologies for significant loss of passive external rotation with the arm at the side, as well as loss of active and passive motion in other planes of motion. Attention should be paid to other shoulder pathologies resulting in significant loss of external rotation with the arm at the side including proximal humeral fracture, severe osteoarthritis, acute calcific bursitis/tendinitis, and a locked posterior dislocation. Early frozen shoulder may be difficult to differentiate from rotator cuff tendinopathy because motion may be minimally restricted and strength testing may be normal. The patient with a slight loss of passive external rotation motion at the side and relatively full motion in all other directions should be cautioned to return for further evaluation if the patient experiences a rapid progression of shoulder pain and stiffness.

Imaging

Diagnosing adhesive capsulitis is often achieved by physical examination alone, but imaging studies can further confirm the diagnosis and rule out underlying pathology.

Radiography rules out pathology to the osseous structures. Arthrography has been used to determine decreased glenohumeral joint volume associated with adhesive capsulitis [49].

The arthrographic criteria of adhesive capsulitis include the following: limited injectable fluid capacity of the glenohumeral joint [7–10 cc], a small dependent axillary fold, and irregularity of the anterior capsular insertion at the anatomic neck of the humerus [50].

Binder et al. [51] demonstrated bone scans possess little diagnostic or prognostic value for frozen shoulder.

Ultrasonography has gained favor because it can help differentiating rotator cuff tendinopathy from adhesive capsulitis. A recent study revealed fibrovascular inflammatory soft tissue changes in the rotator interval in 100 % of 30 patients with frozen shoulder [52].

Recently, magnetic resonance imaging (MRI) has been proposed for helping the differential diagnosis by identifying soft tissue abnormalities of the rotator cuff and labrum [53]. MRI has identified abnormalities of the capsule and rotator interval in patients with frozen shoulder. Sofka et al. [54] demonstrated how MRI of the shoulder is an effective and noninvasive means of diagnosing suspecting cases and also

provides information that may assist the clinician in differentiating between the early and late stages. Capsule and synovial thickness, as measured in the axillary pouch, showed the greatest correlation with clinical stage of adhesive capsulitis. Earlier, more hypervascular stages exhibited greater combined synovial and capsular thickening, while later more fibrotic stages demonstrated only capsular thickening. Hyperintensity of capsular signal was most closely associated with stage 2 disease. Rotator interval scarring was a nonspecific sign of adhesive capsulitis and was not found to correlate with clinical stage.

Treatment: Indications and Contraindications

Treatment for adhesive capsulitis should be addressed to the underlying pathology. The choice of nonoperative or operative treatment depends on recognition of the clinical stage at presentation because the condition progresses through a predictable sequence. Neviaser and Hannafin analyzed the current treatments for adhesive capsulitis [55].

Nonoperative treatment is represented by pharmacological treatment of the synovitis and inflammatory mediators and physical modalities to prevent or modify capsular contracture. With surgery it is possible to treat both the inflammatory component via synovectomy and the capsular contracture through capsular release and/or manipulation under anesthesia.

Nonoperative Treatments

Pharmacological Treatment

For treating inflammation, pharmacology gives us two weapons: nonsteroidal anti-inflammatory drugs (NSAIDs) and steroidal anti-inflammatory drugs (SAIDs).

About NSAID's use in adhesive capsulitis, despite their widespread use, in literature, there are few works. Although of theoretical benefit, NSAID treatment has yet to be proven effective as an isolated measure considering that there is not any level I or II placebo-controlled studies or comparisons between NSAIDs alone and an untreated group.

The treatment with oral SAIDs appears to provide rapid relief of pain compared with controls, but this sustained benefit is not confirmed at longer follow-up. About the use of oral steroid treatment for adhesive capsulitis in literature, there are no studies of sufficient duration useful for reporting long-term complications such as avascular necrosis of the femoral head, but the well-known side effects remain a theoretical concern.

SAIDs can be used also via intra-articular injections. Their efficacy has been extensively studied. In literature, there are many level I studies on this subject.

Physical Therapy

Physical therapy is the most prescribed treatment for adhesive capsulitis. Its goals are to prevent capsular contracture and to improve motion in the latter stages of disease. However, there are few supporting evidences that provide its benefit.

Despite the lack of high-grade evidence clearly supporting the use of physical therapy, many lower-level studies report its benefit, and its use in the treatment of adhesive capsulitis is almost universal [56]. Gentle stretching and active motion within the pain-free range appear to be sufficient, and the treatment need not be unduly painful.

Operative Treatments

Many studies in literature reported an approximately 10 % rate of patients do not respond to the variety of nonoperative treatments. Levine et al. [57] identified those patients who failed to improve or were worse after 4 months as most likely to fail nonoperative treatment. However, the indications for more invasive options remain highly subjective and need to be individualized to each patient.

Manipulation under anesthesia (MUA), hydrodilation, suprascapular nerve block (SSNB), and arthroscopic or open capsular release have all been described as possible options when physical and pharmacological therapies have failed.

Suprascapular Nerve Block

This procedure has traditionally been done by anesthesiologists in hospital pain clinics, but new techniques permit this procedure to be done in the office setting [58].

The rationale of its use in adhesive capsulitis is that temporary disruption of efferent and afferent pain signaling may allow "normalization" of the pathological, neurological processes perpetuating pain and disability. Pain relief may then translate into better shoulder function.

Hydrodilation

Hydrodilation, or "brisement," has been used as an alternative to operative procedures. This involves increasing intracapsular pressure and expanding capsular volume through injection of fluid until capsular rupture. It can be done under local anesthesia and takes only 15 min to complete. Various liquids have been used, and the procedure can be done in conjunction with arthrography. In the past, results have been variable and confounded by the use of dilation in combination with other treatments such as manipulation.

Manipulation Under Anesthesia

Closed MUA has been used extensively with consistently satisfactory results in both short- and long-term follow-up. It should be performed stabilizing the scapula with

one hand, while the other hand is used to grasp the affected arm above the elbow. The manipulation progresses through a smooth sequence of movements. The arm is first externally rotated then brought into full abduction above the patient's head. It is then lowered to 90° of abduction and internally rotated. Complications of this technique have been reported including humeral fracture, subscapularis rupture, labral tears, and injury to the biceps tendon. These complications are minimized with proper technique [59].

Arthroscopic Release

With the advent of arthroscopy, surgeons have chosen to address this condition arthroscopically in addition to or instead of closed manipulation. Arthroscopic release has become the most popular method of treating refractory adhesive capsulitis and has supplanted MUA as the treatment of choice.

Patients appear to achieve more significant and rapid improvements in motion and pain than the less precise manipulation and do not face the same risk of complications. Furthermore, arthroscopic procedure permits to confirm diagnosis and staging of disease. If necessary, a therapeutic synovectomy can be performed, and potential secondary causes of symptoms can be recognized.

Open Release

Nowadays, the indications for an open capsular release are very few, and this procedure is rarely performed. This approach carries the morbidity of an open procedure including prolonged recovery, postsurgical stiffness, and restricted postoperative therapy. Patients who have failed arthroscopic and closed manipulation should be candidates for open intervention.

Decision-Making Algorithm

Treatment should be stage based; the progressive pathological changes reflected in clinical stages should be treated.

Patients presenting in the painful stages (pre-adhesive stage or freezing stage) are treated with an intra-articular injection of 40 mg of methylprednisolone acetate mixed with local anesthetic to disrupt the inflammatory process and to discriminate between stage 1 and 2 diseases [60]. In addition, oral NSAIDs are routinely used in patients at all stages of disease for the analgesic effect and to facilitate both physical therapy and sleep. We do not use oral steroids because the same benefit can be achieved with local injection without systemic side effects.

Physical therapy remains the mainstay of treatment despite the lack of high-grade evidence. For patients in stage 1, the goals of therapy are to interrupt the inflammation and diminish pain [40]. Education, activity modification, and gentle range of motion exercises are prescribed. As pain can alter glenohumeral mechanics, therapy should focus on reestablishing proper scapulohumeral rhythm. Exercises such as closed kinetic chain scapular stabilization, joint mobilization, continuous passive movement, hydrotherapy, and a home exercise program are included. Home therapy is based on passive range of motion and pendulum exercises within the pain-free zone [61].

Patients in stage 2 should also minimize capsular adhesions and restrictions of motion in addition to reducing pain and inflammation. Passive joint glides are used to increase capsular mobility [62]. Home exercises are expanded to include cane exercises focusing on internal and external rotation range. Active exercises in the plane of the scapula are added to range of motion protocols aimed at preserving motion.

In the later stages (3 and 4), we do not use corticosteroid injection because the inflammatory phase of the disease has passed [60]. The objective of therapy in stage 3 is treatment of the marked loss of motion and correction of scapulohumeral mechanics. We recommend thus aggressive stretching for this stage after an intensive active warm-up to enhance soft tissue circulation. Heat can be used to promote relaxation of the surrounding musculature. Prolonged, low-load stretching is more effective than brief, high-load stretching [63].

The limits of motion can be pushed, but the patient should not have significant pain. Strengthening of the scapular muscles continues, and strengthening of the rotator cuff muscles can be initiated if range of motion permits. Specific therapy for stage 4 does not greatly differ from stage 3. Further cuff strengthening including conditioning is initiated as motion improves.

We do not have experience with hydrodilation or SSNB, and even if literature reports excellent results with this treatment options, we treat those patients who have failed nonoperative treatment with arthroscopic capsular release. The indications for surgical intervention are patient specific. We agree with Levine et al. that patients who are regressing despite appropriate therapy are candidate to surgical intervention [57].

Generally, we prefer to wait a minimum of 6 months from the onset of symptoms before offering a surgical option to the patient. The decision of surgery treatment is made by the patient after a clear discussion of his prognosis, disability, and desired activity level.

We prefer the arthroscopic release of the capsule to traditional MUA because it allows synovectomy if there is significant synovitis (Fig. 29.2) and a more precise release, including the posterior capsule, because loss of internal rotation is typically significant and the pathological process involves the entire capsule [64].

Arthroscopic Treatment: Surgical Technique

The procedure is performed under regional anesthesia. Before surgery, we evaluated passive range of motion in supine position and under anesthesia. The patient can be

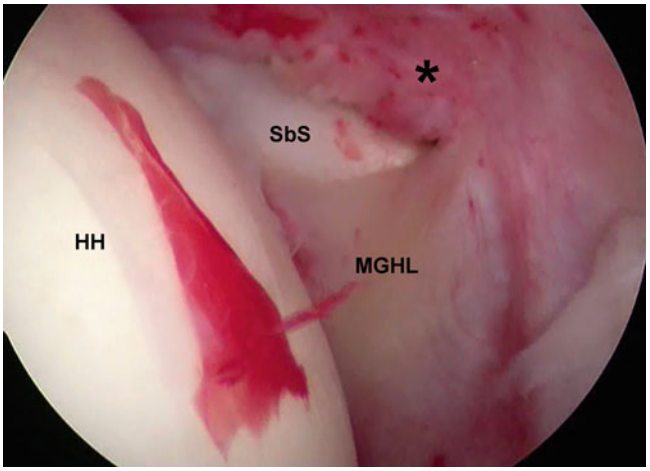

Fig. 29.2 Arthroscopic view of adhesive capsulitis (left shoulder; beach-chair position). An intense synovitis (*asterisk*) is present in the anterior-superior quadrant of the joint (*HH* humeral head, *SbS* subscapularis tendon, *MGHL* middle glenohumeral ligament)

positioned in the beach-chair or lateral decubitus position, depending on the surgeon's preference. The irrigation pump pressure is kept between 45 and 50 mmHg.

A posterior viewing portal is established. Diagnostic arthroscopy from this portal allows to recognize the areas of synovitis, capsular retraction or hypertrophy, and associated pathologies, such as rotator cuff tears (Fig. 29.3). An anterior interval portal is then established, just above the superior margin of subscapularis, and partial synovectomy of the long head of biceps, rotator cuff at its articular side and anterior-superior capsule is performed (Fig. 29.4). A basket meniscus cutter or radiofrequency electrocautery is used to release the anterior-superior capsule, the superior glenohumeral ligament, and the coracohumeral ligament at its coracoid insertion. The release is begun at the 1-o'clock position by placing the instrument medial to the superior labrum and directing it inferiorly (Fig. 29.5). Hypertrophic tissue between the subscapularis tendon and the anterior-superior capsule obliterating the subscapularis recess is excised using a motorized shaver. Middle glenohumeral ligament and anterior capsule are released 1 cm from the glenoid rim to reduce the risk of iatrogenic instability (Fig. 29.6). At this point, the range of motion is evaluated. If external rotation deficit in abduction is still present, we perform release of the anterior-inferior glenohumeral ligament and anterior-inferior capsule (Fig. 29.7). As the capsule is progressively divided, there is an increase in the available intra-articular space and ease of accessing the remaining capsule. From the anterior portal, release can be completed until the 6-o'clock position. Release at about the 6-o'clock position should be performed using a manual instrument (meniscus cutter) next to glenoid labrum to reduce the risk of axillary nerve injuries (Fig. 29.8). The joint is then viewed from the anterior portal, and the basket meniscus cutter or electrocautery is placed posteriorly. The release is again begun superiorly at

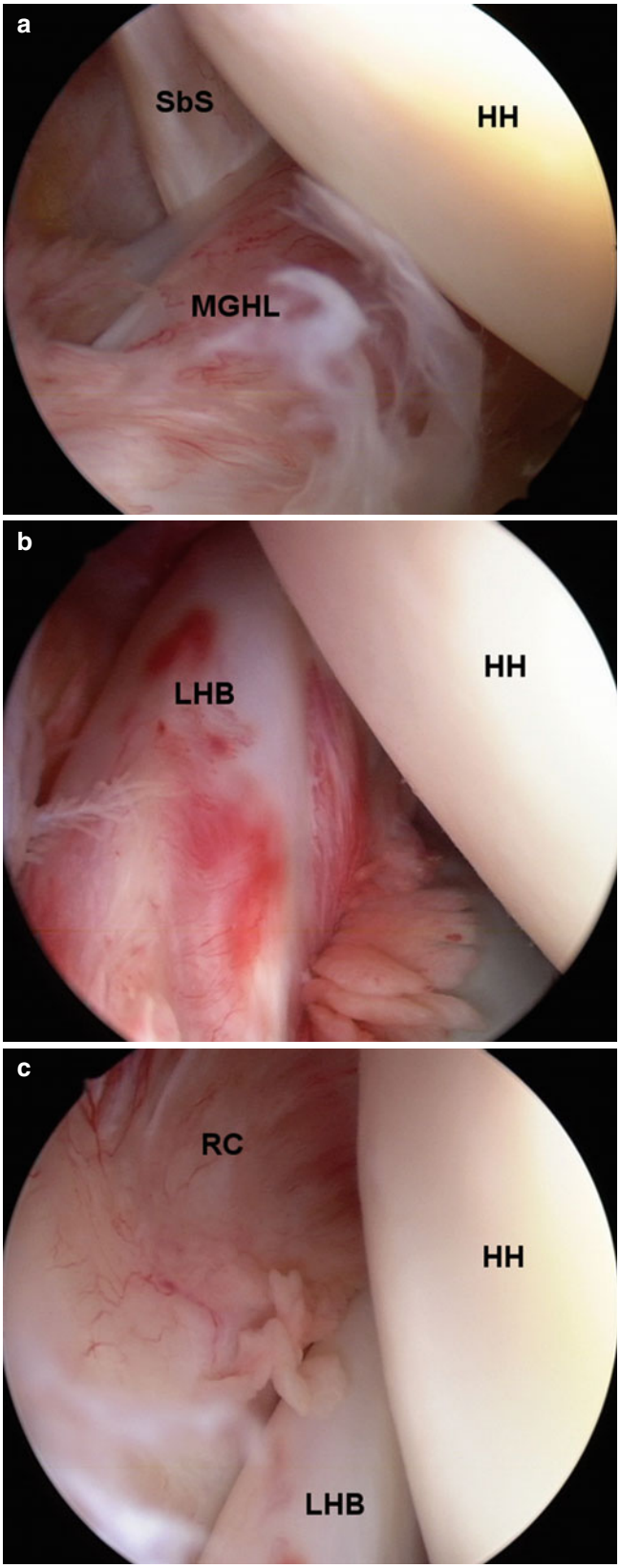

Fig. 29.3 Arthroscopic view of adhesive capsulitis (right shoulder; lateral decubitus position). (**a**) The middle glenohumeral ligament (*MGHL*) is thickened (*HH* humeral head, *SbS* subscapularis tendon). (**b**) The long head of the biceps (*LHB*) is hyperemic. (**c**) A layer of hypertrophic and hyperemic synovial tissue covers the articular side of the rotator cuff (*RC*)

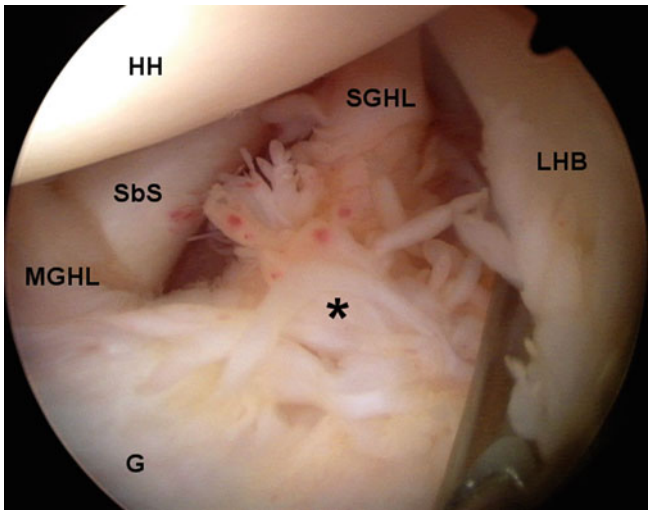

Fig. 29.4 Arthroscopic view of adhesive capsulitis (left shoulder; lateral decubitus position). The superior glenohumeral ligament (*SGHL*) has a cord-like appearance. A motorized full-radius shaver is used through the anterior interval portal to remove hypertrophic synovial tissue (*asterisk*) from the anterior-superior quadrant of the joint (*HH* humeral head, *SbS* subscapularis tendon, *MGHL* middle glenohumeral ligament, *LHB* long head of the biceps, *G* glenoid)

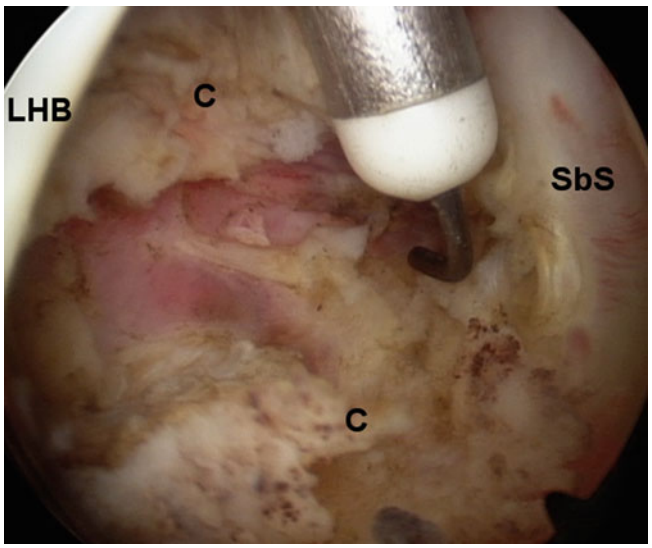

Fig. 29.5 Arthroscopic view of adhesive capsulitis (right shoulder; lateral decubitus position). A radio-frequency electrocautery is used to release the anterior-superior capsule (*C*) (*SbS* subscapularis tendon, *LHB* long head of the biceps)

11-o'clock and progresses posteriorly and inferiorly to the 7-o'clock position (Fig. 29.9).

The first time is recommended to maintain the inferior capsule intact to protect the axillary nerve. A manipulation maneuver is completed at the end of the procedure to assure adequate release. We do not perform the MUA before arthroscopy because it leads to bleeding within the joint and impairs visualization. Patients are placed in a sling postoperatively.

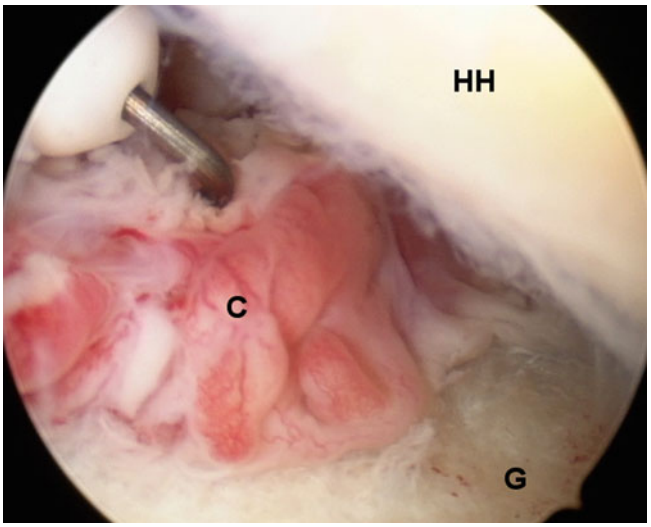

Fig. 29.6 Arthroscopic view of adhesive capsulitis (right shoulder; lateral decubitus position). Anterior capsule (*C*) is released 1 cm from the glenoid rim (*HH* humeral head, *G* glenoid)

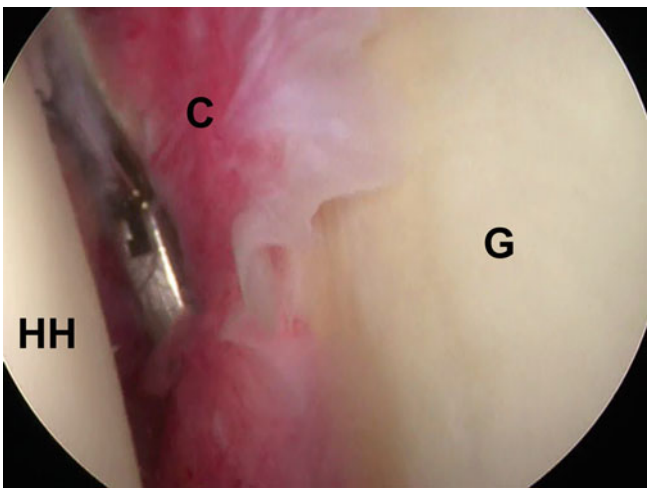

Fig. 29.7 Arthroscopic view of adhesive capsulitis (left shoulder; beach-chair position). The release of the anterior-inferior glenohumeral ligament and anterior-inferior capsule (*C*) is performed with a basket meniscus cutter (*HH* humeral head, *G* glenoid)

Postoperative Care

All patients begin physical therapy the first day after surgery. We have found it beneficial to demonstrate the gains in motion to patients in the recovery room by fully abducting the arm overhead, while they are still under regional anesthetic block but no longer sedated. This allows the patient to recognize that the mechanical block to motion is gone and allays fears about quickly beginning rehabilitation.

Outpatient therapy is begun with a protocol similar to that described for stage 2 treatment. Progression is similar to the preoperative protocol described above but is more rapid and

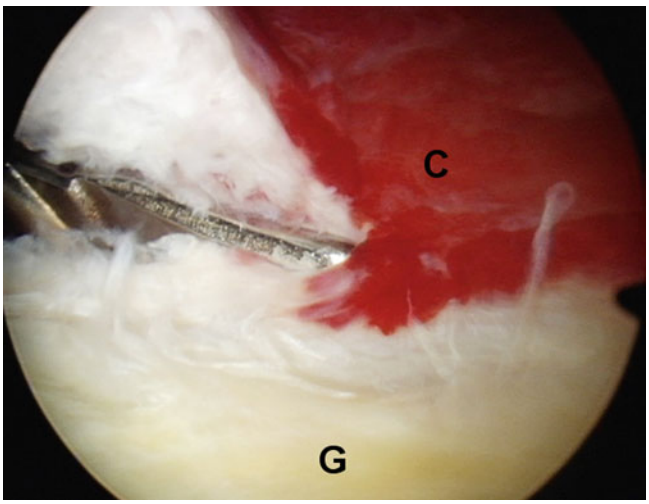

Fig. 29.8 Arthroscopic view of adhesive capsulitis (right shoulder; lateral decubitus position). The release of the inferior capsule (*C*) at about the 6-o'clock position is performed next to glenoid labrum with a basket meniscus cutter to reduce the risk of axillary nerve injuries (*G* glenoid)

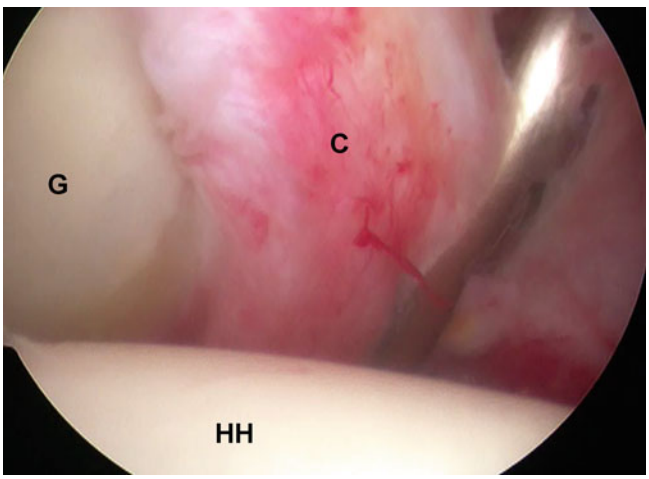

Fig. 29.9 Arthroscopic view of adhesive capsulitis (left shoulder; beach-chair position). The scope is in the anterior interval portal. The basket meniscus cutter is placed posteriorly. The release of the posterior capsule (*C*) is begun superiorly (at 11-o'clock or 1-o'clock position for the right of left shoulder, respectively) and progresses posteriorly and inferiorly (to the 7-o'clock position or 5-o'clock position for the right or left shoulder, respectively) (*HH* humeral head, *G* glenoid)

based on the resolution of postoperative pain and return of rotator cuff function.

Literature Review

Pharmacological Treatment

Comparisons of different NSAIDs to one another have been published. Some level I and level II randomized studies reported that using either naproxen or indomethacin for 4 weeks improves pain symptoms from baseline, but no significant change in objectively assessed motion was found for either treatment [65, 66]. The efficacy of COX-II inhibitors and other oral NSAIDs has not been evaluated.

Two level I studies compared oral steroid treatment with placebo [67, 68]. Blockey et al. [67] in a double-blind randomized controlled trial, in which a cortisone acetate suspension given in a tapered fashion over 4 weeks' time to a similarly administered placebo, noted that all treated patients improved "vigorous" shoulder exercise after only 1 week of treatment. Buchbinder et al. [68] compared a 3-week course of oral prednisolone and placebo. The treatment group showed statistically less pain and improved function at the end of 3 weeks. Examination at 6 and 12 weeks, however, showed no significant difference between the groups. The authors attributed this to a rebound effect in the treatment group occurring after the course of steroid was concluded.

Binder et al. [69] in a level II study compared the treatment with oral prednisolone for 6 weeks or no treatment. All patients performed pendulum exercises three times per day. A more rapid improvement in pain symptoms was seen in the treatment group at 5 months but was not maintained through the 8-month follow-up.

Intra-articular Steroid Injections

Rizk et al. [70] compared intra-articular methylprednisolone and lidocaine to an intra-articular lidocaine placebo and two control groups who received the same injections into the subacromial bursa.

Blinded assessments of pain and range of motion revealed no significant difference in shoulder range of motion between groups. Those treated with the intra-articular steroid showed a more rapid improvement of symptoms, but this difference was no longer than 2–3 weeks.

Bulgen et al. [71] in randomized study compared 4 treatment options: intra-articular injection of methylprednisolone, mobilization with a physiotherapist, ice treatments following proprioceptive exercises, and no treatment. The authors noted that all patients reported improved pain at 6 months. Those treated with steroid injections had a better improvement in range of motion at 4 weeks. At 6 months, however, there was no difference between the groups.

Van der Windt et al. [72] randomized a cohort of 109 patients to receive either 40-mg intra-articular injections of triamcinolone acetonide or physiotherapy two times per week for 6 weeks. The authors reported a 77 % rate of success for patients treated with injection compared with 46 % of those treated with physiotherapy. Success was defined as patients who rated themselves having had a full recovery or much improvement based on pain and functional scales. This

difference was statistically significant and persisted until the final assessment at 1 year.

Ryans et al. [73] in their level I study confirmed these findings of more rapid improvement in patients treated with intra-articular triamcinolone injection as compared to controls, which dissipates after longer follow-up beyond 6 weeks.

Hazelman [74] reviewed 130 patients with adhesive capsulitis treated with intra-articular injections of hydrocortisone. He noted that the efficacy of intra-articular treatment inversely correlates with the duration of symptoms. This may reflect a greater efficacy in the early, inflammatory stages of the disease.

Physical Therapy

A Cochrane database review of physiotherapy for painful conditions of the shoulder concluded that deficiencies in the literature resulted in little overall evidence to guide treatment and found no evidence that physiotherapy alone is of benefit in adhesive capsulitis [56].

In a level I study, Vermeulen et al. [61] evaluated the rehabilitation intensity issue and found that there was little difference between those who engaged in low-grade mobilization techniques compared with groups using high-grade techniques. Low grade is defined as movement within a pain-free zone, while high-grade mobilization included movements into the stiff, painful range.

In a level IV investigation, Griggs et al. [75] prospectively evaluated 75 patients with stage 2 disease treated with a specific four-direction stretching program and found 90 % achieved a satisfactory outcome. Stretching was limited to the range of tolerable discomfort.

Suprascapular Nerve Block

Dahan et al. [76] in a double-blind randomized trial comparing 17 patients treated with a series of three bupivacaine SSNBs with 17 patients treated with placebo injections noted that the treatment group showed a significant 62 % improvement in overall pain compared with 13 % improvement in controls. There was no difference in shoulder function between the two groups. Although this amount of pain relief is impressive, the value of this study is severely limited because the outcome was measured at 1 month only.

In a prospective randomized comparison of intra-articular triamcinolone acetonide (20-mg) injection to SSNB (9.5-mL 0.5 % bupivacaine and 20-mg triamcinolone) by Jones and Chattopadhyay [77], the nerve block cohort showed a significantly greater reduction in pain and improved shoulder range of motion at 3 months' follow-up. Pain relief from the SSNB was greater as early as 1 week from the injection. Sleep disturbance showed a trend toward greater improvement, but the difference did not reach statistical significance. This is likely because of the small number of patients enrolled in the study, which was 15 per group. These investigations suggest promise for SSNB; however, the exact therapeutic mechanism remains unclear. Larger studies with longer follow-up are needed to establish the role for SSNB in treating adhesive capsulitis.

Hydrodilation

Quraishi et al. [78] performed a level II randomized controlled trial on 36 patients (38 shoulders) to compare hydrodilation with normal saline and MUA. All patients were described as stage 2. Those who underwent MUA also received a 30-mg intra-articular injection of triamcinolone. Despite small numbers, the authors demonstrated an improvement in Constant score that was statistically greater in those receiving hydrodilation than in the MUA group. Visual analog pain scores were also statistically better in the hydrodilation group. Both differences persisted until the study concluded after 6 months. No differences in range of motion were found between the groups. Although this investigation suggests benefit, experience with this technique is limited, and more investigation is required to determine its ultimate role in the treatment of adhesive capsulitis. A Cochrane database review was unable to draw firm conclusions about the efficacy of this technique because of the small number of trials with few patients and different comparison interventions [79].

Manipulation Under Anesthesia

High-level studies are few. Kivimaki et al. [80] performed a level I study to compare MUA with a home-based exercise program. One hundred and twenty-five patients were randomized to undergo home exercise or home exercise and MUA. The manipulation group had slightly better mobility at 3-month follow-up examinations with statistically significant improvement in shoulder flexion, but this was not sustained at 6 and 12 months. For refractory loss of motion, MUA appears to be a reliable treatment.

Arthroscopic Release

The earliest description of arthroscopic resection of the shoulder capsule is that of Conti in 1979 [81].

Several level IV studies have reported on the benefits of using arthroscopy to address capsular contracture. Pollock et al. [82] reported 83 % excellent or satisfactory results when arthroscopic debridement of the rotator interval was done in conjunction with MUA.

Warner et al. [83] were able to achieve significant improvement of motion in all planes in patients treated with arthroscopic anterior release who had previously failed MUA.

Ogilvie-Harris et al. [84] in a level III comparison of MUA and arthroscopic release of the joint capsule reported that patients who were treated via arthroscopy were twice as likely to be pain-free at 2 years' follow-up.

Although arthroscopic procedure includes a release of the superior glenohumeral ligament and rotator interval, for some authors it also includes the release of the intra-articular portion of the subscapularis [IASS]. No significant deficits or instability have been reported with releasing this portion of the tendon. However, the need for release of the IASS has not been proven in comparison trials [85].

Even if there is not a consensus about how much of the capsule should be released remains a matter of debate, some authors have advocated performing a 360° release while maintaining the IASS.

Jerosch [86] described a release in which anterior, posterior, and inferior portions of the capsule up to the 5-o'clock position were sectioned with electrocautery. The most inferior part of the capsule is then cut with a small angled meniscus cutter to better protect the adjacent axillary nerve, producing a circumferential release. No axillary nerve injuries were reported in the initial series of 28 patients.

The posterior release is thought necessary to improve significant internal rotation deficits [87]. Snow et al. [88] performed a level III investigation to compare anterior release with anterior and posterior release and demonstrated that there is no improved range of motion with a more extensive release. Further investigation is needed to determine how much of the capsule requires release.

Open Release

Ozaki et al. [89] in a level IV study treated 17 patients who had failed nonoperative measures with an open excision of the rotator interval. Sixteen patients had complete pain relief and a return of motion equal to the other side after 3 months.

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Degenerative Acromioclavicular Joint Disease

30

Giovanni B. Vinanti, Daniele Scrimieri, and Andrea Grasso

Epidemiology

The prevalence of the acromioclavicular (AC) joint osteoarthritis (OA) among general population is very high [1, 2]. We can distinguish a primary and secondary form. The first is related to some risk factors, such as age, even though it has been widely demonstrated that the AC joint undergoes early degenerative changes after the third decade of life. Therefore, an initial OA of the AC joint can appear also in patients in their 40s and is part of an early degenerative ageing process and not necessarily painful [3–5] (Fig. 30.1). Other risk factors can be intense manual labor or intense sports activities.

The secondary form can be a consequence of osteolysis of the distal clavicle, systemic diseases (i.e., rheumatic pathologies), traumas, postural alterations, or overload by proximal migration of the humerus head due to a massive rotator cuff deficiency. Secondary OA of the AC joint has not age- and gender-specific prevalence.

Pathophysiology

Primary Osteoarthritis

The most frequent disorder of the AC joint is the degenerative OA. DePalma [6] first demonstrated that many people have degeneration changes of the AC joint with ageing. His work has highlighted a pattern of changes linked to age which are evident from the third–fourth decade of life and increase exponentially with time [6].

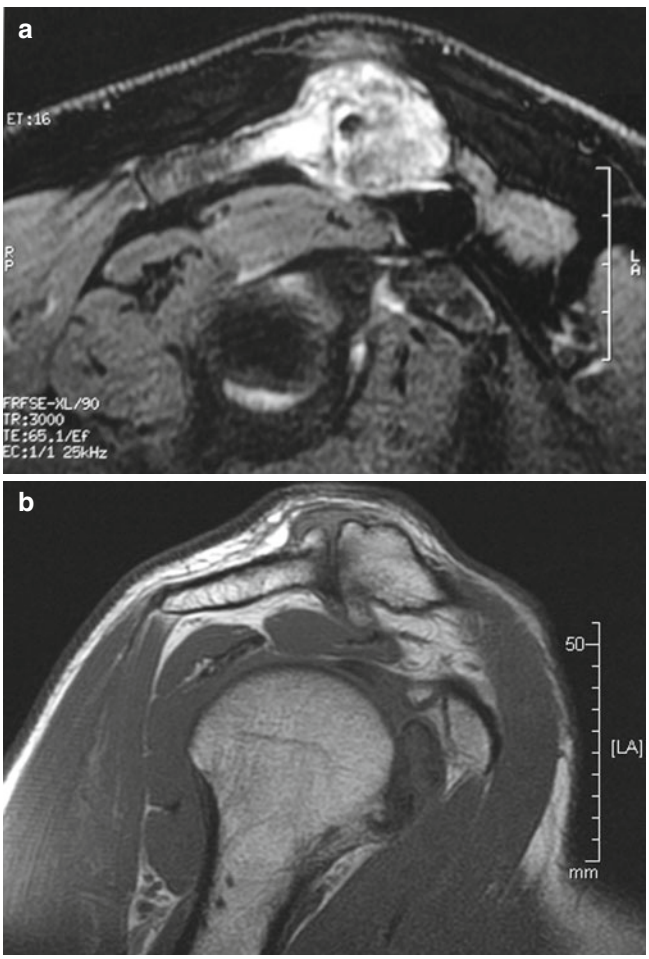

Fig. 30.1 (**a**) MRI showing severe hypertrophic tight OA of the AC joint with cancellous bone edema associated. (**b**) MRI showing severe hypertrophic OA of the AC joint with large clavicular osteophyte impinging the supraspinatus tendon

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Osteolysis of the Distal Clavicle

Osteolysis of the distal clavicle is characterized by synovial hyperplasia, destruction of the articular surface of the distal clavicle, bone necrosis, vascular proliferation, fibrous tissue invasion, and presence of inflammatory cells in the bone marrow. The intra-articular disk is usually intact, and the acromial surface of the joint is not involved [7, 8].

Various mechanisms have been suggested to explain the development of the osteolysis, such as vascular compromise, micro-fractures, and dystrophic sympathetic dystrophy [7, 9–12].

The relationship between osteolysis of the distal clavicle and traumatic events has been documented [7, 9, 13, 14]. Even the role of repeated microtraumas and overuse due to heavy manual labors has been proofed to be related with osteolysis of the distal clavicle [10, 15–18]. Osteolysis has also been reported following surgery to the AC joint [19]. Nevertheless, in some cases, it has not been possible to identify a cause [20].

Rheumatic Diseases

The AC joint is affected at least in 50 % of patients with rheumatoid arthritis [21]. Also cases of gout and pseudogout affecting the AC joint have been reported [22–25].

Posttraumatic Osteoarthritis

About 40 % of traumatic events to the shoulder produce an injury to the AC joint [26]. The majority of these injuries consist of minor bone contusions and capsuloligamentous strains and sprains. However, high-speed and high-energy traumas can induce severe injuries to the AC and coracoclavicular (CC) ligaments and to the trapezium and deltoid muscle insertions as well.

AC joint dislocations represent 12 % of the dislocations affecting the scapular girdle and 8 % of all the dislocations [27]. They mostly occur as a consequence of a direct trauma to the shoulder during sports activities, such as ski [28, 29] and rugby [30]. High-energy traumas (car or motorcycle accidents) can also damage structures close to the AC joint, generating ribs, scapula, or clavicle fractures, as well as sternoclavicular joint dislocation [31] or brachial plexus injuries [32, 33].

The most common mechanism of AC joint dislocation consist of a force directly applied above the acromion, as it occurs during a fall on adducted arm or by impact of an object against the shoulder. Rarely, a compressive load applied above the distal clavicle can cause its dislocation below the coracoid process [34, 35].

Indirect AC joint injuries are exceptionally rare. A fall on an adducted upper limb is an example, being able to push the humerus head against the undersurface of the acromion and causing injuries of varied entity to the AC joint [36].

Rockwood et al. [37] classified AC dislocations according to the extension and severity of the injuries to the ligaments and surrounding soft tissues: type I, incomplete injury of the AC ligaments and integrity of the CC ligaments; type II, complete injury of the AC ligaments and incomplete injury of the CC ligaments; type III, complete injury of the AC and CC ligaments; type IV, complete injury of the AC and CC ligaments with posterior dislocation of the distal which penetrates into the trapezium muscle; type V, superior dislocation of the clavicle with disruption of the trapezium and deltoid muscle insertions; and type VI, inferior dislocation of the distal clavicle below the coracoid process.

Although in type I and type II AC joint dislocations there is not a complete displacement of the distal clavicle and the most appropriate treatment is conservative [27, 38–44], it is possible that following even just a single trauma, degenerative changes of the AC joint or, more rarely, osteolysis of the distal clavicle can develop [40, 45, 46].

Postural Alterations

The chronic scapular protraction can lead towards a mechanic overload of the AC joint. Patients having this alteration of scapular posture present a prominence of the inferior angle of the scapula, which can be present also during the abduction of the upper limb [47]. Kibler described in details a painful syndrome caused by this alteration (see Chap. 19).

Bilateral anatomical alterations of the scapular posture can be related to hyperkyphosis of the thoracic spine or to neuromuscular paralysis. In the latter case, the pathology can be unilateral or bilateral.

Massive Inveterate Rotator Cuff Tears

A massive chronic rotator cuff tear leads to a progressive cranial migration of the humerus head, which creates an excessive burden on the AC joint and progressive degenerative changes. Eccentric glenohumeral arthropathy is frequently associated with a para-articular cyst at the AC joint [48–54]. This cyst is usually created by a subsidence of the superior capsule. This is the most vulnerable region of the AC joint capsule, because anterior and the posterior capsule are reinforced by the insertions of the deltoid and trapezium muscles, respectively [55] (Fig. 30.2).

Other Causes

Benign or malignant tumors as well as synovial proliferative diseases very rarely affect the AC joint.

AC joint infections are not common also in patients in compromised general conditions [56]. Many AC joint infections are caused by surgeries or infiltrations made for articular disorders. Rarely is the infecting organism bloodborne or acquired directly from infected tissue in the vicinity of the joint. Furthermore, tuberculous localizations and cryptococcus and candida infections have been noticed [57–62].

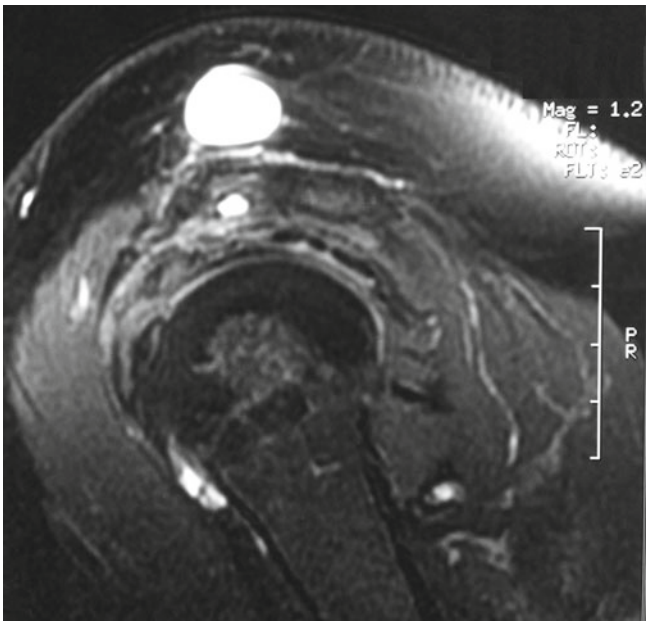

Fig. 30.2 MRI showing large para-articular superior cyst of the AC joint

History

Pain is the most common symptom in AC joint disorders. Almost all patients—when invited to describe the source of pain—indicate the AC joint [63]. Such pain tends to irradiate towards the neck and occasionally towards the chest. Indeed, very often a painful pathology of the AC joint requires a differential diagnosis with cervical, otorhinolaryngological, or odontological pathologies.

Physical Examination

With the patient sitting or standing, the examiner must perform a thorough inspection checking first the scapular posture, since a chronic scapular protraction leads irremediably to an overload on the AC joint encouraging therefore early degenerative phenomena. It is important to identify the osseous profile and search for possible asymmetries.

A unilateral protrusion at the level of the AC joint could indicate a previous trauma with clavicular subluxation, an acute inflammatory process, or a para-articular cyst (Fig. 30.3).

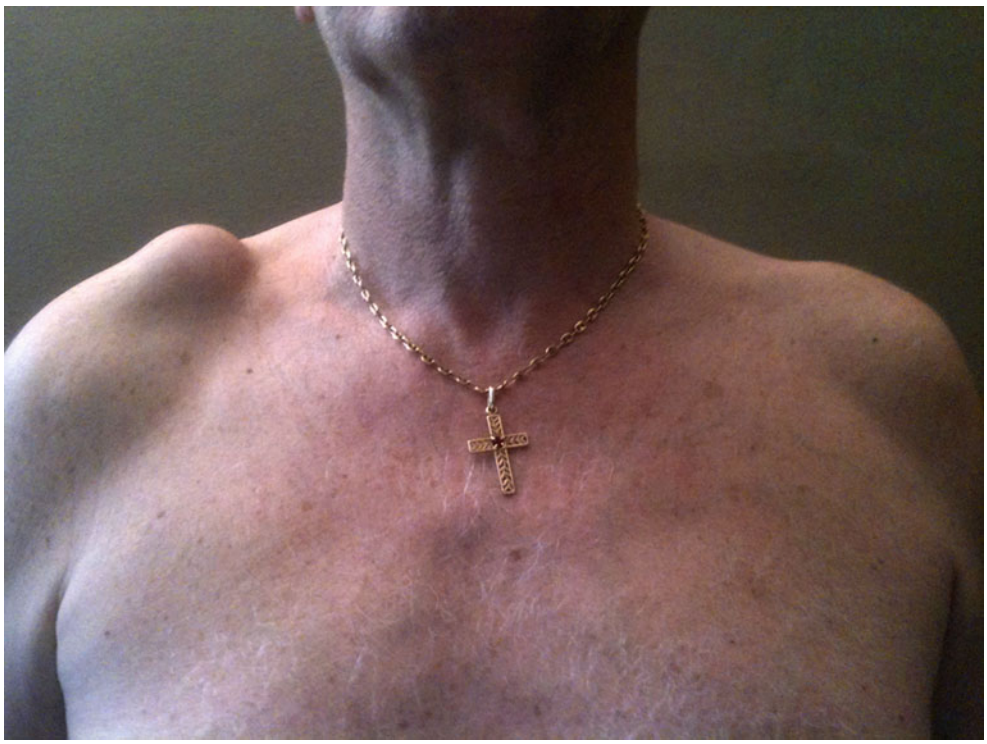

Fig. 30.3 Clinical appearance of large para-articular superior cyst of the AC joint with considerable shoulder asymmetry

Both shoulders must be examined in their active and passive mobility. Usually, a degenerative process of the AC joint does not determine shoulder stiffness, neither serious antalgic functional deficits. The movement which sometimes can be partially limited because of pain is flexion-adduction-internal rotation of the arm (such as washing the opposite shoulder or taking a wallet from the trouser pocket).

Sensitivity and strength of the upper limbs must be checked; this is particularly useful in particular in the differential diagnosis with a cervical radiculopathy.

Asymmetric tenderness at the level of the AC joint indicates the source of symptoms, especially if the pain produced is similar to the patient's experience. Sometimes it is very difficult to identify and palpate with certainty the AC joint in obese or particularly muscular patients. In these cases, it is useful to refer to the triangle created by the clavicle, scapular spine, and the neck base: AC joint is directly anterior to the examiner's finger placed medial to the acromion process, at the lateral apex of the triangle.

Several tests to exacerbate pain in a degenerative AC joint disease have been described, but none of them can be considered specific to this pathology:

- AC pain test: The test is performed by applying a digital pressure in correspondence of the joint, and it is positive if it causes a sharp pain.
- Cross-arm test: The patient's arm is held in forward flexion to 90°, and the examiner adducts the arm across the body towards the opposite shoulder; it can be considered positive if it provokes pain [64].
- O'Brien test: The shoulder is forward flexed to 90° and adducted of 10° towards the midline; the patient resists a downward force first with the arm in internal rotation and then in external rotation. The test is considered positive if pain is produced in correspondence of the AC joint only in the internal-rotation maneuver [65, 66].

Sometimes the suppression test with local anesthetic can be useful, as it eliminates the AC pain in a replicable way and helps in making differential diagnosis [67].

Imaging

The standard radiograph scan is diagnostic to confirm of the clinical suspicion. An anteroposterior view in the plain of the thorax, a detail of the AC joint with cephalic tilt of 10° (Zanca view), and an axillary lateral view are recommended.

Nevertheless, the authors prefer magnetic resonance imaging (MRI) because it allows an analysis of the AC joint at different levels, it enables to identify a possible edema of the cancellous bone of the distal clavicle (which could contraindicate a surgical treatment), and finally because it allows a thorough analysis of the shoulder ascertaining the presence of concomitant pathologies, such as a rotator cuff tear. In some occasions, it might be necessary to integrate the investigation with a CT scan or a bone scan.

Conventional Radiology

Anteroposterior View at Reduced Voltage

It is necessary to ask the radiologist for an image of the AC joint and not of the shoulder because in the last case we would obtain overexposed dark radiograms of the AC joint which could hide traumatic or degenerative changes. The joint can be easily visualized using 50 % of the voltage normally used for the glenohumeral joint exposition.

Zanca View

Sometimes, fractures of the acromion or the distal clavicle, osteolysis, or AC joint OA is not noticeable on standard views because the distal clavicle is obscured by the overlapped shadow of the scapula spine. To obtain a clear vision, Zanca [68] recommended a cephalic tilt of 10° in the beam of radiation.

Axillary Lateral View

With the limb abducted 70–90°, the cassette must be positioned above the shoulder and the x-ray tube inferior to the axilla. This projection can reveal small intra-articular fractures not visible in the anteroposterior radiographs, leading to an incorrect diagnosis and prognosis. Furthermore, this projection shows an anterior or posterior dislocation of the clavicle and the degree of displacement of fractures to the distal end of the clavicle.

Alexander View

Alexander [69, 70] has presented a modification of the true scapulolateral view which can be useful in evaluating injuries to the AC joint. Either standing or sitting, the patient is invited to bring the shoulders forward while a radiograph in true scapulolateral view is taken. If there are not any injuries to the AC ligaments, a dislocation or an overlap of the clavicle and acromion lateral portion will not be noticed.

Computed Tomography

Occasionally, none of the routine radiographic images are able to delineate clearly the extension of a pathology of the distal clavicle or of the AC joint, and it might be necessary to use CT scan.

The main objective of the CT scan is to obtain a more accurate picture of the morphology of the distal clavicle, of the coracoid process, and of their reciprocal anatomic relationships. This can help in the analysis of complex injuries,

such as fractures, in tumors or infections, especially when images are elaborated in three dimensions (3D-CT).

Bone Scan

When a slight shoulder pain is not accompanied by other signs and symptoms which facilitate diagnosis, the bone scan can be helpful to highlight early stages of a degenerative OA, infections or traumatic osteolysis of the distal end of the clavicle before changes become visible to the conventional radiographies. The scanning is positive when an increased captation in radioactive tracer is highlighted [71]. Walton et al. [67] found that the bone scan has a higher accuracy than MRI and conventional radiology in determining anomalies of the AC joint.

Magnetic Resonance Imaging

The MRI allows a complete and accurate analysis of the AC joint and allows the identification of subtle degenerative injuries. As already described, AC joint degeneration is very frequent already after the third or fourth decade of life but is not always symptomatic. Therefore, any instrumental image at the level of the AC joint must strictly relate to the clinical presentation [72].

Differently from the other imaging methods, MRI can easily reveal cancellous bone edema of the distal clavicle or more rarely of the acromion which produces an increase in T2 signal [73] (Fig. 30.4). Such alteration in signal is quite frequent and has not always clinical relevance [74, 75].

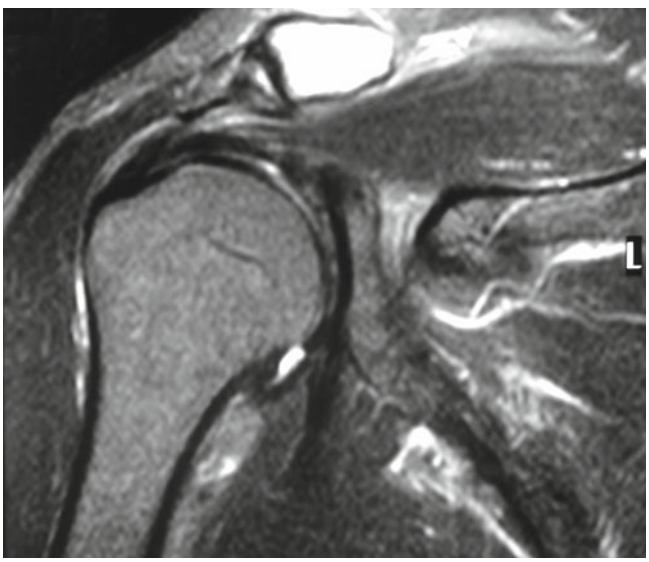

Fig. 30.4 MRI of the distal end of the clavicle with severe cancellous bone edema and OA of the AC joint

Treatment: Indications and Contraindications

Conservative Treatment

Conservative treatment should be preferable as first-line option in all pathological conditions affecting the AC joints. This approach allows to clarify the differential diagnosis according to the results obtained and to avoid unnecessary surgical treatment.

The treatment of degenerative AC joint disease aims to reduce pain and to resolve possible causes. In particular, the conservative treatment includes, in the first instance, functional rest to reduce joint loads, nonsteroidal inflammatory drugs (NSAIDs), and rehabilitation.

When identified, treatment has to act specifically on the cause, such as medications for rheumatic diseases or correction of postural problem.

The conservative treatment may involve also local corticosteroid injections that the authors prefer not to perform intra-articularly because this maneuver might be quite invasive, especially in taut joints due to OA. The authors prefer to infiltrate 40 mg of methylprednisolone using two accesses, superior and subacromial, that allow to inject the drug in proximity of the superior and inferior joint capsule.

Surgical Treatment

It is necessary to select the patient who is eligible to the surgical treatment for degenerative AC joint disease, mainly when the conservative treatment has failed and the surgeon is sure that the referred symptoms are related to the AC joint.

The main purpose of the surgical treatment is to perform an articular resection by removing the intra-articular soft tissues and the most distal part of the clavicle. This procedure prevents further contact between acromial and clavicular surfaces under different loading conditions.

Another purpose of the surgical treatment is to preserve the articular stability without damaging the AC ligaments, because postoperative pain is often due to joint instability. Finally, it is important to preserve the clavicular insertion of the trapezium and deltoid muscles because their weakness may impair strength of the shoulder girdle. Cook and Tibone [76] found about 20 % reduction of strength in weight lifters who underwent an open excision of the distal clavicle.

When cancellous bone edema of the distal clavicle involves the distal third of the clavicle for the most part and the pain in such area is quite intense, the authors prefer deferring a possible surgical solution and treat the pathology with physical therapy, clodronic acid, and NSAIDs. We have noticed that bone suffering can compromise surgical results, perpetuating the pain also during the postoperative phase.

Open Treatment

Distal clavicle resection, as described by Mumford and Gurd separately [77, 78], seems a reliable treatment for patients with posttraumatic OA. The standard technique consists of incision above the AC joint, dissection of the deltoid and trapezium fascia and excision of the two distal centimeters of clavicle. Even if the results of this technique are satisfactory in several series, the open treatment is frequently associated to a high morbidity. The most common complications are the painful and non-cosmetic surgical scar, the residual AC joint instability and muscular weakness.

Arthroscopic Treatment

The improvements in shoulder arthroscopic surgery have made accessible the subacromial space and the AC joint. Nowadays, indications to arthroscopic distal clavicle excision are superimposable to those of open treatment and arthroscopy should be always preferable for its lower morbidity.

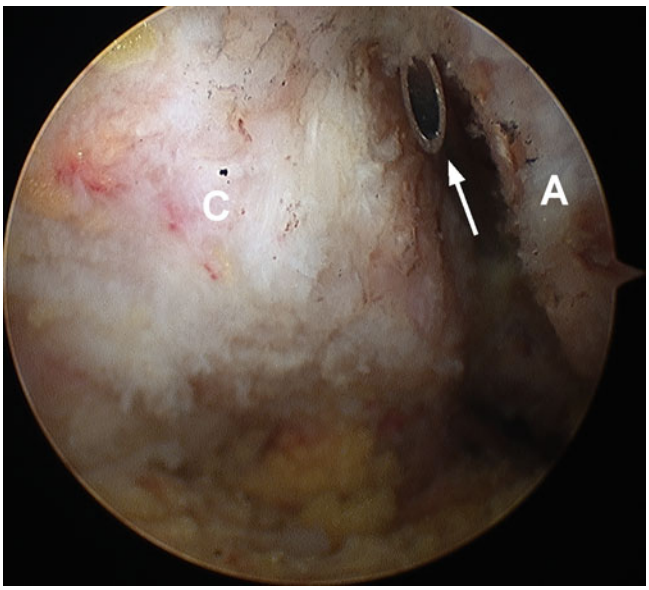

Fig. 30.5 Arthroscopic view of the AC joint (*right shoulder*) from the posterior portal. Note the spinal needle (*arrow*) used to establish the anterior portal (*C* clavicle, *A* acromion)

Arthroscopic Treatment: Surgical Technique

The approach to the AC joint can be direct or indirect, that is, through the subacromial space. It is clear that compared to the direct one, the indirect approach offers the advantage to examine the glenohumeral joint and the entire subacromial space. In addition, this approach does not weaken the superior capsular ligaments and it does not require a dedicated tool.

Portals

AC arthroscopy by an indirect approach requires a standard posterior portal to visualize the AC joint and an anterior and a lateral operative portals. The anterior portal is located at the anterior margin of the joint, so that the instruments may work easily along the articular axis. AC joint is correctly identified with a spinal needle (Fig. 30.5). The superior portal described in the literature is not necessary, and it is even not recommended by the authors because it might damage the superior capsular ligaments.

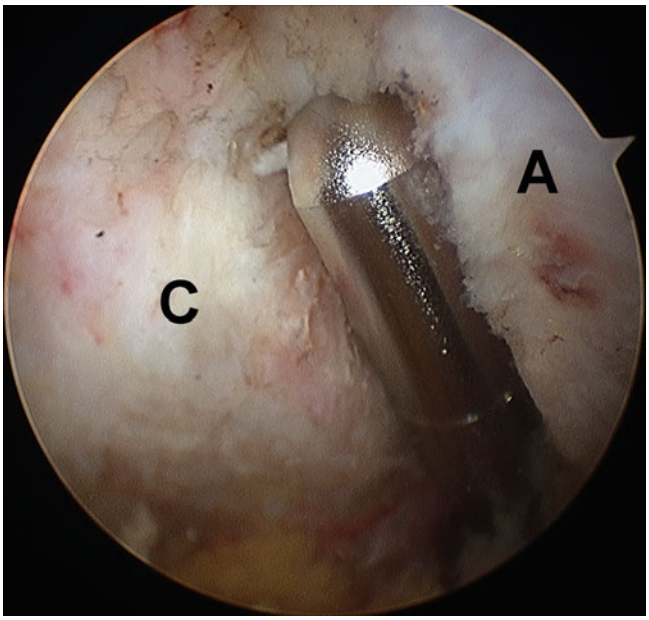

Fig. 30.6 Removal of articular and peri-articular soft tissues performed with a 5 mm full-radius shaver (*C* clavicle, *A* acromion)

Step-by-Step Procedure

The first step includes the accurate removal of bursal and scar tissues and the residual intra-articular disk by a motorized instrument and/or radiofrequencies (Fig. 30.6). This step can be adequately performed with the scope in the posterior portal and using the lateral portal to remove the extra-articular tissue and the anterior portal to remove the intra-articular tissue.

After obtaining a complete exposure of the undersurface of the acromion and of the distal clavicle, the second step includes partial to minimal removal of acromial surface from the lateral portal to make more visible the clavicular end.

The third step includes the removal of clavicular bone surface (Fig. 30.7). Through the anterior portal, proceed to create a line of about 1 cm in width and of 4 mm in depth on the lateral-inferior margin of the distal clavicle. This line represents a guide for the progressive final resection.

Regarding the amount of clavicle resection, data reported in the literature are not univocal. Some authors perform an

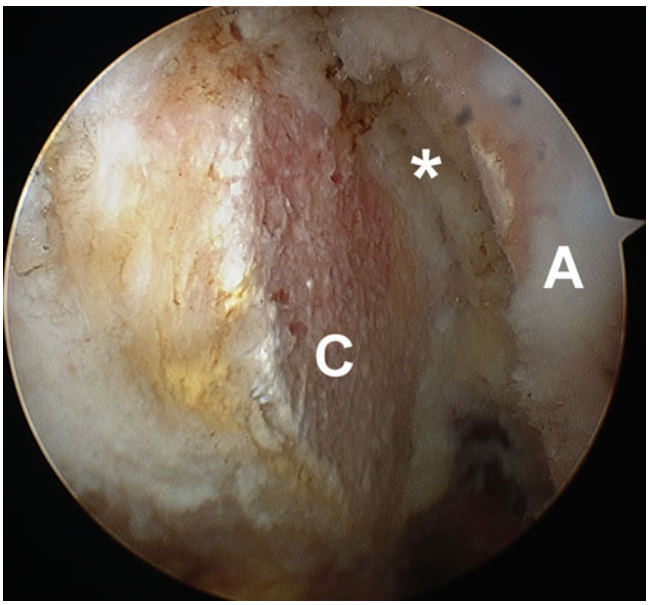

Fig. 30.7 Final inspection from the posterior portal. Note resection of the distal clavicle and integrity of the superior articular capsule (*asterisk*) (*C* clavicle, *A* acromion)

excision of more than 3 cm of bone (partially also from the acromion), while others less than one centimeter. The clinical results seem to prefer resections of less than 10 mm, and experimental studies have shown that even a 5-mm resection makes a contact between the joint surfaces mechanically impossible in axial compression.

Direct Approach

The direct approach proposed for the first time by Johnson and Flatow [79] allows to complete the procedure without violating the subacromial space. This approach is technically more difficult, especially in tight joints. It includes the beach chair position of the patient and two superior portals: one just behind to the AC joint and the other about 8 mm anterior to the AC joint.

The use of a spinal needle is crucial to determinate the joint orientation because the space is narrow, especially at the beginning. The procedure requires initially a 2.7 mm arthroscope and a 2 mm trimmer. When an adequate space is obtained, it is possible to use bigger size arthroscopy equipment to complete surgery.

Postoperative Care

After surgery, the arm is immobilized in a sling with 10–15° of abduction and neutral rotation for 3–4 weeks. The sling is removed during the day for the hygiene. We recommend to put a pillow under the shoulder or the arm while sleeping for more comfort. Hand-squeezing exercises should start immediately as well as the active motion of the elbow and wrist, with shoulder in neutral rotation at side. Ice therapy for 4–5 times a day for 15 min helps to relief pain and swelling. During the first week, the patient starts pendulum exercise. The goal of this first phase is to control pain.

Passive range-of-motion exercise are allowed 10 days after surgery, while active-assisted motion exercise can be done in supine position with a wand under 90° of forward flexion, 60° of abduction, and external rotation as tolerated. During this period, scapular control should be recovered with scapular retraction exercises without resistance, and pool therapy is strictly recommended. The goal of this second phase is to keep the shoulder pain free and to achieve 90° of forward flexion and 60 of abduction.

After the third week, or at the removal of the sling, the complete range of movement should be restored. Once this goal is achieved and the patient is able to control the scapula correctly, it is possible to start active exercises for the rotator cuff and the deltoid. Exercises should be performed until 90° of forward flexion and 60° of abduction between the fourth and the eighth week and up to 120° or more for both after the second month.

Return to normal activities of daily living under the 90° plane is allowed after the first month, mild sports activity (running) between second and third month, while 6 months or more should be waited to perform contact sports or heavy manual works.

Literature Review

Several studies showed that either the open or the arthroscopic techniques obtained excellent clinical results [80–86]. Both techniques can provide adequate amount of bone resection. Gartsman et al. [87] reported that bone resected was 14.7 and 14.8 mm, with the open and arthroscopic techniques, respectively. Fischer et al. [88] found that preserving the superior portion of the clavicle in order to not damage the superior capsular ligaments caused a constant deterioration of the initial good results, with up to 39 % of recurrent pain at 8.5 months after surgery. Extension of the bone resection to the anterior-inferior acromion, when indicated, might bring to excellent results. Levine et al. [89] demonstrated that the patients who underwent an arthroscopic resection of distal clavicle and acromioplasty obtained 71 % of excellent results, 16.5 % of good results, and 12.5 % of poor results.

Indeed, arthroscopic treatment preserves the superior capsular ligaments and reduces the risk of weakness of extrinsic ligaments [90]. Flatow et al. [79] demonstrated that a shorter coracoclavicular distance (acromioclavicular stability index) was obtained arthroscopically compared to the open technique.

Finally, regarding the preservation of clavicular insertions of trapezium and deltoid muscles, Cook and Tibone [76]

found a 20 % force reduction in body builders treated with open resection of distal end of clavicle.

Summary

The degenerative AC joint disease is one of the most frequent causes of shoulder pain. Etiology is relatively various and a thorough clinical examination and history taking are crucial to reveal the possible causes. Clinical presentation is usually sufficiently indicative but is often confused with symptoms caused by cervical pathology. Imaging might be of great diagnostic value, where the MRI is the most complete exam.

The treatment should initially be conservative. Subsequently, surgical treatment and in particular the arthroscopic treatment can be indicated. Arthroscopy provides optimal clinical results by respecting the superior capsular ligament, the trapezium and deltoid muscular insertions, and the patient's aesthetical appearance.

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Arthritis and Synovitis of the Shoulder

31

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Epidemiology

By 2030, an estimated 67 million Americans ages 18 years or older are projected to have doctor-diagnosed arthritis [1]. Osteoarthritis (OA) affects many joints throughout the body. Although not as common as other locations such as the hip and knee, shoulder OA can be equally troubling for patients. The loss of shoulder function and therefore associated limitations of upper extremity motion can lead to depression, anxiety, activity limitations, and job performance problems [2]. Shoulder OA and the specific causes can be grouped into primary and secondary categories. Primary or idiopathic OA has no specific identifiable cause. Secondary OA results from an identifiable cause or predisposing factor, which include but are not limited to, shoulder trauma [3], dislocations [4], or chronic rotator cuff tear [5]. Epidemiologic data has demonstrated that glenohumeral arthrosis typically manifests after the sixth decade of life and women are more likely to have primary glenohumeral OA than men [6]. Younger patients, however, are also occasionally afflicted, and these patients represent a unique management challenge as many of these patients wish to maintain demanding lifestyles.

Advances in the management of rheumatoid and other inflammatory arthropathies have resulted in less need for surgical intervention. The use of disease modifying antirheumatologic drugs (DMARDs) has greatly reduced the prevalence of arthroplasty for the treatment of inflammatory arthritis [7]. However, the use of arthroscopy for debridement and synovectomy continues to be an effective treatment modality for those who fail nonoperative measures.

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Pathophysiology

The glenohumeral joint under ideal circumstances articulates through a smooth, congruent, and well-lubricated articular joint surface. Glenohumeral OA may result from disruption of these joint surfaces by any number of pathologic states [3–5]. These conditions are relatively common in the older populations. Degenerative disease of the shoulder joint results in a characteristic wear pattern between the glenoid and humeral articular surfaces. The glenoid cartilage and subchondral bone are typically worn posteriorly. At times this pattern may leave the articular cartilage intact anteriorly, resulting in a biconcave glenoid. The cartilage of the humeral head is eroded in a pattern of central baldness that is often surrounded by a rim of remaining cartilage and osteophytes. In inflammatory arthritis, the cartilage height loss is usually observed in a more symmetric distribution, demonstrating cartilage loss across the humeral head and glenoid surface.

History

Glenohumeral arthritis is classically characterized by pain, weakness, restricted motion, and loss of function. Advanced cases of arthritis are often refractory to rest, anti-inflammatory medications, and exercise. Associated cartilage loss and osteophyte formation lead to mechanical symptoms and impingement. Patients often describe a "grinding" sensation and mechanical blockage limiting glenohumeral motion. A thorough history should include a questioning regarding the onset of the problem, and the specific mechanism of any injuries. Surgical history is important as prominent hardware can lead to degenerative changes and mechanical symptoms, and chondrolysis is a well-described phenomenon following arthroscopic shoulder surgery. Further questioning should elucidate the extent of any functional difficulties or limitations and if the symptoms have been progressive or static nature. A past history of steroid medication or trauma which resulted in fracture may suggest the diagnosis of avascular necrosis [8].

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Systemic manifestations of underlying rheumatologic or autoimmune disease may be uncovered with directed questioning. Polyarticular complaints of pain, swelling, erythema may be the initial presenting symptom of a systemic disorder in a young patient presenting for evaluation. Any recent history of sepsis, degenerative joint disease, a family history of rheumatoid or autoimmune arthritis can provide helpful information to direct further evaluation.

Clinical Examination

A complete examination of the patient with shoulder arthritis includes the evaluation of the neck and cervical spine to rule out radiculopathy or myelopathy as the cause of the patients shoulder complaints. The focused shoulder examination begins with inspection to evaluate for muscle atrophy or deformity. Patients with isolated shoulder arthritis often have normal shoulder on initial inspection. Tenderness of the glenohumeral joint, greater tuberosity or biceps tendon may be present as well as presence of a joint effusion. The hallmark examination finding of shoulder arthritis is crepitation and mechanical block to both active and passive range of shoulder motion. A thorough assessment of rotator cuff function and evaluation for signs of impingement as well as biceps tendonitis and acromioclavicular joint pain are important; patients with arthritis often have additional shoulder pathology. A complete shoulder evaluation also includes an evaluation of shoulder stability, although uncommon in the face of arthritis of the shoulder. Neurologic and vascular function should also be evaluated as well, in addition to any other joints that may be symptomatic.

Imaging

The diagnosis of shoulder arthritis is based on a clinical examination and radiographic findings. The most important modality for the evaluation of shoulder arthritis is conventional radiography. Early in the development of glenohumeral arthrosis subtle radiographic findings may suggest the diagnosis when the symptoms are mild. Glenohumeral OA can be associated with inferior humeral or glenoid osteophytes, joint space narrowing, and subchondral cysts. In inflammatory arthropathy the presence of osteopenia or periarticular erosions can be seen [9–11].

Standard films of the shoulder joint are obtained consisting of an anteroposterior (AP), axillary and scapular-y view. To visualize different aspects of the humeral head, the arm can be internally or externally rotated. An axillary projection or superoinferior view helps to show the relationship of the glenoid fossa and the humeral head. In order to see the glenoid in profile, the patient can be rotated 40° toward the affected side, which is a posterior oblique view or Grashey projection.

Computed tomography (CT) is effective in portraying OA as it displays the degree of joint destruction. CT scans help to define the abnormalities of joint alignment, loose or foreign bodies, and osteophytes. The extent and location of osteophytes that need to be removed at the time of surgery make CT scan a valuable tool for preoperative planning.

Magnetic resonance imaging (MRI) is useful in providing contrast between bone and soft tissue and can detect early OA changes. The different signal intensities help to delineate the differences between bone, articular cartilage, fibrocartilaginous labrum, and synovium. MRI is also sensitive in providing information on effusions, osteochondral bodies in the joint, and cysts that are caused by OA. The evaluation of rotator cuff tendon, biceps tendon, and labral pathology is greatly facilitated by the use of MRI.

Treatment: Indications and Contraindications

The initial management of shoulder arthritis involves nonoperative measures including anti-inflammatory medications, exercise, physical therapy, and injections. When conservative treatment fails in elderly patients, total shoulder arthroplasty (TSA) is a successful operation with excellent functional outcomes and high satisfaction in subjective patient assessment [12, 13]. In younger patients the clinical results of shoulder arthroplasty in the short and midterm are good. However, reports of significant glenoid lucency or loosening in nearly 30 % of patients leads to concerns regarding the long-term survival of shoulder arthroplasty in the young, active patient [13]. Given these concerns and the less than ideal results of shoulder arthroplasty in young patients with chondrolysis [14], alternative surgical treatments that do not involve joint replacement are an attractive option. Shoulder arthroscopy with associated procedures including humeral head osteoplasty, microfracture, glenoid resurfacing, capsular release, and arthroscopic axillary nerve release has been reported for the treatment of shoulder arthritis [9–11, 15, 16]. Arthroscopic synovectomy is a successful treatment for early-stage rheumatoid arthritis of multiple joints including the shoulder [17].

Arthroscopic management of shoulder arthritis is a useful treatment in young or active patients for whom it is advisable to delay shoulder arthroplasty [18]. Shoulder OA is frequently seen concurrently with subacromial bursitis, acromioclavicular joint arthritis, labral tears, tendinopathy of the long head of the biceps tendon [18], and adhesive capsulitis [10]. Arthroscopic treatment of these concurrent disorders combined with debridement of the arthritic glenohumeral joint including osteophyte excision and capsular release has

shown good short-term results [19]. Younger patients with significant arthritis often have had prior operations for shoulder instability or labral tears. In these cases prominent hardware or infection may be contributing to the degenerative changes and addressing these complications is essential for successful outcome. Arthroscopic biologic resurfacing of the glenoid has recently been described with promising short-term results [20, 21]. In our experience patients less than 60 with painful shoulder arthritis who are unwilling or unable to conform to activity restrictions associated with TSA are good candidates for arthroscopic procedures. Contraindications to arthroscopic management of shoulder arthritis include patients with advanced degenerative changes, significant glenoid bone wear, and posterior subluxation of the humeral head.

Arthroscopic debridement of patients with severe glenohumeral arthritis has shown to relieve pain in nearly 80 % of patients with severe glenohumeral arthritis by 3 months and may provide relief for more than 4 years [15]. Arthroscopic management appears to be more successful in shoulders with a lesser degree of OA [11]. Further studies are needed to continue to evaluate the overall efficacy of shoulder arthroscopy in the treatment of shoulder OA.

Arthroscopic Treatment: Surgical Technique

There has been a recent explosion of new equipment and implants for arthroscopic shoulder procedures. Description of all the available devices is beyond the scope of this chapter. The essential tools include a 30° arthroscope, a fluid pump system, and standard arthroscopic instruments including arthroscopic shavers and burrs and a radiofrequency ablation wand. The use of arthroscopic cannulas and retractors can be a great aid leading to success of the operation.

Patient Positioning and Examination Under Anesthesia

Appropriate room setup and positioning greatly affects the ease of shoulder arthroscopy, and development of a simple and reproducible method with the resources available at your institution is essential. The use of surgical a team that is experienced in arthroscopic surgery greatly improves the ability to perform all arthroscopic shoulder procedures.

Examination under anesthesia is performed on every shoulder prior to the initiation of surgery. The correlation of preoperative pain and physical exam findings may be corroborated with the exam under anesthesia and the presence of a joint contracture or instability may alter the course of treatment.

Both the beach chair and lateral decubitus positions can be used for the arthroscopic management of shoulder arthritis. Our preference is to place the patient in the beach chair position with the head of the bed elevated to 80°. Care must be taken to pad all boney prominences and position the head and neck in neutral alignment. Access to the posterior aspect of the shoulder is essential and the entire scapula should be free from the edge of the table. The surgical arm is placed into an articulated hydraulic arm holder (Spider Arm Holder, Tenet Medical Engineering, Calgary, Alberta, Canada) which facilitates exposure, especially when there are limited or inexperienced surgical assistants.

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

A posterior viewing portal (2 cm medial and inferior to posterolateral corner of the acromion) is established using a blunt trocar with the arm placed in 15° of abduction and 30° of forward flexion with the assistant providing a gentle lateral distraction to avoid damage to the articular surfaces. The 30° arthroscope is introduced and diagnostic arthroscopic examination of the joint is performed. In the severely contracted or arthritic shoulder entering the glenohumeral joint can be difficult and using a spinal needle to localize the joint and infusion of sterile saline to distend the joint capsule may aid accessing the joint with the scope trocar. Thorough evaluation includes visualization of the articular cartilage of the humeral head and glenoid, labrum, biceps tendon, inferior recess, and articular surface and insertion of the subscapularis, supraspinatus, infraspinatus, and teres minor (Table 31.1, Fig. 31.1).

Step-by-Step Procedure

After completion of the diagnostic arthroscopy, attention is drawn to addressing the specific pathology encountered. In most instances, there are degenerative labral tears, synovitis or cartilage lesions, and osteophytes that require debridement. An anterior working portal in the rotator interval is the main working portal and allows for access to the majority of the joint. Accessory portals can be created to facilitate access to other parts of the joint including the 5-o'clock portal and the posterolateral portals. These portals are important in the visualization of the inferior recess and performing procedures including removal of loose bodies, debridement of the inferior humeral head osteophyte, inferior capsular release, and decompression of the axillary nerve. The use of a 70° arthroscope can also facilitate the visualization of parts of the joint with limited access including the anterior and posterior capsular attachments of the glenohumeral ligaments and the inferior recess (Table 31.2).

Table 31.1 Arthroscopic portal placement

Portal	Tip/pearl	Location
Posterior portal	Starting portal with best visualization of the glenohumeral joint. Can be used for debridement of posterior capsule and labrum	1–3 cm distal and 1–2 cm medial to posterolateral tip of acromion
Anterior portal	Working portal for debridement of glenohumeral joint cartilage, synovectomy of anterior, superior, and posterior joint. Useful for visualization of posterior labrum and capsule	Halfway between the AC joint and the lateral aspect of the coracoid. Placed using arthroscopic visualization and spinal needle localization into the triangle formed by the labrum (medial border), biceps tendon (superior border), and subscapularis (inferior border)
Posteroinferior portal (7-o'clock portal)	Visualization of the inferior recess and axillary pouch. Best visualization of inferior humeral osteophytes. Can also be used for debridement, removal of loose bodies, or resection of humeral osteophytes	5 cm distal to the posterolateral corner of the acromion. Placed using arthroscopic visualization and spinal needle localization into inferior recess at the medial and central aspect of the joint capsule. No sharp dissection to avoid axillary nerve injury
Anteroinferior portal (5-o'clock portal)	Visualization of the inferior recess and axillary pouch. Used for debridement, removal of loose bodies, resection of osteophytes, or retractor to protect axillary nerve	1 cm distal and lateral to the coracoid tip. Placed using arthroscopic visualization and spinal needle localization into the inferior recess through the inferior and lateral aspect of the subscapularis tendon. Avoid axillary nerve at inferior border of the subscapularis
Lateral portal	Instrumentation of the subacromial space for bursectomy and acromioplasty	2–3 cm distal to the lateral edge of the acromion in line with the posterior aspect of the acromioclavicular joint

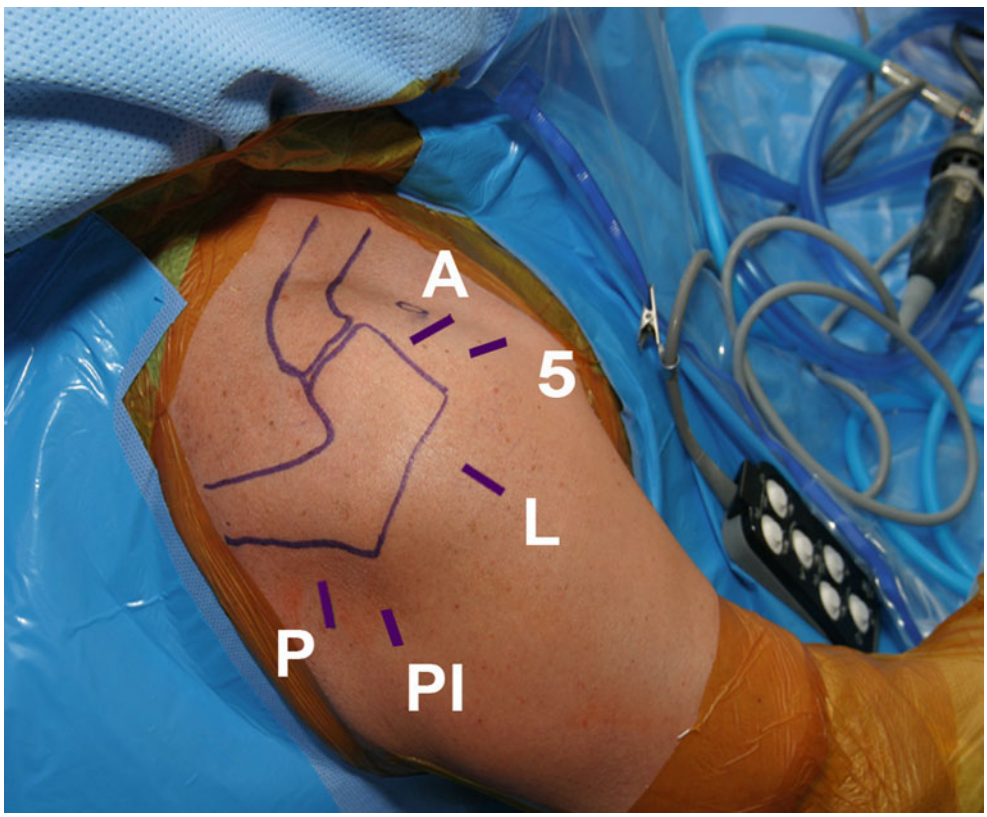

Fig. 31.1 Arthroscopic portal placement. *P* posterior, *PL* posterolateral, *L* lateral, *5* 5 o'clock, *A* anterior (see Table 31.1 for complete description)

There is a myriad of pathology that exists within the arthritic shoulder joint including loose or foreign bodies, cartilage lesions, synovitis, capsular contractures and degenerative tears of the labrum, rotator cuff, or long head of the biceps tendon. It can be challenging to determine what is causing the patients symptoms entirely, so all pathology should be addressed at the time of surgery. The cartilage should be evaluated on the glenoid and humeral articular surfaces and can be graded using the Outerbridge's classification [22] (Table 31.3).

The use of a full radius resector through the anterior portal allows for debridement of the majority of the articular cartilage of the glenoid and humeral head as well as any degenerative labral tears. In the presence of inflammatory arthropathy with an inflamed synovium a complete synovectomy should be performed. Full thickness cartilage defects that are less than 2 × 2 cm and are well shouldered by stable cartilage are amenable to microfracture. The microfracture awl should penetrate the subchondral bone plate to allow for the efflux of blood and marrow elements into the defect. The ultimate goal is the development of a fibrocartilaginous covering of the defect (Fig. 31.2). Loose or foreign bodies should be removed from the joint. The presence of prominent suture anchors or other fixation devices can be present. Having the appropriate instruments to remove the implants can be helpful or if unavailable a trephine can be used to core out the device (Fig. 31.3).

Table 31.2 Arthroscopic procedures for the treatment of shoulder arthritis

Removal of loose/foreign bodies
Chondroplasty/debridement of articular cartilage tears
Debridement of degenerative labral tears
Synovectomy
Address associated soft tissue pathology (rotator cuff/biceps tendon tears)
Removal of glenoid and humeral head osteophytes
Microfracture or abrasion arthroplasty of focal full-thickness cartilage lesions
Capsular release if contracture present
Subacromial bursectomy +/– acromioplasty
Distal clavicle excision if symptomatic acromioclavicular joint arthritis

Table 31.3 Outerbridge classification [22]

Grade	Pathology
I	Softening and swelling of the cartilage
II	Fragmentation and fissuring in an area half an inch or less in diameter
III	Same as grade 2 but an area more than half an inch in diameter is involved
IV	Erosion of cartilage down to bone

Rotator cuff pathology should be addressed as indicated with debridement or repair depending on the extent of the tear. The biceps tendon can often be a pain generator in patients with arthritic conditions, and pathology is treated as indicated with debridement, tenotomy, or tenodesis depending on the degree of injury and surgeon preference. Our preference in young active patients or manual laborers is to perform biceps tenodesis arthroscopically in the proximal aspect of the intertubercular groove using a biceps tenodesis screw. In elderly patients or non-laborers a simple tenotomy has been shown to be as effective in pain relief without significant limitation in strength or function [23, 24].

After treatment of the cartilage and soft tissue pathology, attention is directed to removal of any impinging osteophytes. Glenoid osteophytes are often encountered and should be removed using the full radius shaver or burr (Fig. 31.4). The

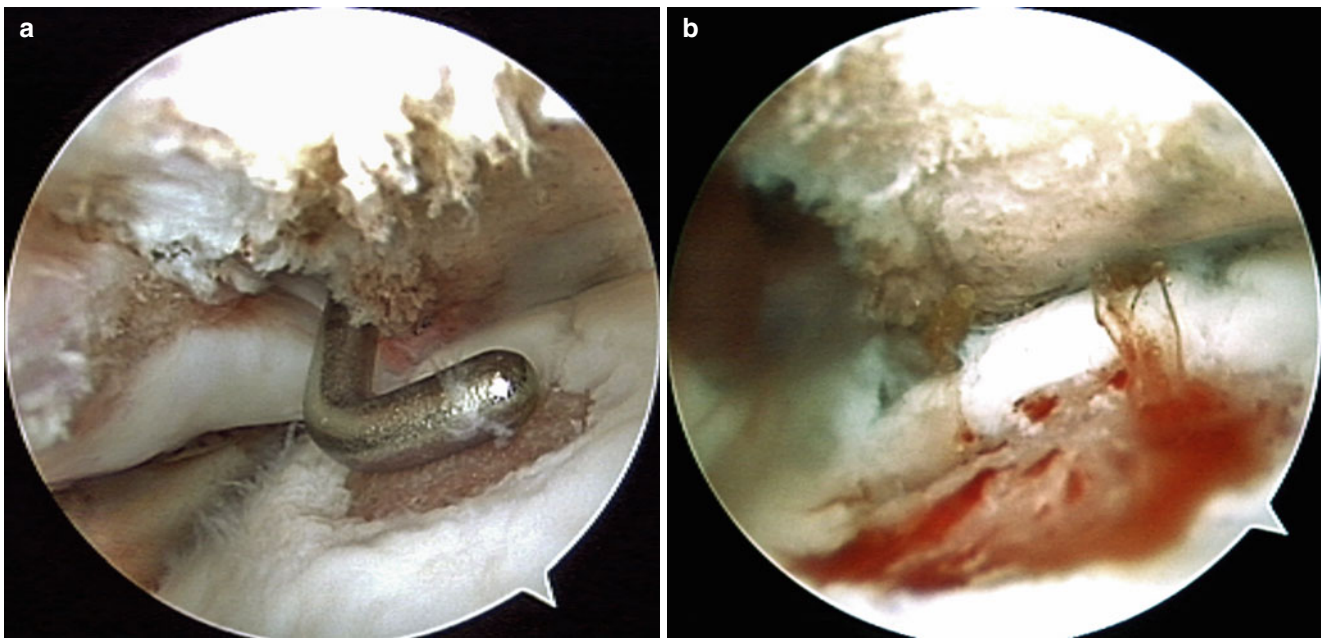

Fig. 31.2 (**a**) A full-thickness cartilage defect measured with an arthroscopic probe that would be amenable to microfracture. (**b**) The same defect shown in (a) one following the microfracture procedure demonstrates the egress of blood and marrow contents following subchondral penetration

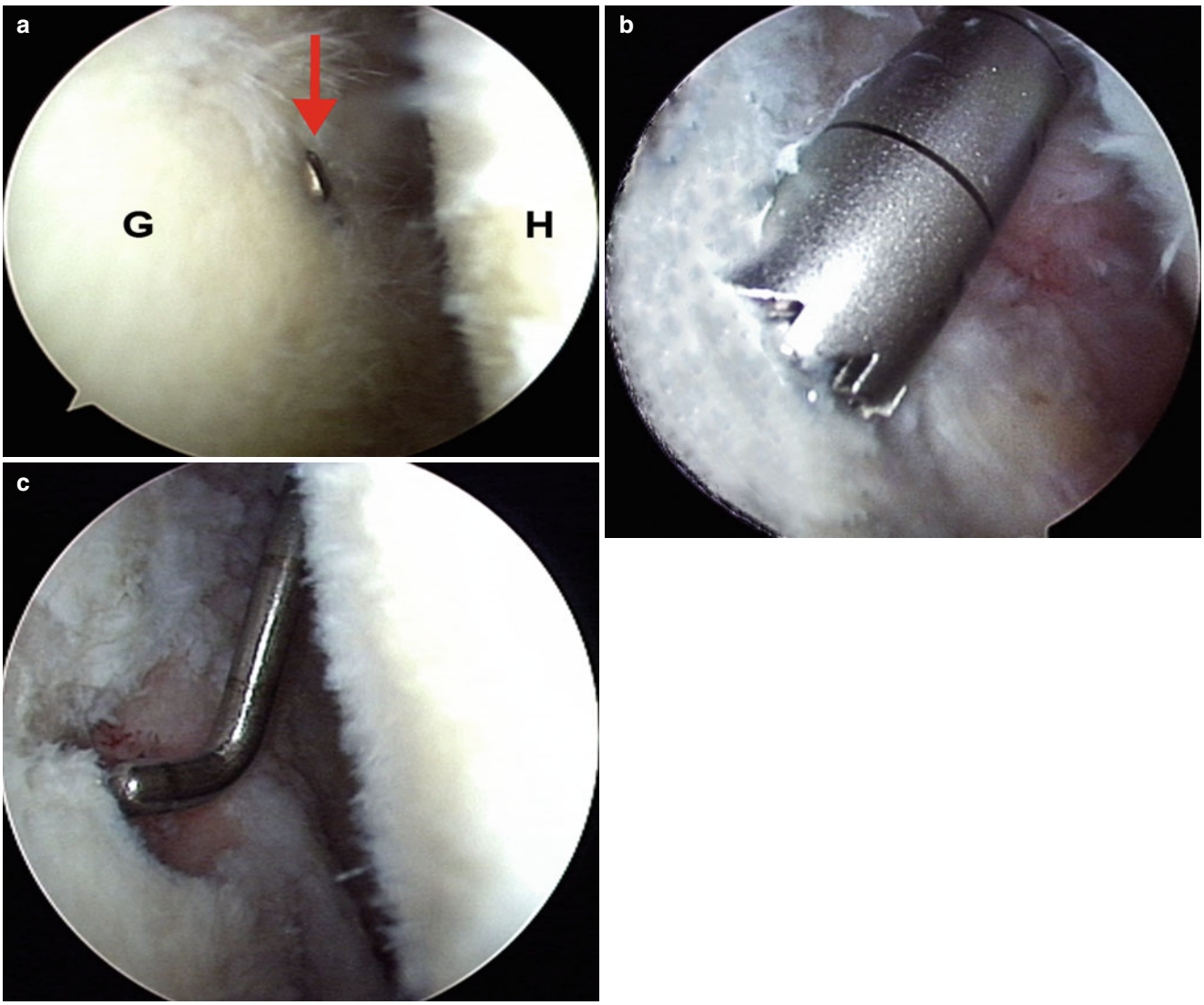

Fig. 31.3 (**a**) Prominent glenoid anchor (*arrow*). (**b**) The figure demonstrates the process of prominent glenoid anchor removal. (**c**) Arthroscopic view of the glenoid from the posterior viewing portal following the removal of a prominent anchor

hallmark of shoulder arthritis is the inferior humeral osteophyte. This osteophyte often limits range of motion by impinging on the glenoid rim, and it has been suggested that it can also cause pressure on the axillary nerve resulting in shoulder pain similar to quadrangular space syndrome [9]. Removal of this osteophyte is a key step in the arthroscopic management of the arthritic shoulder. The use of the 5-o'clock and 7-o'clock portals greatly improves the visualization and removal of the inferior humeral osteophytes. The resection is started posteriorly with the burr in the 7-o'clock portal and a retractor can be placed in the 5-o'clock portal to protect the axillary nerve. Avoiding penetration or release of the inferior capsule helps to protect the axillary nerve, and the use of suction on the burr should be avoided when working in the inferior recess. The plane between the osteophyte and normal humeral bone is separated by a thin layer of fibrous tissue which can be used to determine adequate resection. The resection is carried as far anteriorly as possible, to completely resect the anterior most aspect of the osteophyte the scope may need to be changed to the 7-o'clock portal with the burr placed in the 5-o'clock portal (Fig. 31.5).

The last step within the glenohumeral joint is to perform a contracture release as needed. In most cases there will be an internal rotation contracture which can be treated by performing a rotator interval release. In cases of more significant contracture the anterior, posterior and inferior capsule may need to be released. Capsular releases are continued until the patient has full range of motion including forward flexion, abduction, and internal and external rotation at both neutral and 90° of abduction. The capsular release is easiest to perform using a radiofrequency wand. Caution should be taken with the use of radiofrequency within the joint to pre-

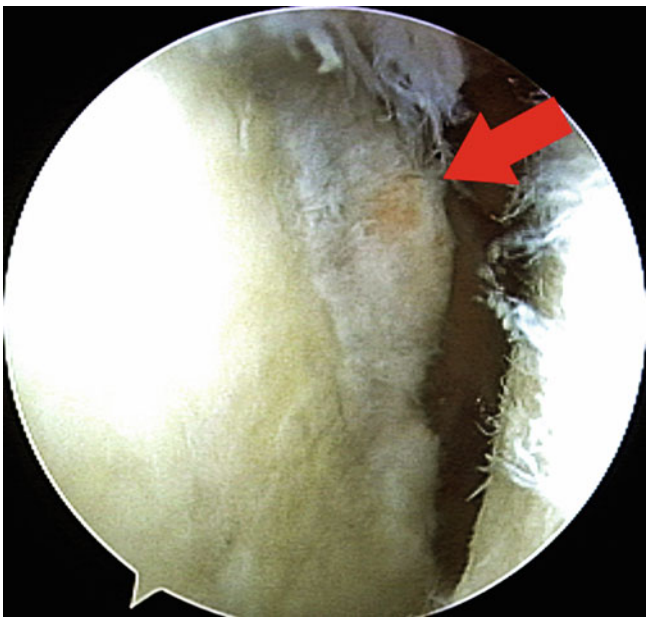

Fig. 31.4 Glenoid osteophytes, viewed from the posterior portal, are often encountered and should be removed using the full radius shaver or burr

vent overheating of the arthroscopic fluid which can result in increased cartilage damage. Additionally, care must be used with using radiofrequency for release of the inferior capsular structures due to the close proximity of the axillary nerve.

Following completion of the glenohumeral arthroscopy, the arthroscope is transitioned to the subacromial space. A hypertrophic or inflamed bursa is common and complete bursectomy should be performed. The bursal side of the rotator cuff and anterior acromion are examined for evidence of impingement and acromioplasty is performed as needed. If the patient demonstrates radiographic and clinical evidence of acromioclavicular joint arthritis, then distal clavicle excision is performed. In our experience the presence of symptomatic acromioclavicular joint arthritis is rare in the patient with glenohumeral arthritis.

Once the shoulder arthroscopy and any ancillary procedures are complete, hemostasis is obtained and the arthroscopic portals are then closed with an absorbable buried stitch followed by steri-strips and Dermabond (Ethicon, Inc., Cornelia, GA, USA) skin closure.

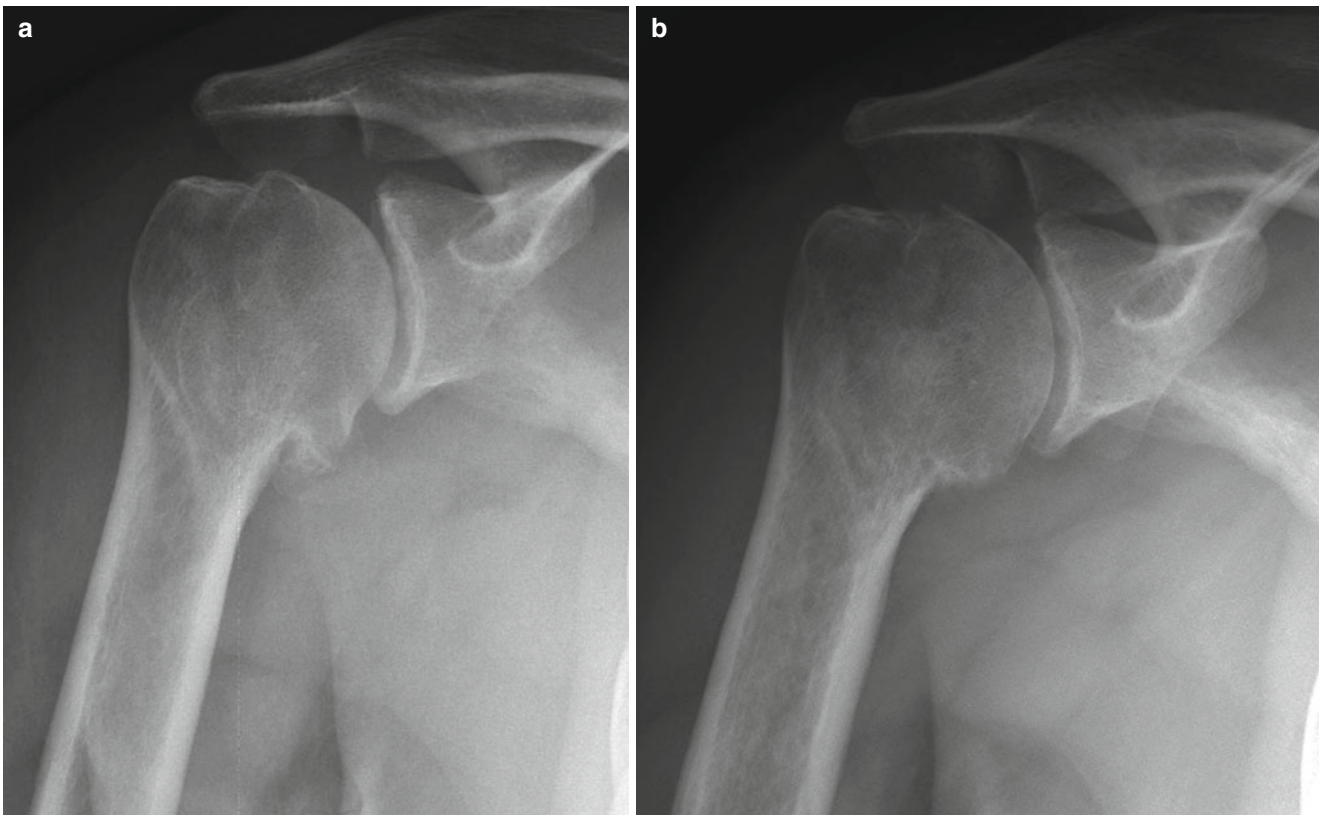

Fig. 31.5 The hallmark of shoulder arthritis is the inferior humeral osteophyte, as demonstrated in the preoperative radiograph (**a**). This osteophyte often limits range of motion by impinging on the glenoid rim and has been removed arthroscopically as demonstrated in the postoperative radiograph (**b**)

Postoperative Care

The patient is placed in a sling and discharged home once meeting postoperative care unit criteria. Gentle range of motion activities are initiated immediately and physical therapy within the first several days.

Literature Review

The existing literature on the outcomes of the arthroscopic management of shoulder arthritis is limited to level IV and V evidence. Historically the short- and midterm outcomes have been successful in between 50 to 80 % of patients. There are no reports of long-term outcomes following arthroscopic management of shoulder arthritis. The available literature has significant variability in the indications, procedures performed, follow-up, and outcome measures such that direct comparison between studies is nearly impossible (Table 31.4).

An important part of the arthroscopic management of shoulder arthritis is addressing associated pathology. Reports by Ellman et al. and Guyette et al. evaluated glenohumeral debridement with subacromial decompression and noted good results in short-term follow-up in patients with mild to moderate arthritis. The procedure was less successful in patients with advanced glenohumeral arthritic changes. There have not been any studies that have reported the results of biceps tenodesis or distal clavicle excision in the treatment of glenohumeral OA.

Recent reports have shown favorable results in greater than 80 % of patients who are treated with glenohumeral debridement, excision of impinging osteophytes, and capsular release. Millett et al. proposed axillary nerve decompression as an important part of the arthroscopic management of arthritis. Their belief is that an osteophyte in the axillary region may compress the axillary nerve and potentially contribute to posterior shoulder pain in a manner similar to quadrilateral space syndrome. In 27 shoulders they reported a high patient satisfaction rate, decreased pain, increased range of motion, and improved American Shoulder and Elbow Surgeons scores. In the reported time frame, only 1 patient had elected to undergo TSA, and they reported no complications in their series [9].

There has been recent interest in biologic resurfacing procedures for the treatment of arthritis in the younger patient

Table 31.4 Arthroscopic management of shoulder arthritis: summary of studies

Authors	Date	*N*	Follow-up (months)	Surgical technique	Outcome	Level of Evidence
Millett and Gaskill [9]	2011	26	20	Glenohumeral debridement Capsular release Inferior humeral osteoplasty Axillary nerve decompression	"High" patient satisfaction "High" patient satisfaction "Improved" ASES scores No reported complications	IV
Van Thiel et al. [11]	2010	81	27	Glenohumeral debridement – multiple concomitant procedures	Pain: VAS 4.8 → 2.7 ROM: Flexion – 137 → 157 Abduction – 129 → 145 External rotation – 48 → 63 ASES: 51.8 → 72.7 SST:6.1 → 9	IV
Kerr and McCarty [16]	2008	19	20	Glenohumeral debridement – 19 patients concomitant procedures	Shoulder function greater than 60 % in all but 3 patients 3 patients progressed to shoulder arthroplasty Outcome of unipolar lesions better than bipolar lesions ASES – 75.9	IV
Richards and Burkhart [19]	2007	8	13.7	Glenohumeral debridement Release rotator interval, anterior capsule, posterior capsule, and axillary recess	Improved ROM: Forward elevation: 21.4° External rotation: 16.6° Internal rotation: 31.1°	IV
Weinstein et al. [10]	2000	25	34	Glenohumeral debridement Partial synovectomy Subacromial bursectomy	Pain: 2 patients complete relief 18 patients occasional mild 5 moderate to severe ROM: improvement was noted	IV

population. There have been recent reports of arthroscopic techniques for biologic resurfacing of the glenoid with promising results in the short term. De Beer et al. in 2010 analyzed 32 consecutive patients who underwent an arthroscopic debridement and biological glenoid resurfacing using an acellular human dermal scaffold. The Constant and Murley score increased from a median of 40 points preoperatively to 64.5 at the final assessment. The procedure resulted in a "successful outcome" in 23 patients (72 %) and as a "failure" in 9 patients (28 %). There were five conversions to prosthetic arthroplasty and five complications, including transient axillary nerve paresis, foreign-body reaction to biological material, interlayer dissociation, mild chronic nonspecific synovitis and posttraumatic contusion [20]. The results of biologic resurfacing with a porcine submucosal xenograft were examined by Savoie et al. Glenoid resurfacing with the biologic patch provided statistically significant improvements for young patients with severe glenohumeral arthritis as measured by the VAS, ASES, UCLA, Rowe, Constant-Murley, and SF-12 scores at 3–6 years of follow-up [21].

Summary

Shoulder arthritis can be a severely debilitating condition with patients reporting pain and limited function with the affected extremity. Total shoulder arthroplasty is an excellent procedure for the elderly or low-demand patient; however, concerns regarding implant failure and need for revision surgery are concerning in the young and active patient population. Arthroscopic techniques have gained popularity due to the low morbidity and retention of the patient's own anatomy. The results of arthroscopic procedures for shoulder arthritis have variable outcomes in the literature. With more aggressive debridement, excision of osteophytes and capsular release good results can be expected in up to 80 % of patients in the short to midterm. Further study is needed regarding the best techniques and appropriate indications for the arthroscopic management of shoulder arthritis.

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Part IV

Complex and Revision Procedures in Shoulder Arthroscopy

Failed Instability Surgery

32

Alessandro Castagna, Raffaele Garofalo,
and Eugenio Cesari

Epidemiology

Glenohumeral instability is very common in the general population and many surgical techniques have been described for the treatment of this condition, each with different indications according to the pathological findings and patient's age, gender, and activity level [1–5]. Despite the evolution in knowledge of the pathology and surgical techniques treating shoulder instability, a certain risk of failure, reported in a percentage between 4 and 30 %, still exists after primary surgical stabilization: arthroscopic, open or with bone block procedure [1, 6–8]. Nevertheless, the percentage of failure could be even higher when not only a recurrence of instability (dislocation or subluxation) but also a painful or a stiff shoulder is considered as a failure [2].

A failure of instability surgery leads to increased morbidity to the patient, increased pain, decreased activity level, prolonged time away from work and sports, and a general decrease in quality of life. This emphasizes the importance of a proper assessment of the patient with shoulder instability at the time of the first operation in order to reduce the risk of this critical complication.

In this chapter we analyze the etiologic risk factors associated with a failed instability surgery. The analysis of these failures (recurrent instability, apprehension, painful and stiff shoulder) can help to better understand the pathology and improve the treatment strategy at the time of the first surgery.

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Pathophysiology

In case of failure of instability surgery, we have to understand if there was a mistake in the diagnosis, indication to treatment, or surgical technique; in fact, not all the surgical techniques are the same and have the same efficacy.

Failure of shoulder stabilization procedures is often related to unaddressed pathology. In particular, the reasons can be related to an untreated associated laxity, or a pathology of rotator interval, unaddressed bony defect, or biologic reasons. The knowledge of pathophysiology of shoulder instability is very important in the work-up of patient with failed instability surgery.

Glenohumeral joint is an intrinsically unstable joint because the large humeral head articulates with the shallow narrow glenoid. This geometry provides a functional benefit by allowing for a large arc of motion but also confers an inherent instability. The stability of this joint is related to a complex network between static factors like bone structures, capsuloligamentous and labral complex, and dynamic factors, such as rotator cuff muscles that acts compressing the humeral into the glenoid socket during the range of motion, and scapular stabilizer muscles, which orient the scapula during scapulothoracic and scapulohumeral movements. In consequence, failure of each of these stabilizing mechanisms can lead to shoulder dislocation and recurrent instability. Furthermore, activity level should be included in the factors associated with shoulder stability.

Traumatic episode of dislocation leads to a detachment of the labral complex anterior or posterior, according to the direction of dislocation, with or without an associated bone lesion. Dislocation, however, also leads to a detensioning or a tear of the capsuloligamentous complex. In the first treatment planning, bone lesions and associated rotator cuff tear are two very important factors to recognize, because otherwise they can be responsible of treatment failure. Glenoid defect can result from a fracture associated with a labral detachment (glenoid fracture or bony Bankart according with the size of fractured bone) or from an attritional bone

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loss, as we observe in cases of recurrent shoulder dislocation. Itoi et al. [9] in a cadaver study showed that a glenoid defect greater than 21 % of the glenoid width substantially reduced the translational force required for glenohumeral dislocation. The amount of glenoid bone defect should be identified preoperatively using a CT scan. Some authors describe the possibility to measure this defect arthroscopically. Burkhart et al. [10] suggest that the bare spot can be used as a reference point to determine glenoid bone loss because it is located at the center of a circle of the articular margin of the inferior glenoid below the level of the mid-glenoid notch. Using this technique, they recommend that bone loss of the anteroinferior glenoid can be expressed as a percentage of glenoid width and that glenoid bone grafting should be considered for defects greater than 25 %. However, this criterion is not long useful because of the variability of the position of the bare spot.

Significant humeral head defects are also associated with recurrent shoulder instability or failed surgery. With anterior shoulder dislocation, the humeral head impression fracture involves the postero-superolateral humeral head and is termed as Hill-Sachs lesion. With a posterior shoulder dislocation, the impression defect is anteromedial and is termed reverse Hill-Sachs or McLaughlin lesion. In the past, a significant defect was defined when it was greater than 20–40 % of the humeral head surface. More recent laboratory evidence suggested that defects as small as 12.5 % of the humeral head can be also significant, and certainly 25 % bone humeral head defects have biomechanical consequences that may affect joint stability [11]. However, Sekiya et al. [12] recently reported that an isolated 25 % Hill-Sachs defect cannot be responsible for recurrent instability if the function of the capsule is restored to the intact status. This difference can be related to the fact that the authors did not take into account the position of the humeral defect. Other studies, in fact, showed that the position of the defect has a critical role in determining the failure of instability repair. Burkhart and DeBeer [13] first suggested that Hill-Sachs lesions with a long axis parallel to the anterior glenoid rim when the shoulder is in abduction and external rotation were more likely to result in subluxation or dislocation. They defined this defect as "engaging Hill-Sachs lesion." On the other hand, non-engaging Hill-Sachs lesions diagonally cross the anterior glenoid rim during abduction and external rotation and, therefore, do not commonly contribute to recurrence of instability. Balg and Boileau [14] noted that if this defect is visible on the radiographs with the arm in external rotation, this can be a risk factor for surgical failure after a soft tissue procedure. More recently, Yamamoto et al. [15] introduced the concept of "glenoid track" showing that a Hill-Sachs lesion is at risk of engagement and dislocation if it extends medially over the medial margin of the glenoid track.

Although bone defects are a very important issue in failed stability surgery, other aspects should be also taken into account. There is consensus on the importance of an intact capsule for the stability of the shoulder joint. According to Rowe et al. [16] and Bigliani et al. [17], the capsule is always overstretched after shoulder dislocation, and capsular redundancy must be addressed at the time of surgery. Levine et al. [18] noted that the most frequent causes of unsuccessful shoulder stabilization were related to the failure to correct an excessively large anteroinferior capsular pouch and detached capsulolabral complex. A generalized ligamentous laxity or a patulous, poor-quality capsular tissue can also result in failure of surgery also in the absence of bone defects. Fujii et al. [19] reported that in patients with a higher frequency of dislocation, capsule often showed histologic degenerative changes and could not be good for repair. Finally, humeral capsular avulsion from the anterior or posterior side should be diagnosed preoperatively or during surgery. If not adequately addressed, these lesions can result in a failure of stabilization surgery [20].

History

A useful way to determine any failed surgical instability procedure is to consider the following possible causes as preoperative, intraoperative, and postoperative.

Preoperative factors typically include the failure to diagnose the pathology and associated lesions (e.g., the presence of significant bony defects in the humerus or glenoid or both, associated laxity or defects of the rotator interval, or neurological and psychological components). Furthermore, age and activity level of the patient should be also taken into account. Excessive soft tissue laxity and high sport activity level can suggest an increased risk of surgical failure in the younger patients. Patients over 45 years of age with a recurrent instability should be suspected of having an associated rotator cuff tear.

Intraoperative factors include surgical errors, such as non-anatomic repair of the capsuloligamentous-labral complex, insufficient number of suture anchors used to stabilize the torn labrum [1], unaddressed capsular redundancy which would have required capsular plications, and undiagnosed anterior or posterior humeral capsular avulsion [20].

Postoperative factors include inadequate duration of postoperative immobilization and unwillingness of patients to adhere to rehabilitation program and correct timing for resumption of at-risk activities.

Patients who present with failed shoulder instability surgery typically complain about recurrent instability, pain, discomfort, and/or shoulder stiffness. All these symptoms, isolated or combined, can impair patients' satisfaction and preclude their return to previous activities.

If failure is due to recurrence of instability, specific questions to be asked include whether or not recurrence was traumatic, and if there is apprehension or instability in the mid-ranges of motion. Because unrecognized or underestimated capsular laxity and glenohumeral bone defects are responsible for many atraumatic failures, clinical history should focus on distinguishing these lesions from other sources of instability surgery failure. Patients, who probably have these pathological findings, often report atraumatic dislocation events associated with activities of daily living and occasionally describe dislocations that occur during sleep. In addition, subluxation or dislocation episodes may occur at lower degrees of abduction and external rotation than what patients typically report. Furthermore, repetitive episodes of dislocation or subluxation may exacerbate capsular laxity or attritional bone loss from the glenoid. Patients with large glenohumeral bone defects or with a humeral capsular avulsion often describe a dramatic traumatic episode requiring glenohumeral reduction in the emergency department [21].

Possible causes of shoulder stiffness should be accurately investigated. In this case it is important to understand the time and position of immobilization and compliance of the patient to postoperative cares, and it is also important to review the charts about preoperative and intraoperative examination. Rarely, preoperative loss of motion in patients suffering from recurrent dislocations may not be simply related to apprehension, but to stiffness developed over time by the patient because of limited use of the shoulder in an attempt to avoid further episodes of dislocation. A stiff shoulder can also be associated with a persistent feeling of instability, caused by a non-anatomic repair of capsulolabral complex.

Patients who underwent a previous Latarjet or Bristow procedure and who are complaining about shoulder pain should be carefully evaluated. In fact, the pain can be also associated with a residual instability or just related to the screws or stretching of the musculocutaneous nerve.

Clinical Examination

Clinical examination is essential to evaluate a patient with a stabilization surgery failure. Examination starts with inspection of shoulder, then range of motion is assessed on comparing the affected shoulder with the contralateral one.

Excessive asymmetric passive range of motion can indicate capsular laxity. On the contrary, assessment of loss of passive range of motion is helpful in determining which part of the capsule is the most affected by stiffness, if any.

External rotation greater than 85–90° with the arm at the side indicates a congenitally weak anterior capsule or anterior capsular laxity and can be a risk factor for failed instability surgery. An additional assessment of glenohumeral joint laxity should include the "sulcus" test. This test is used to assess the integrity of the rotator interval, which is formed by the coracohumeral ligament, and superior glenohumeral ligament. The test is performed by pulling inferiorly on the humerus with the arm at zero degrees of abduction in neutral and external rotation. If the sulcus sign disappears in external rotation, the rotator interval is deemed competent. On the other hand, asymmetrical loss of passive external rotation with the arm at the side may be indicative of over-constraint of the subscapularis, rotator interval, and/or middle glenohumeral ligament and may herald a potential technical issue in that the primary instability pathology (inferior glenohumeral ligament) was not treated.

Inferior capsular laxity can be identified with the Gagey test. Hyperabduction to 105° or asymmetrical hyperabduction test more than 20° indicates inferior capsular laxity due to stretching of the inferior glenohumeral ligament [22]. Asymmetrical loss of motion in abduction and external rotation (ABER) may identify non-anatomic over-constraint of the inferior glenohumeral ligament. Tightness of the posterior capsule limits cross-body adduction and internal rotation.

The scapular mechanics should be evaluated during active motion. An abnormal scapulohumeral rhythm or scapular winging can predispose to secondary glenohumeral instability and must be addressed before any revision stabilization procedure.

Shoulder stability should also be assessed with provocative tests to evaluate the extent and direction of any instability. A positive apprehension test with the arm at 45° of abduction may be indicative of substantial bone loss.

Pain or grinding during motion, with or without stiffness, should alert the surgeon to additional pathology, such as chondrolysis and anchor or screw malposition. The presence of crepitation with ABER position may increase the suspicion of an engaging Hill-Sachs defect.

Neurological status and strength of the injured shoulder should be compared with the contralateral shoulder and strength in all directions should be evaluated. Weakness in one or more directions should alert the surgeon to the presence of concomitant pathology, such as rotator cuff tear or suprascapular nerve palsy. Patients with associated rotator cuff injury may be at higher risk for treatment failure. Specific attention to subscapularis function using the belly-press test and lift-off maneuver should always be performed. In patients who have undergone previous open surgery, failure of subscapularis repair or subscapularis dysfunction may be present and has been reported in a percentage of 23 % in a previous study at an average of 4 years after open Bankart repair [23]. More recently, Scheibel et al. [24] reported decreased subscapularis volume and diameter and clinical dysfunction in 70 % of patients after open Bankart repair with subscapularis tenotomy.

Imaging

Imaging is very helpful in diagnosing failed instability shoulder surgery. In our practice, work-up starts with standard radiographs, including true anteroposterior (AP) view (Grashey view), AP views in internal and external rotation, scapular Y view, and axillary lateral view. These images allow to find out metal hardware loosening (screws or suture anchors), nonunion of bone grafts (i.e., the coracoid graft in case of failed Latarjet procedure), and signs of chondrolysis or early osteoarthritis. Moreover, we can get information about the presence and location of a Hill-Sachs lesion. Hill-Sachs lesion detectable on the AP view in external rotation could be the cause of failure of a previous arthroscopic shoulder stabilization [14]. Magnetic resonance (MR) is the modality of choice to evaluate soft tissues, including the glenoid labrum, glenohumeral ligaments, and rotator cuff. Sagittal oblique MR scans may be used to evaluate the muscle belly of the subscapularis for atrophy and fatty infiltration in patients with failed open procedure related to an insufficient or injured subscapularis.

MR arthrography (MRA) is helpful to identify untreated concurrent glenohumeral pathology as cause of failure, such as superior glenoid labrum anterior to posterior (SLAP) lesions, rotator cuff tears, patulous capsular stretching, anterior humeral avulsion of the glenohumeral ligaments (HAGL), and posterior or reverse HAGL (PHAGL or RHAGL) [20]. Computed tomography arthrography (CTA) can provide same information regarding soft tissues and can also be used to identify and quantify bone loss of the glenoid and humeral head. Three-dimensional (3D) CT has been noted to be the most reliable imaging modality for predicting glenoid bone loss. Furthermore, it can show the number and location of suture anchors and any sign of bone resorption around implants [25].

Once that clinical and imaging work-up has been completed, the planning for revision treatment should be done accordingly.

Decision-Making Algorithm

Proper decision making must take into account all the factors related to the patient history, clinical examination, imaging, and previous surgery. Preoperative planning for revision surgery is mostly based on the following issues: type of previous surgery, presence of bone loss, supposed quality of capsulolabral tissue, combined lesions, and patient's activity level.

The ideal candidate for an arthroscopic revision surgery is the patient with traumatic unidirectional instability with a repairable Bankart lesion and good-quality capsular tissue. Arthroscopic revision surgery is also preferred when preoperative work-up showed the presence of other combined repairable lesions (e.g., superior labral tears, PHAGL lesions, rotator interval insufficiency, or reparable rotator cuff tears) which can be best addressed arthroscopically. Unfortunately, some of these lesions, such as an unaddressed PHAGL lesion, cannot be easily recognized before surgery. Therefore, we recommend an arthroscopic revision surgery when in the preoperative work-up there is no evidence of pathological findings that contraindicate an arthroscopic surgery, such as a significant bone defect, or when we suspect that the quality of capsular tissue is poor.

When an at-risk Hill-Sachs lesion is evident without significant associated glenoid bone loss, an arthroscopic Bankart repair associated with a posterior "remplissage" should be done.

Arthroscopic management can be also considered in patients complaining about recurrent instability after a Latarjet procedure and in whom the preoperative work-up has showed an healing of bone graft and not concerns about screw malpositioning or loosening.

Shoulder stiffness associated or not with a residual apprehension or instability often requires a tailored treatment, which can be better achieved arthroscopically.

Open or arthroscopic bone block procedures (Bristow-Latarjet, Heyden-Hebynette, J-plasty) should be performed when preoperative evaluation showed a significant glenoid bone defect, when we suspect the quality of capsulolabral tissue is poor, and in case of failed Latarjet procedure because of bone block nonunion or resorption.

When the failure of previous open procedure is related to an insufficient or ruptured subscapularis, treatment options include subscapularis repair if possible, pectoralis major transfer, or allograft reconstruction.

Patients with seizure disorders, scapular dyskinesia, multidirectional instability, and voluntary instability are not optimal candidates for revision surgery. For these patients, nonsurgical treatment should be maximized before surgical alternatives are considered.

Clinical Case/Example

A 21-year-old man complained of pain in the right shoulder associated with stiffness and functional impairment. At the age of 16 years, he had a first episode of anterior traumatic dislocation of the right shoulder that was self-reduced. Since then, he had many episodes of recurrent anterior instability which were always self-reduced.

At the age of 18 years, he underwent surgery consisting of arthroscopic repair with three knotless metallic single-loaded suture anchors. Postoperative course was without problems and the patient resumed his sports activities. One year after the surgery, he sustained a new episode of dislocation related to a minor trauma. Dislocation was again self-reduced. After this episode he suffered of several episodes of recurrent anterior

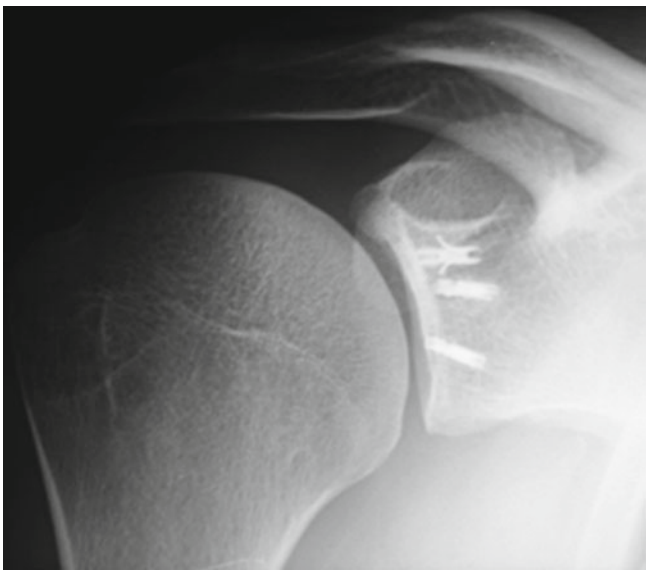

Fig. 32.1 The postero-anterior view of the right shoulder shows the presence of metallic anchors of the first operation, without any migration. No signs of chondrolysis were visible

instability, always self-reduced. At the age of 20 years, the patient underwent a revision procedure consisting of arthroscopic Bankart repair with three biodegradable single-loaded suture anchors. After 1 year he started to complain of shoulder pain associated with a functional impairment.

At clinical examination he had some pain. Assessment of range of motion showed a forward flexion of 160°, abduction of 160°, external rotation with the arm at side of 15°, and internal rotation at the level of T12. Sulcus sign was negative. No signs of rotator cuff tears were found. A posterior pain during elevation and internal rotation was also observed. Radiographic series showed no mobilization of suture anchors used in the previous surgeries nor signs of chondrolysis (Fig. 32.1). MRI showed no signs of pathological findings in the anterior part of the joint (Fig. 32.2). Because of stiffness, the patient underwent a rehabilitative treatment for 3 months. However, he failed to improve with rehabilitation and an arthroscopic surgical management was proposed.

Patient was placed in a lateral decubitus position with the right arm placed in sleeve traction. Five kilogram of balanced suspension was used with the arm in 70° of abduction and 20° of forward flexion. The scope was introduced through a standard posterior portal for diagnostic arthroscopy. A classic anterosuperior portal just anterior to the long head of the biceps tendon was made with an inside-out technique and a 5.5-mm cannula was inserted. Diagnostic arthroscopy was performed by switching the scope between the two portals. In the anterior part of the shoulder, as suspected before surgery, we found signs of capsulitis with a thickness inflamed tissue at the level of the rotator interval. With the scope in the

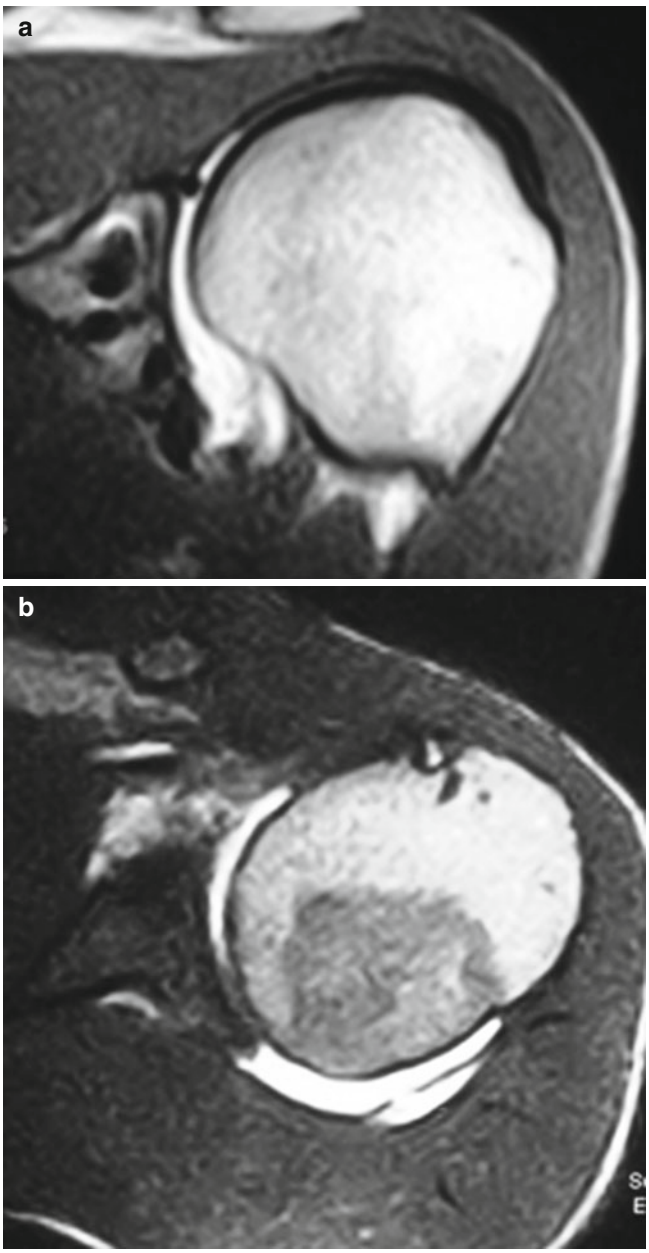

Fig. 32.2 MR exam of the right shoulder. (**a**) Sagittal oblique scans showed no signs of pathological findings in the anterior part of the joint. (**b**) On the axial scans an abnormal capsular pouch at the posterior aspect of the joint can be observed

anterosuperior portal and looking to the posterior part of the shoulder joint, we found a PHAGL lesion (Fig. 32.3).

Arthroscopic signs of the PHAGL lesion consisted of a direct visualization of the muscle fibers of the posterior cuff through the avulsed joint capsule, which was detached from the humeral neck and retracted into the posterior gutter. Once the diagnosis had been made, the scope was inserted in the posterior portal, and using a radiofrequency device through the anterosuperior portal, we performed an aggressive arthroscopic release of the rotator interval, particularly

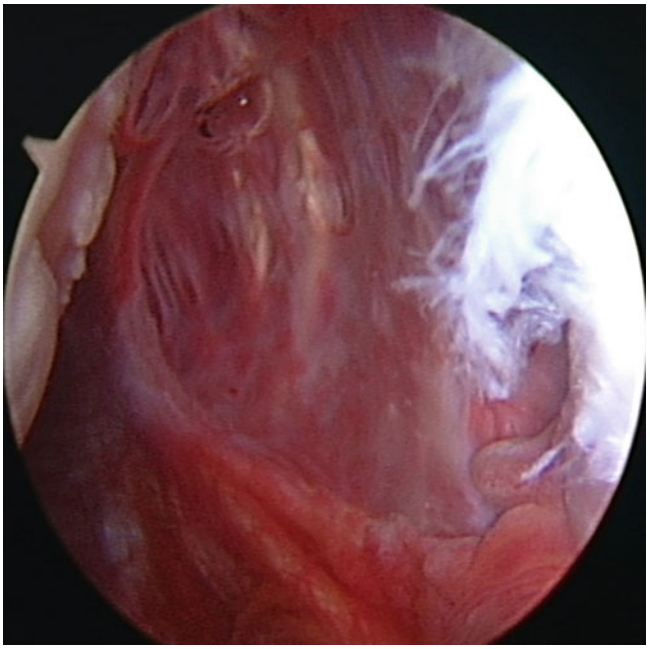

Fig. 32.3 Arthroscopic view of the PHAGL lesion in the right shoulder as viewed from the anterosuperior portal

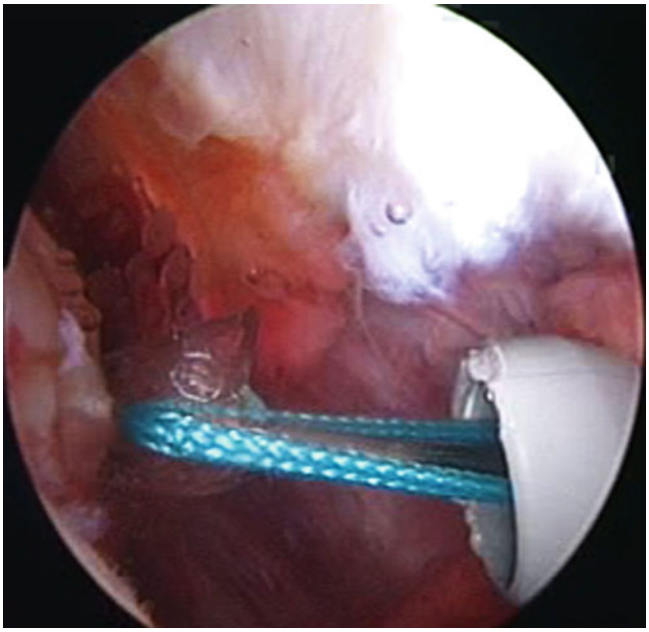

Fig. 32.5 With the scope in the anterosuperior portal, a biodegradable suture anchor is inserted through the posterior portal. The anchor is single-loaded loaded with a #2 high-strength braided suture

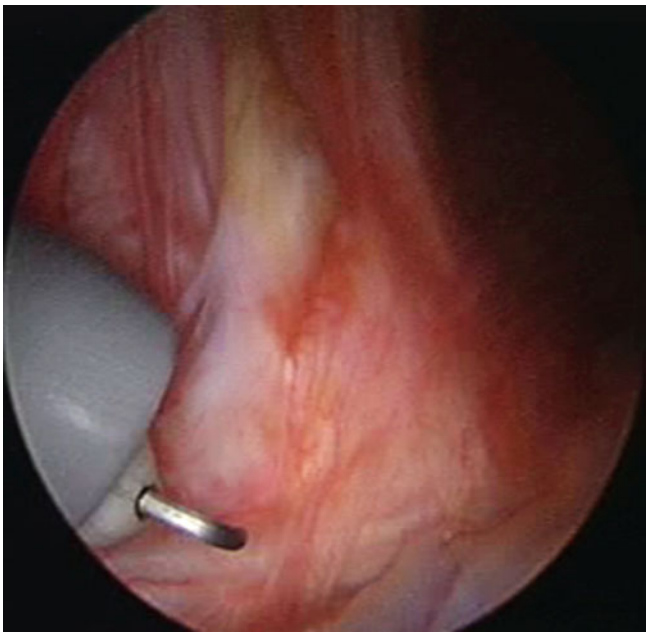

Fig. 32.4 The radio-frequency device introduced into the joint through the anterosuperior portal is used to perform arthroscopic release of the rotator interval

of the coracohumeral ligament (Fig. 32.4). Then we prepared to repair the PHAGL lesion. At beginning a mid glenoid anterior portal with an "ouside-in" technique, just above the subscapularis tendon fibers has been created. Through this portal a 8.25 mm cannula was inserted. This portal was used for suture management. At this point the scope was inserted through the anterosuperior portal, and in the posterior portal, we inserted the 5.5-mm cannula. With the scope in the anterosuperior portal, a shaver was inserted in the posterior cannula to perform a soft tissue debridement and light decortication of the remnant of the humeral insertion of the capsule and postero-inferior glenohumeral ligament on the posterior aspect of the humerus. Repair was performed by using a combination of side-to-side sutures and one suture anchor inserted posteriorly on the humeral neck. A biodegradable single-loaded suture anchor was inserted through the posterior portal at the most inferior aspect, close to the split of the capsule off the humeral head (Fig. 32.5). The detached capsule was repaired from inferior to superior by using a crescent suture hook and a Suture Shuttle Relay (ConMed Linvatec, Largo, FL, USA) (also a penetrator grasping device can be used according to the surgeon's preference and lesion as well). The capsular tissue was punched and penetrated at the more proximal edge (Fig. 32.6). The suture shuttle was passed through the tissue and retrieved out the mid-glenoid portal together with a limb of the suture coming from the anchor. Then we loaded the suture shuttle with the suture limb in the mid-glenoid portal and both were pulled back through the tissue and out the posterior portal. The second limb of the suture was retrieved out the anterior mid-glenoid portal, and the needle was passed through the capsule about 1 cm away from the first pass to create a stout bridge of soft tissue, the so-called Italian loop. This stitch can be useful to hold the capsule in patients with very flimsy tissue (Fig. 32.7) (when we use this stitch, the suture tails should be tied with a non-sliding knot. In cases a simple suture is made, a sliding-locking knot might be used to fix

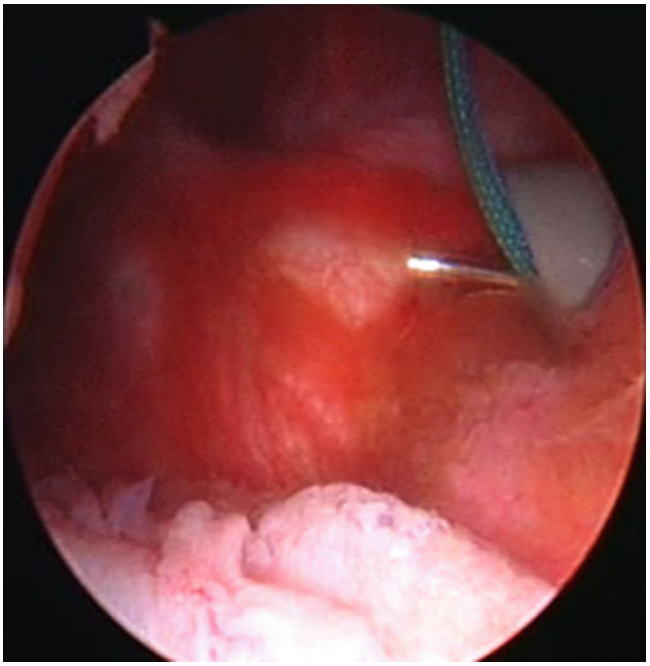

Fig. 32.6 The crescent suture hook is passed through the more proximal part of the detached posterior capsule

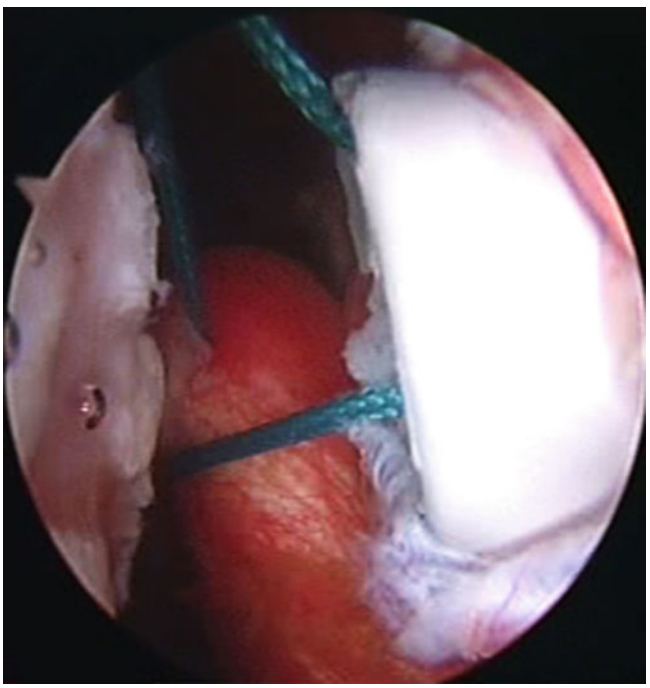

Fig. 32.7 Suture coming from the anchor is passed through the capsule and is ready to be tied to the humeral neck

the capsule to the humeral head). This was the first step of repair: to reattach the capsular edge to the bone (Fig. 32.8). To complete the repair, the horizontal split of capsular tissue was closed with side-to-side repairs using #2 nonabsorbable high-strength braided sutures. Side-to-side repair was accomplished with the same instruments (a crescent suture hook with a shuttle suture system) and managing the sutures through the mid-glenoid anterior portal.

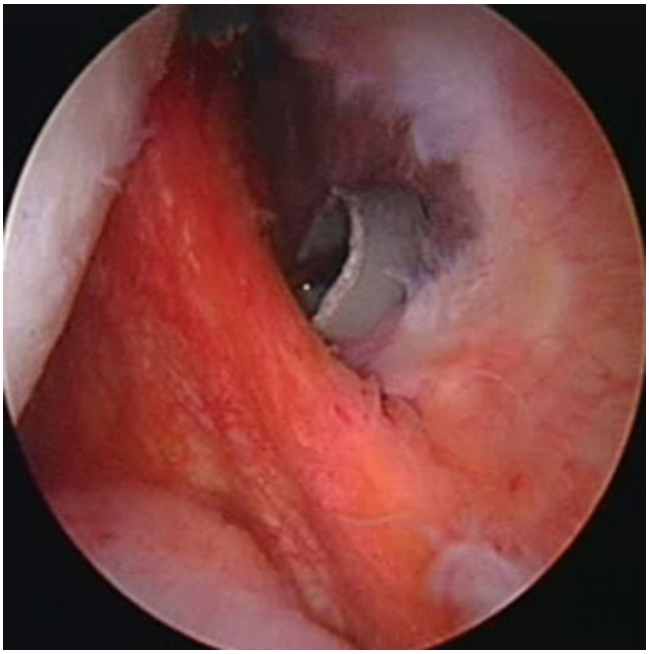

Fig. 32.8 Final view of the repair of the capsule to the bone. The observed residual horizontal split can be definitive closed by a side-to-side repair

Postoperative Care

After surgery, the patient was immobilized with the arm in a brace (Ultrasling II; Donjoy, DJO LLC, Vista, CA, USA) with 25° of abduction and 30° of external rotation for 4 weeks. Taking into account associated capsulitis, immediate supervised daily rehabilitation was started consisting of pendulum exercises and passive shoulder forward flexion, abduction, and external rotation in the scapular plane with the arm at side. Active scapular-stabilizing exercises were immediately started. No internal rotation was allowed for the first 6 weeks. Active shoulder exercises were started at 6 weeks postoperatively. At 8 weeks, patient progressed to resume full passive and active range of motion. Strengthening of scapular stabilizer and rotator cuff muscle with Thera-Band® progressive exercise program (Hygenic Corp., Akron, OH, USA) started at 10 weeks. At 6 months after surgery, the patient was allowed to proceed with all work and sports demands.

Literature Review

The risk of failure after primary surgical shoulder stabilization, both arthroscopic and open, with or without bone block procedure, is reported between 4 and 30 %, on considering

pain, stiffness, and recurrence of instability [1, 6–8]. Knowledge of the causes of failure after surgery is important not only to improve the outcome of surgery but also for the success of revision after failed repair.

Recurrence of instability is the main complication of arthroscopic stabilization of the shoulder. Failure rates after open and arthroscopic stabilization have been reported to range from 2 to 8 % and 4 to 13 %, respectively [26]. The risk factors associated with recurrence after repair should be evaluated by history, clinical examination, functional demand, previous surgical technique, and imaging. The young age of onset of instability, male sex, and long time elapsed between the first dislocation and surgery are predictive of failure after arthroscopic Bankart repair [27]. Excessive soft tissue laxity related to the pattern of collagen fibers, high level of activity, and limited compliance with the postoperative management can be the causative factors of high recurrence rate reported in this population [28].

Levine et al. [18] pointed out that the most frequent cause of unsuccessful shoulder stabilization is the failure to correct an excessively large anteroinferior capsular pouch associated with the detached capsulolabral complex. Malicky et al. [29] showed that there is a plastic (irreversible) strain of the anteroinferior capsulolabral complex associated with multiple episodes of shoulder subluxation or dislocation [29]. Furthermore, poor quality of the capsular can be related to multiple dislocations, previous surgery, or connective tissue disorders. A failed surgery can also be related to the incapacity to recognize and address combined superior or posterior capsular lesions [20].

Burkhart and De Beer [10] stressed the importance of reconstructing bony defects during arthroscopic procedures. According to their findings, arthroscopic Bankart repair can provide the same results in terms of recurrence as an open Bankart procedure in the absence of significant bony defects. Tauber et al. [30] showed that about 50 % of patients requiring revision surgery had a bony Bankart defect extending to the anteroinferior portion straight downward from the glenoid notch. Conversely, Boileau et al. [1] evaluated the factors influencing recurrence following arthroscopic Bankart repair and showed that avulsion fracture did not represent an identifiable risk factor.

It is important to assess the pattern of glenoid bone defect, in order to discriminate between loss of contour of the anterior glenoid related to bone erosion and glenoid avulsion fracture. Patients with an eroded anterior glenoid often have an associated attenuation of the anteroinferior capsulolabral complex. The deficiency of this structure allows recurrent subluxations or dislocation that contributes to erode the anterior glenoid. Several authors suggested that in case of bone deficiency <20 % of the glenoid width, an arthroscopic revision can be done with good outcomes, particularly in patients not involved in contact sports [31–33]. Mologne et al. [34] performed arthroscopic Bankart repairs in patients with bone defects equal to 25–30 % of the glenoid width and reported a failure rate of 14 %. They noted that all failures occurred in patients who had bony erosion where no bony fragment was identified. Improved results (92–93 % good to excellent) have been reported when a bony fragment was identified and incorporated into the repair, and healing was achieved in a near-anatomic position [35].

Recurrence rate after arthroscopic revision surgery has been reported ranging from 10 to 27 %, with authors reporting greater than 73 % of good to excellent results [36–38]. Several advantages of arthroscopic revision surgery have been advocated, including the ability to recognize and address the various soft tissue pathologies encountered, minimization of iatrogenic damage to the tissues (especially the subscapularis tendon), decreased pain, and cosmesis [8, 36]. Patient selection and surgical technique are crucial in optimizing success of arthroscopic revision surgery for shoulder instability. Emphasis has been put not only on anatomic repair of the capsulolabral lesions but also on adequate inferior and postero-inferior capsular plication to eliminate redundancy of the inferior capsular pouch [37].

A sufficient retensioning of the inferior glenohumeral ligament and use of an adequate number of anchors in the lower half of the glenoid is another key point for a successful arthroscopic repair. Boileau et al. [1] noted that at least four anchors (or sutures) should be used to obtain secure shoulder stabilization in primary surgery and in case of revision surgery sutures should be in number of seven [1]. Bedi et al. [39] established that in revision arthroscopic surgery a minimum of three double-loaded anchors should be used, and if a patulous rotator interval is noted upon completion of the revision repair, a rotator interval closure can be performed.

Open or arthroscopic revision surgery with bone augmentation should be performed to lengthen the glenoid arc when glenoid bone loss is greater than 25–30 % of the width or in the presence of an engaging Hill-Sachs lesion [10]. Also in cases of an irreparable HAGL lesion because of very retracted capsule, these surgical techniques represent a viable option. The most popular and studied technique is the coracoid transfer, either by Bristow or Latarjet procedure. Burkhart et al. [40] published their results of Latarjet reconstruction for shoulder instability in 102 patients with greater than 25 % loss of the inferior glenoid bone width (inverted pear configuration) and reported a 4.9 % recurrence rate. They concluded that Latarjet procedure can restore stability and function in more than 95 % of patients with significant bone defects [40].

The results of the Latarjet procedure as revision surgery are comparable to those reported for primary repair [41]. However, Gerber et al. [42] found that if recurrence is associated with chronic pain, the pain is likely to persist and compromise the subjective outcome after a Latarjet procedure. The authors

stated that patients have to be specifically evaluated for the presence and cause of pain before revision, and they need to be informed that pain may not be improved [42].

Stiffness is a rarely reported cause of failed instability surgery, especially following an arthroscopic stabilization. However, patients undergoing stabilization surgery for recurrent, traumatic anterior shoulder instability commonly have restricted passive external rotation [43]. Stiffness and substantial loss of external rotation can be the result of anterior capsular over-tightening or excessive plications used to close the rotator interval. Stiffness following subscapularis tightening procedures, especially with loss of external rotation, mainly occurs after open instability repair procedures [30]. Both open and arthroscopic Bankart repair can be complicated by postoperative stiffness. Despite the potential advantage over open repair, the incidence of postoperative stiffness following arthroscopic stabilization ranges from 2 to 15 % [44, 45]. When postoperative stiffness occurs, it is usually associated with prolonged immobilization after surgery, with overtightening of the capsule or non-anatomic capsulolabral repair. Castagna et al. [46] reported on a group of patients complaining stiffness and discomfort after open or arthroscopy anterior shoulder capsuloplasty or shrinkage for treatment of multidirectional instability (MDI). All these patients underwent an arthroscopic revision and in all cases a RHAGL lesion was found associated with stiffness [46].

Summary

Failed shoulder stabilization procedure is a significant challenge from a diagnostic, surgical, and patient's perspective. The causes of failure can be related to patient selection, indication, surgical technique, and postoperative compliance of patients to rehabilitation. Surgeon must have a clear understanding of the causes of failure through a systematic approach to the patient. In cases with recurrent instability without a significant bone loss and with a good-quality capsular tissue, arthroscopic procedure is a viable option. Patients with stiff and painful shoulder can also be treated with an arthroscopic procedure to treat at the same time capsulitis and associated lesions responsible for pain. Bone block arthroscopic or open procedures are reserved to cases with a significant glenoid bone loss, poor-quality capsular tissue, or irreparable avulsion of the anterior capsule from the humeral neck.

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HAGL and Reverse HAGL Lesions

33

Frank Martetschläger, James B. Ames,
and Peter J. Millett

Introduction

Tearing of the anteroinferior glenoid labrum (Bankart lesion) and of the posteroinferior glenoid labrum (reverse Bankart lesion) are well-documented, common injuries following traumatic shoulder subluxation or dislocation. The labral injury, as well as the loss of tension of the attached capsuloligamentous structures, is known to lead to recurrent instability [1].

Injuries to the capsuloligamentous attachments to the humerus are much less common but have recently gained attention due to advancements in arthroscopic experience and imaging techniques. In 1942, Nicola [2] first described an acute shoulder dislocation with avulsion of the anterior band of the inferior glenohumeral ligament (IGHL). In 1988, Bach et al. [3] described a humeral avulsion of the lateral capsule as a cause for recurrent shoulder dislocation. The term "humeral avulsion of glenohumeral ligaments (HAGL)," which is now commonly used for this pathology, was introduced by Wolf et al. [4] in 1995. Although the typical anterior HAGL lesion is more common, posterior injuries do occur. These are referred to as reverse or posterior HAGL (PHAGL) lesions and involve an avulsion of the posterior band of the IGHL from the humeral neck. While rare, these lesions have been shown to contribute to recurrent instability [5, 6].

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Epidemiology

HAGL and PHAGL lesions typically occur in patients with shoulder instability and are often seen in combination with other pathologies such as Bankart and posterior Bankart lesions, respectively [5, 7–9]. The patient's history typically involves high-energy trauma [10, 11]; however, repetitive microtrauma in overhead or throwing athletes has also been reported as a potential cause for this pathology [13, 14]. The incidence of HAGL lesions has been reported to approach 10 % [4, 14] in patients with shoulder instability, rising to nearly18% in patients needing revision procedures [14]. Bokor et al. [14] reported an incidence of nearly 40 % in patients with anterior instability that did not have a distinct Bankart lesion. Therefore, in the absence of a Bankart lesion in an individual with recurrent instability, suspicion for a HAGL lesion should be elevated.

The PHAGL lesion has been reported in patients with posterior instability [5, 15, 16]. Although not well quantified by the scientific literature to date, the incidence of PHAGL lesions is presumed to be much less common than that of HAGL lesions.

Pathophysiology

Stabilization of the humerus is achieved through the concomitant actions of various static and dynamic structures surrounding the joint which ultimately serve to maximize surface contact of the humeral head on the glenoid surface and to prevent anteroposterior translation. Static components include the labrum, tendons, and capsular ligaments, while dynamic components represent muscle contraction, scapulothoracic motion, and, potentially, proprioception [17].

The capsuloligamentous complex includes the coracohumeral ligament (CHL), superior glenohumeral ligament (SGHL), the middle glenohumeral ligament (MGHL), and the inferior glenohumeral ligament (IGHL) complex (Fig. 33.1). The primary static stabilizer preventing

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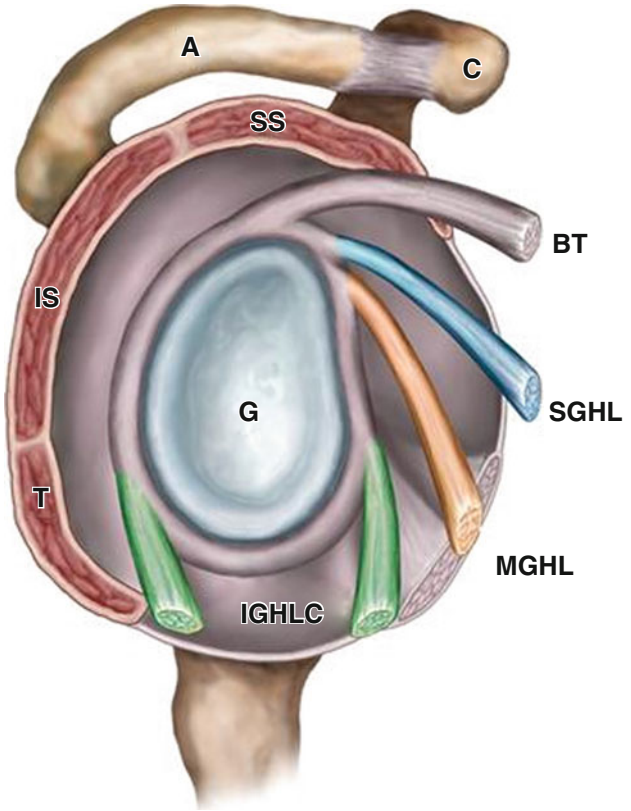

Fig. 33.1 Glenohumeral ligament anatomy. Note the anterior and posterior bands of the IGHL complex with interconnected axillary pouch. *A* acromion; *C* coracoid process; *BT* biceps tendon; *SS* supraspinatus; *IS* infraspinatus; *T* teres minor; *G* glenoid cavity. (Reprinted with permission from American Roentgen Ray Society: Modarresi et al. [33])

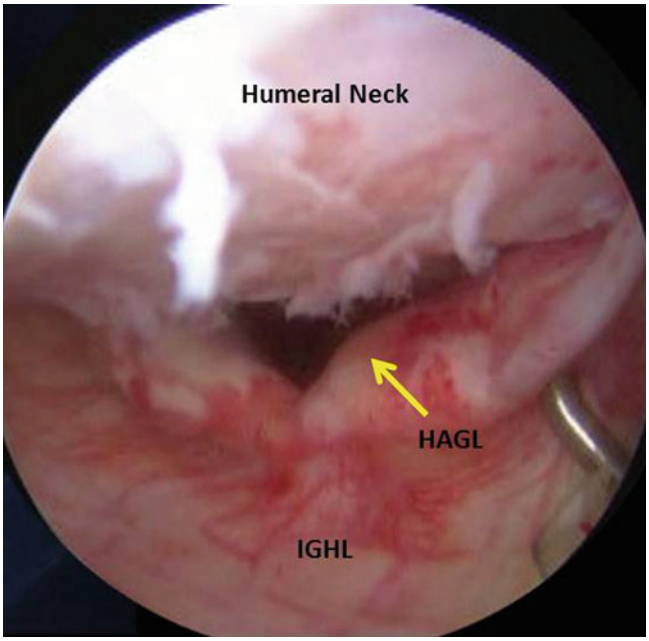

Fig. 33.2 Arthroscopic visualization of HAGL lesion. Note bleeding and fraying of avulsed IGHL

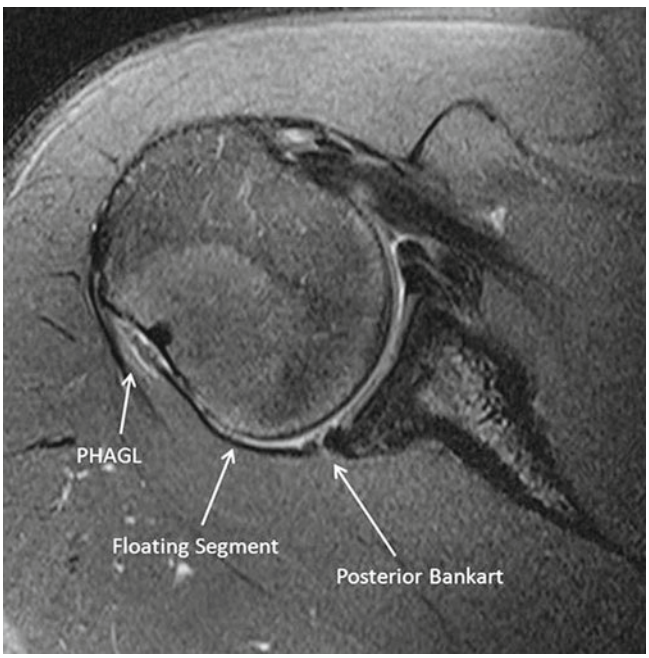

Fig. 33.3 Floating posterior IGHL (*PIGHL*) lesion in a 19-year-old rugby player after a forceful hyperabduction and hyperextension injury. The posterior Bankart lesion propagated superiorly, resulting in a concomitant mild superior labral anterior to posterior (SLAP) lesion

anteroposterior motion is the IGHL complex, which is known to change shape with different arm positions. This plasticity allows for static stabilization of the humeral head in multiple positions, preventing anterior or posterior translation [18, 19, 21].

The IGHL complex consists of an anterior and posterior band along with an interconnecting axillary pouch, which together create a "hammock-like" structure [18–20]. The humeral insertion of the IGHL has been described as "collar-like," with its attachment close to the articular margin, or "V-shaped," with its apex near the cartilaginous rim of the humerus and its base further distal on the humeral metaphysis [6, 18, 20–22]. Assuming a right shoulder, the anterior band originates between 2 and 4 o'clock and the posterior band between 7 and 9 o'clock, both arising primarily from the labrum [22]. Ticker at al. reported the humeral insertion of the IGHL to be distal to the lesser tuberosity anteriorly and distal to the greater tuberosity posteriorly [23].

Traumatic anterior dislocation or subluxation of the humeral head can result in avulsion of the IGHL from either the anteroinferior glenoid (i.e., Bankart lesion) or at its humeral insertion (HAGL lesion) (Fig. 33.2). Although both injuries are the result of arm hyperabduction, it has been noted that HAGL often has an external rotation component [19]. Unidirectional posterior instability can result in reverse lesions such as a posterior Bankart lesion, PHAGL lesion, or a combination of each (floating PIGHL) (Figs. 33.3, 33.4, and 33.5) [5].

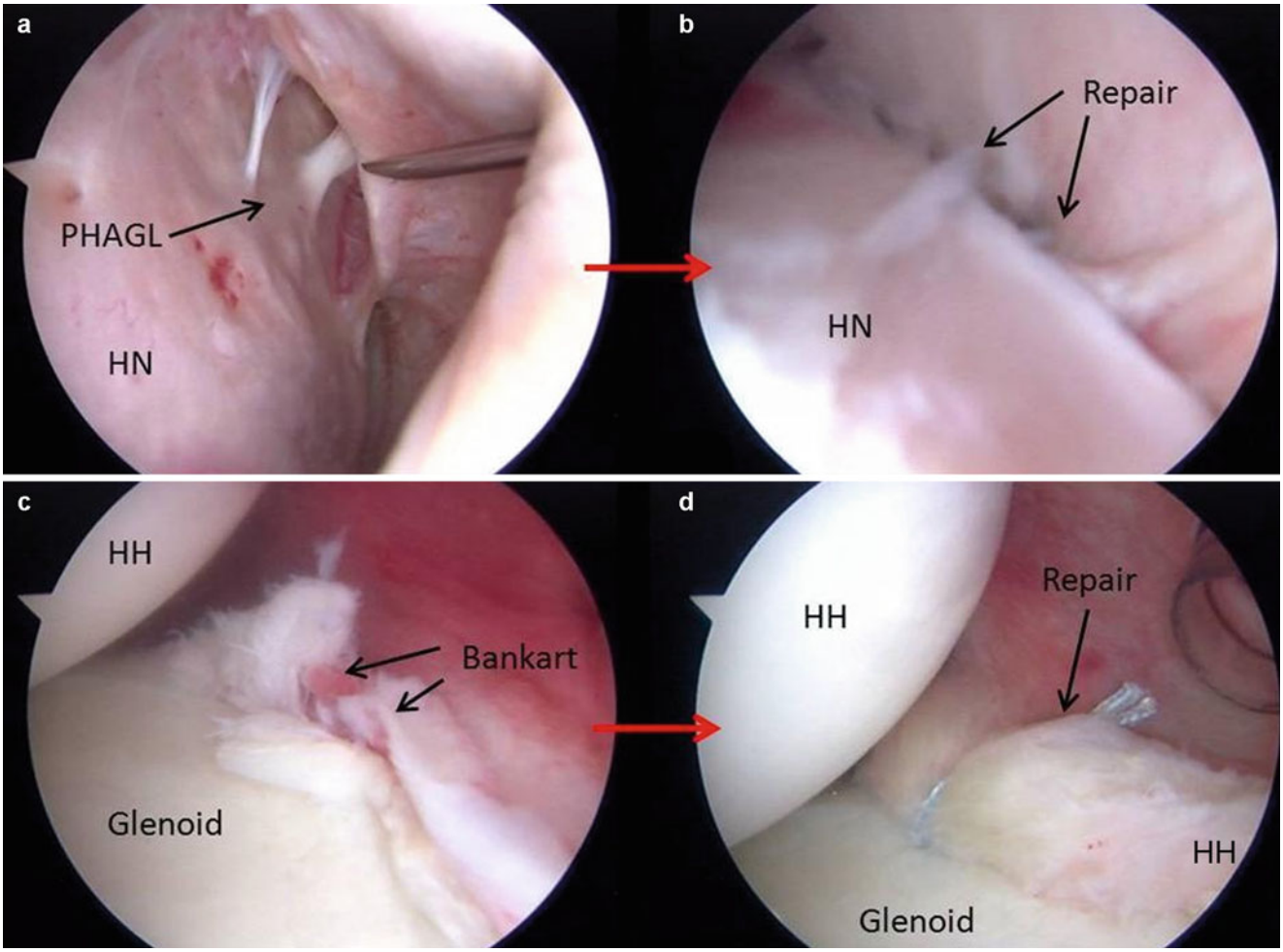

Fig. 33.4 Visualization and repair of a floating PIGHL lesion in a 19-year-old rugby player. Avulsion of the IGHL complex from the humeral neck (*HN*) (**a**) followed by repair (**b**). An associated posterior Bankart lesion (**c**) was also present in this patient and was repaired (**d**), *HH* humeral head

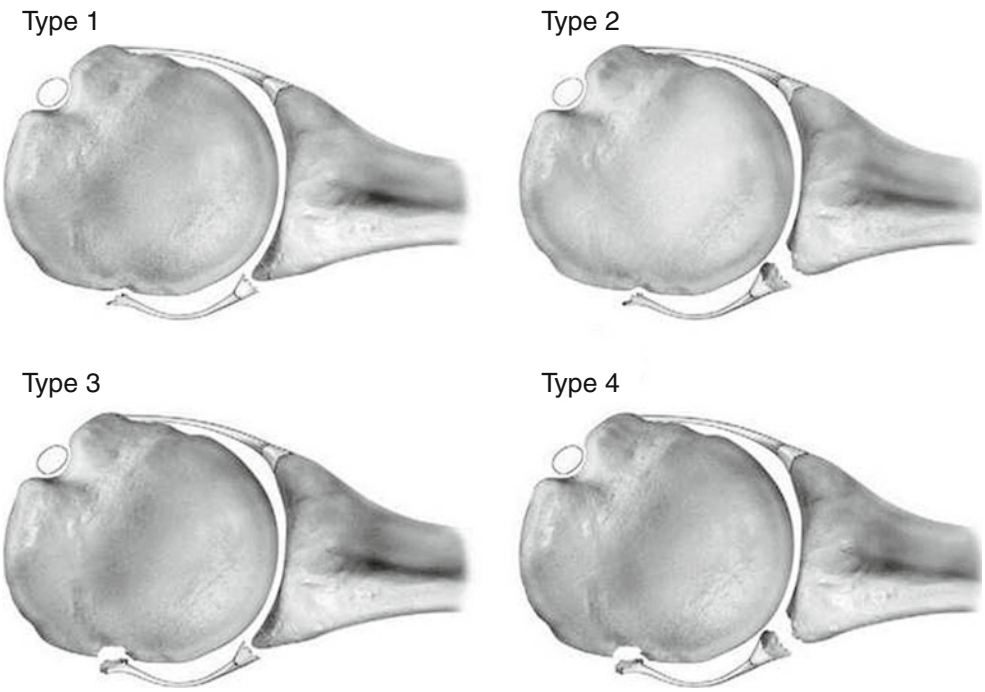

Fig. 33.5 Floating PIGHL subtypes as described by Ames and Millett [5]. Type 1 represents a PHAGL with a concomitant posterior Bankart lesion. Type 2 is a PHAGL with a posterior bony Bankart lesion. Type 3 is a bony PHAGL with a posterior Bankart lesion. Type 4 is a bony PHAGL with a posterior bony Bankart lesion (Reprinted with permission from the *Journal of Bone and Joint Surgery* [5])

Table 33.1 Classification of HAGL lesions according to Bui-Mansfield et al. [6]

Anterior	Posterior
Anterior humeral avulsion of the glenohumeral ligament (AHAGL)	Reverse humeral avulsion of the glenohumeral ligament (PHAGL)
Anterior bony humeral avulsion of the glenohumeral ligament (ABHAGL)	Posterior bony humeral avulsion of the glenohumeral ligament (PBHAGL)
Floating AHAGL	Floating PHAGL

Bui-Mansfield et al. [6] created the West Point classification system for describing anterior and posterior injuries to the IGHL in 2007. In this classification, there are six types of lesions broken into two separate categories depending on the direction of instability (Table 33.1).

History

Careful, detailed history taking is critical to making the diagnosis of HAGL. Patients typically have nonspecific shoulder complaints with a typical history of previous dislocation or subluxation, although this is not always present. Position of the arm at the time of injury is important in that hyperabduction with external rotation is often the mechanism in HAGL lesions while external rotation is not always necessary for a simple Bankart lesion. Furthermore, the direction of instability and history of recurrent instability are important questions to be evaluated. Recurrent instability in a patient with a previous Bankart repair may be a clue that a missed HAGL lesion is present. Also, a patient with recurrent instability in the absence of a Bankart lesion should raise suspicion of an injury to the IGHL complex at its humeral attachment (HAGL or PHAGL).

Clinical Examination

Nonspecific findings in the clinical exam are typical, but it is important to rule out other sources of shoulder pathology before considering the diagnosis of HAGL or PHAGL. Therefore, a complete and thorough examination of active and passive range of motion and strength in forward flexion, abduction, adduction, external rotation, and internal rotation should be determined, beginning with the asymptomatic shoulder. It is especially important to evaluate subscapularis function and strength since a tear of this tendon is often associated with a HAGL lesion.

Since HAGL and PHAGL lesions are associated with shoulder instability, the following provocative maneuvers are performed bilaterally: the load and shift test, the jerk test, the anterior and posterior apprehension test, and the relocation test. Hyperlaxity together with multidirectional instability is also assessed bilaterally and the rotator interval is checked for a sulcus sign. Although these tests are indicative of instability, none are sensitive or specific for the detection of HAGL or PHAGL lesions.

Imaging

Diagnostic imaging for the detection of HAGL and PHAGL lesions has improved in recent years, principally due to better recognition and also treatment of HAGL and PHAGL pathologies. True AP films should be obtained in neutral and internal rotation to identify possible fractures of the greater and lesser tuberosities. Scapular Y films are obtained to evaluate glenohumeral alignment. Axillary views are also obtained to identify lesions of the humeral head (such as a Hill-Sachs lesion) and any corresponding glenoid pathology. An interruption of the subchondral sclerosis line on AP films or abnormal glenoid anatomy or version on axial films may be indicative of chronic instability. A Garth view may reveal "scalloping" of the medial aspect of the surgical neck in cases of HAGL [24] or as a fleck of bone inferior to the anatomic neck of the humerus in bony HAGL lesions [22, 25].

Magnetic resonance imaging (MRI) with or without intra-articular contrast is the imaging modality of choice in cases of suspected HAGL or PHAGL lesions. Coronal oblique or sagittal oblique, T-2, fat-suppressed images are most likely to reveal the diagnosis; however, axial images are also useful (Fig. 33.3) [22]. It is important to note that the axillary pouch contains fluid which typically creates a characteristic U shape on the coronal or sagittal oblique MRIs. Loss of this shape due to contrast or fluid extravasation indicates the presence of a lesion in the IGHL complex [22, 24]. In the acute post-traumatic setting, blood in the joint provides excellent contrast and obviates the need for gadolinium enhancement. Chronic HAGL lesions are difficult to visualize because the IGHL has typically scarred back to its attachment on the surgical neck of the humerus and may or may not result in clinical instability or findings on MRI [17].

Treatment: Indications and Contraindications

Nonsurgical management is typically advocated when the injury to the IGHL complex is intra-substance and does not result in detachment from the humerus. In these cases, care

must be taken to identify any concomitant injuries [6]. Strengthening of the rotator cuff and surrounding musculature is helpful to prevent recurrent instability. Detached lesions are initially managed nonsurgically with physical therapy and range of motion exercises. The incidence of recurrent instability in patients with a HAGL lesion is unknown; however, preliminary evidence suggests that it may be an indication for operative repair. Surgical management of HAGL lesions is most often reserved for high-functioning individuals who desire a return to work or sports or those with recurrent instability. Although outcome data is limited to small case series and reports, some data suggests that arthroscopic repair and open repair result in similar, satisfactory outcomes [7, 12, 14, 26]. The contraindications for surgical repair are similar to those of any open or arthroscopic shoulder surgery.

Decision-Making Algorithm

As mentioned, history, physical examination, and imaging studies are used appropriately to establish the correct diagnosis. Typically, patients with HAGL lesions are initially treated nonsurgically since the rate of recurrent instability resulting from HAGL lesions is unknown. In cases of failed nonsurgical treatment with recurrent instability, ongoing pain, or impairment of shoulder function, surgical repair is indicated [17]. If a HAGL lesion is diagnosed as a concomitant injury during arthroscopy, a repair of the IGHL complex to the humeral insertion site should be performed to minimize the risk of failure and recurrent instability leading to revision surgery.

Clinical Case/Example

A 19-year-old collegiate rugby player suffered a traumatic subluxation of his right shoulder during a rugby match after attempting to tackle an opposing player. The arm was forcefully abducted and extended immediately prior to the injury. Although he denied the incidence of frank dislocation, he did describe subluxation with spontaneous reduction. The patient also reported that he had shoulder discomfort earlier in the match for which he did not seek medical treatment. Prior to the match, the patient was completely asymptomatic without a history of injury or trauma.

Upon presentation, the patient reported feelings of instability, weakness, and vague pain. His pain was 8/10 at its worst and was exacerbated by overhead activities and relieved with rest. He denied neck pain, elbow pain, paresthesias, or any other injuries.

Initial physical examination of the shoulder revealed tenderness to palpation at the posterior shoulder and lateral brachium, while the coracoid, AC, and SC joints were nontender. Active and passive range of motion was adequate. Neurovascular examination was within normal limits. Global rotator cuff function and strength was normal. He did have mildly positive Neer and Hawkins signs with a positive O'Brien's test. Apprehension, relocation, and sulcus signs (in neutral and external rotation) were also negative.

Radiographs of the affected shoulder revealed no bony lesions, while MRI indicated an avulsion of the posterior IGHL complex off the humeral neck (PHAGL) along with a posterior labral lesion extending superiorly near the insertion of the biceps tendon. This combination of injuries resulted on a floating posterior IGHL (Fig. 33.3).

Discussion of the risks, benefits, and alternatives of each therapy modality was undertaken, and largely due to the nature of his sport, the patient decided to undergo PHAGL and posterior Bankart repair.

Exam under anesthesia revealed grade 2 posterior translation, grade 1 anterior translation, and a mild sulcus sign (<1 cm). Diagnostic arthroscopy revealed a mild SLAP lesion and a floating PIGHL (combined posterior Bankart and PHAGL) which was subsequently repaired as described below (Fig. 33.4).

Postoperatively, the shoulder was immobilized in abduction for the first 4 weeks at which point pendulum and passive motion exercises were begun. Active and assisted motion began 6 weeks postoperatively with an avoidance of posterior loading for approximately 12 weeks. Sixteen weeks postoperatively, the patient was cleared to return to sporting activities with encouragement to continue his strengthening regimen. No further problems have been reported by the patient to date.

Arthroscopic Treatment: Surgical Technique

Although reverse HAGL lesions often require an accessory posteroinferior portal for suture passage, the arthroscopic surgical technique for both lesions is similar. The step-by-step technique for repair of HAGL is given below, followed by a description of repair of PHAGL lesions.

Patient Positioning

In the operating suite, the patient is positioned in either a lateral decubitus or beach-chair position. The lateral decubitus position affords improved visualization of the inferior capsule, while the beach-chair position has mainly been adopted for surgeon comfort. Positioning of the patient is ultimately determined by the surgeon as there is no specific data indicating a difference in outcome between the two positions in the repair of HAGL or PHAGL.

The affected arm is placed in a traction device or pneumatic arm holder with 20° of forward flexion and 50° of

abduction. This positioning modifies tension on the inferior capsule such that accurate reattachment of the IGHL to the humeral neck can be achieved.

A bump, made of two of three small towels, is placed under the axilla, inferior and posterior to the inferior angle of the scapula. This configuration allows adequate access to the axillary pouch of the IGHL complex.

Portals

Although a two- or three-portal technique can be undertaken depending on surgeon experience, the three-portal technique is described here.

An axillary pouch portal of Bhatia [27] is first established 2–3 cm inferior to the inferior border of the posterolateral acromial angle and 2 cm lateral to the position of a standard posterior portal. After marking, an incision is made such that the portal is angled 30° medial in the axial plane and slightly inferior to the sagittal plane. Using an 18-guage spinal needle, an outside-in technique is used in order to prevent injury to the axillary nerve and the posterior capsule during cannula insertion.

An anteroinferior portal at 5-o'clock is also established using an outside-in technique with the shoulder in neutral position. The incision is made 1 cm inferior to the superior border of the subscapularis tendon at the 5-o'clock position relative to the leading edge of the glenoid [17].

A posteroinferior portal is then established at the 7-o'clock position, as described by Davidson and Rivenburgh [28]. The incision is made 2–3 cm inferior to a standard posterior viewing portal using outside-in needle localization technique. The function of this portal is for viewing during instrumentation.

Another portal may be established in the rotator interval for suture management. The use of this fourth portal is at the choice of the surgeon.

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

After the appropriate portals have been established, the entire humeral head, glenoid surface, and IGHL complex must be examined for concurrent lesions. It is critical to view the entire attachment of the IGHL to the humeral neck. The literature describes several cases of missed HAGL lesions due to a lack of visualization of the entire IGHL attachment. Missed HAGL lesions, as described above, can result in recurrent instability, further surgery, and patient dissatisfaction. It is also pertinent to visualize the muscular striations of the subscapularis through the IGHL defect such that damage is to be avoided.

Step-by-Step Procedure (Box 33.1)

At this point, it is important to keep the shoulder in a neutral position such that the capsuloligamentous structures of the joint are sufficiently lax to afford accurate and precise relocation of the anterior band to the humeral neck. An arthroscopic burr is used to abrade the surface of the humeral neck at the precise location of desired reattachment of the anterior band. Suture anchors are then placed at this location and the resulting loose suture is retrieved using the designated 5-o'clock suture management cannula. The suture is advanced from the 5-o'clock portal through the IGHL using a desired suture-passing device. Horizontal mattress sutures are then used to reattach the capsular tissue back to the surgical neck of the humerus. After tying, the performance of anterior and posterior arthroscopic drawer tests is essential to confirm appropriate tension of the joint capsule and subsequent stability.

Box 33.1: Tips and Tricks

The arthroscopic HAGL repair can be challenging even for the experienced arthroscopist. The following tips can help facilitate the procedure and avoid severe complications:

- Make sure you are familiar with the instruments and implants needed for this procedure.
- Use spinal needles to assure correct portal placement and proper working angles.
- Allow enough time for a thorough diagnostic arthroscopy to prevent missing an important lesion.
- Avoid overtightening the capsular complex as this may lead to impaired shoulder motion.
- If using the beach-chair position, use your assistant to help distract the humerus anterolaterally to improve visualization.

Reverse HAGL

This procedure is largely similar to the repair of the anterior HAGL with a few exceptions. The same 5-o'clock, 7-o'clock, and axillary portals are established. An additional posteroinferior portal for suture passage is often necessary in posterior lesions. It is vitally important to avoid damage to the posterior capsule with the arthroscopic cannula. The posterior humeral neck is prepared with an arthroscopic burr at the desired location of reinsertion of the posterior band. Suture anchors are placed at the reinsertion site and horizontal mattress sutures are advanced through the posterior band. The IGHL is then reduced to the humeral neck. It is extremely important to avoid overtightening of the posterior capsule as this can lead to dramatic limitations of internal

rotation postoperatively, potentially decreasing patient satisfaction and function.

Postoperative Care

The postoperative course for patients with HAGL and PHAGL is similar. The patient is placed in a shoulder immobilizer with an abduction pillow for approximately 6 weeks. Physical therapy is initiated at a point 3 weeks after surgery, focusing on progressive passive range of motion. It is important to avoid anterior loading of the IGHL complex in HAGL and posterior loading in PHAGL so as not to compromise the surgical repair. Active range of motion exercise is begun at 6 weeks after surgery followed by rotator cuff, deltoid, trapezius, and biceps strengthening at 8 weeks.

Literature Review

The work of Nicola [2] in 1942 first described anterior shoulder dislocation with avulsion of the anterior band of the IGHL. Wolf et al. [4] followed by referring to the lesion as humeral avulsion of the glenohumeral ligament (HAGL) in 1995. They described an open repair through a deltopectoral approach with detachment of the subscapularis tendon in two patients and an arthroscopic repair in four patients. Excellent clinical results with a follow-up of at least 36 months were reported in all six patients with free shoulder motion and return to sports.

A review of the literature indicates that anterior HAGL lesions can be treated successfully with an open arthroscopic surgery [3, 4, 14, 29–31]. Arciero and Mazzocca [26] proposed a mini-open technique for the repair of HAGL lesions since the all-arthroscopic repair was considered extremely difficult due to limited exposure along the anteroinferior pouch of the humeral neck. For this technique, the lower third of the subscapularis is incised and the tendon is then lifted up exposing the humeral ligament avulsion. Excellent initial results have been reported in 8 patients without recurrent instability or subscapularis weakness. The advantages of the all-arthroscopic repair of a HAGL lesion include direct identification of the lesion itself as well as concomitant injuries, minimization of soft tissue trauma, avoidance of subscapularis detachment, and less postoperative pain [24].

In 2007, Castagna et al. [32] reported on 9 cases with PHAGL lesions treated arthroscopically. In all patients, the diagnosis was not made preoperatively, exemplifying the difficulty in clinical diagnosis of these lesions. After a mean follow-up of 34 months, all patients were free of pain and symptoms and were able to perform all activities of daily living and resume the same sports activities with same frequency. Recently, the pathoanatomic variants of floating PHAGL lesions were described by Ames and Millett [5]. Arthroscopic treatment of a PHAGL with a concurrent posterior osseous Bankart lesion was described along with a new, four-part subclassification system for floating PHAGL lesions (Fig. 33.5).

Summary

HAGL lesions are relatively uncommon and most reports on this pathology are limited to small case series. However, its incidence may approach up to 10 % in instability cases [4] and an undiagnosed HAGL lesion can lead to recurrent instability and failure of surgical treatment [7, 14]. Therefore, a thorough history and clinical examination along with the appropriate imaging studies are necessary for correct diagnosis. While an MRI can make the diagnosis of a HAGL lesion, in many cases the diagnosis is made during arthroscopy and direct visualization [32]. Arthroscopic treatment of HAGL and PHAGL has shown to yield good clinical outcomes [4, 5, 31, 32]; however, the literature is lacking any prospective comparative reports on specific treatment strategies for these lesions. Given the decreased morbidity of the arthroscopic approach, it is the author's recommendation that, when technical skill is available, these lesions be treated arthroscopically.

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Arthroscopic Treatment of Bony Bankart Lesions

34

Hiroyuki Sugaya

Epidemiology

Glenoid anterior rim fractures, accompanied by acute glenohumeral dislocation and subluxation with tremendous amount of external force [1], usually result in persistent instability of the glenohumeral joint [2]. According to a three-dimensionally reconstructed computed tomography (3D-CT) study, the prevalence of anterior glenoid bony lesion has been reported as high as 90 % in shoulders with chronic recurrent traumatic anterior instability and an associated bony fragment is present in about half of shoulders with anterior glenoid bony lesion [3]. Further, bone loss in shoulders associated with a bony fragment is relatively significant compared to that in shoulders with attritional glenoid without bony fragment [3, 4].

Pathophysiology

In shoulders with bony Bankart lesion, bony fragment is firmly attached to the labrum because the majority of the anterior glenoid rim fractures are avulsion type fracture [4–6]. It is widely recognized that in acute cases, such glenoid fractures with a large fragment [7] and/or displacement of more than 10 mm [8] and associated instability [2] require immediate surgical fragment reduction and fixation using screws [7] or suture anchors [6, 9, 10], either open or arthroscopically. On the other hand, in shoulders with recurrent instability, surgeons need to consider entire glenohumeral ligament pathology, such as capsular lesions or elongation of the capsule itself, in addition to the bone loss [1]. Glenoid bone loss tends to be significant when medium to large bony fragment is present [3, 4]. Therefore, many surgeons tend to ignore the fragment and prefer to perform the coracoid transfer [11, 12], which is an invasive and non-anatomical procedure. However, since these bony fragments are proved to be viable even in chronic stage [13], these shoulders are favorable candidates for arthroscopic bony Bankart repair associated with capsular tensioning of the entire glenohumeral ligament [4, 5, 14, 15].

History

The diagnosis of recurrent traumatic anterior glenohumeral instability is usually made easily on the basis of the history of distinct dislocation or subluxation and the positive apprehension sign. However, when we see collision athletes, care should be taken because they may not experience clear dislocation or subluxation and only complain of pain or weakness when they bring their arm to maximum external rotation in abduction [14, 15].

Clinical Examination

The *anterior apprehension test* is done with the patient in the supine position. In this test, the shoulder is moved passively into maximum external rotation with the arm at the side; 30, 60, 90, 120, and 150° of abduction; and maximum flexion [14, 15].

The *posterior apprehension test* is done with the arm at maximum internal rotation in 90° of abduction. The feeling of apprehension is reported in each arm position. However, the most important and reliable physical examination can usually be done with the patient under anesthesia, comparing stability testing to the contralateral shoulder.

Imaging

Radiographic images are sometimes helpful in detecting the Hill-Sachs lesion and the anterior glenoid rim lesion, especially during the first patient visit. Bernageau described a unique

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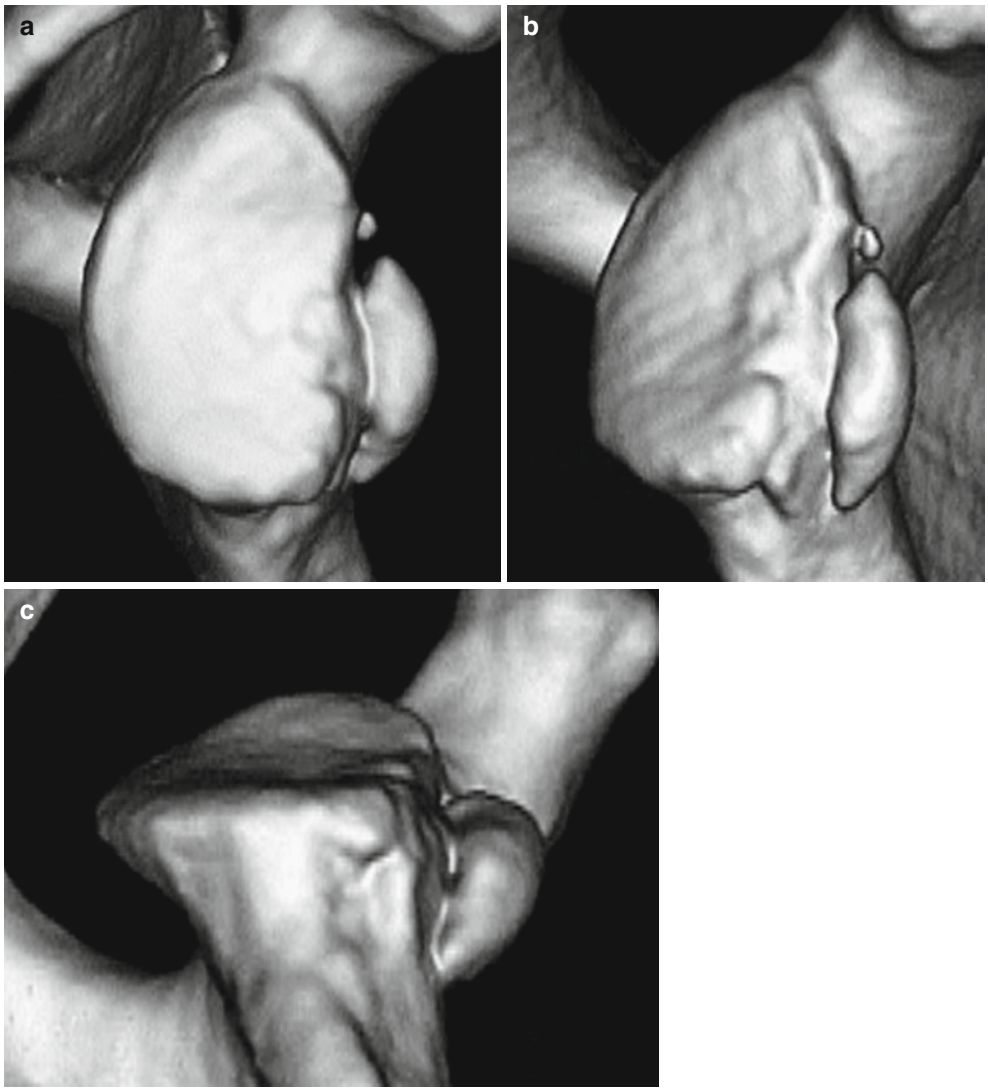


Fig. 34.1 A bony Bankart lesion in the 3D-CT images. En face view (**a**), oblique view (**b**), and inferior view (**c**) of 3DCT

method for detecting an anterior glenoid rim lesion with the patients in the standing position [16, 17]. However, this technique requires fluoroscopic control in order to obtain optimal diagnosable images, and therefore, radiation exposure is a relevant issue [8]. We have developed a modified Bernageau method with the patient lying on her/his axilla in the most relaxed position [14, 15]. With this method, clear X-ray images can be obtained more easily with a high probability of ascertaining bony pathology without using fluoroscopic imaging [14, 15].

MRI provides only limited information for shoulder instability. However, MR arthrography (MRA) is helpful in detecting a soft tissue lesion such as Bankart lesion, capsular pathology, and/or humeral avulsion of the glenohumeral ligament (HAGL lesion). Nonetheless, the final diagnosis of soft tissue pathology can be made most accurately through diagnostic arthroscopy.

3D-CT is the most important imaging study in order to assess glenoid morphology accurately [3]. In a shoulder with bony Bankart lesion, detecting accurate configuration of the bony fragment during surgery is not necessarily easy because the bone fragment is covered by the surrounding soft tissue. Through preoperative 3D-CT, surgeons can assess the size and shape of the bony fragment in shoulders with a bony Bankart lesion (Fig. 34.1) [3, 4, 18].

Treatment: Indications and Contraindications

Surgery is indicated for all patients with a bony Bankart lesion who want to recover a stable shoulder. Patients with uncontrolled epilepsy or who are noncompliant are not candidates for arthroscopic stabilization.

Clinical Case/Example

A 24-year-old male snowboarder suffered right shoulder dislocation 3 years ago when he fell on his right arm. Since then, he suffered repeated dislocations five times during the

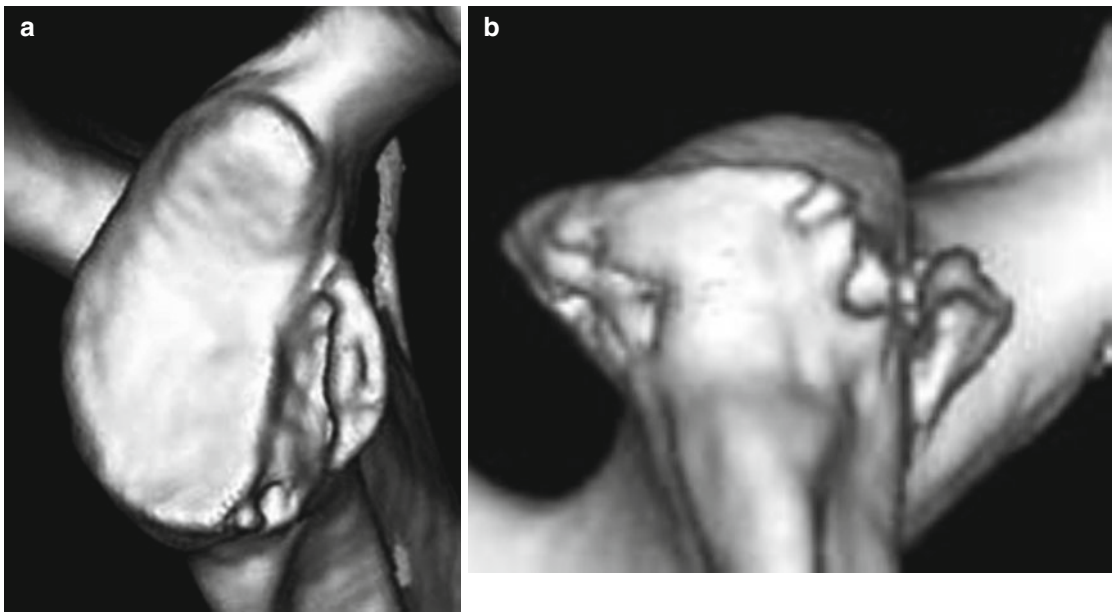


Fig. 34.2 3D-CT images of a bony Bankart lesion seen in 24-year-old male snowboarder. (**a**) "En face" view. (**b**) Inferior view

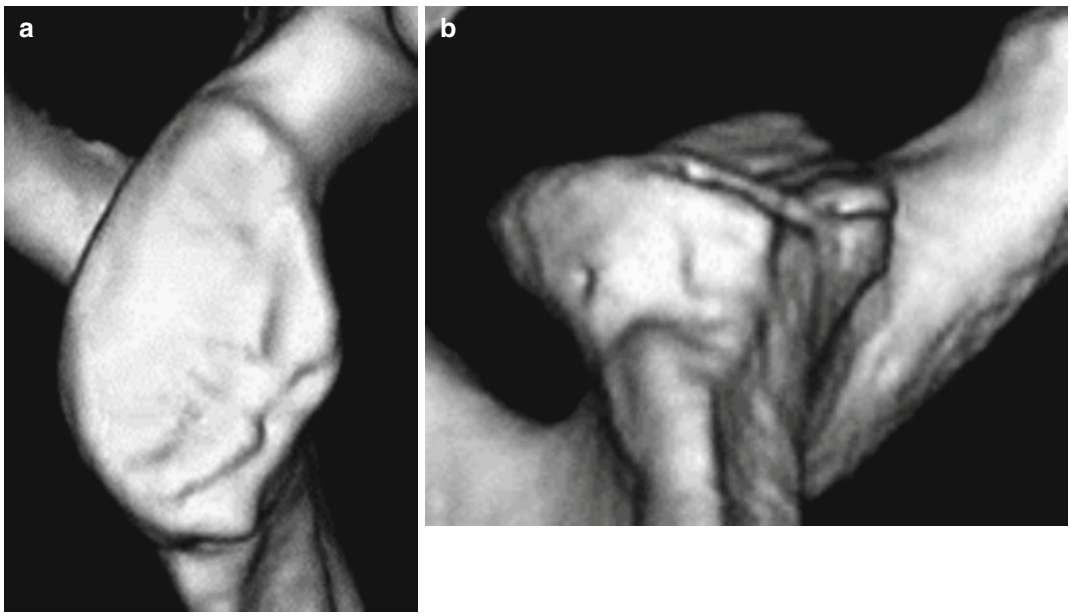


Fig. 34.3 3D-CT images 5 years after arthroscopic bony Bankart repair. The glenoid morphology is almost normalized. (**a**) "En face" view. (**b**) Inferior view

past 3 years. 3D-CT demonstrated distinct bony Bankart lesion with medium-sized fragment (Fig. 34.2) and MRA demonstrated Bankart lesion and redundant inferior glenohumeral ligament without evident capsular tears. The patient underwent arthroscopic bony Bankart repair and resumed stable shoulder and returned competitive snowboarding 6 months after surgical stabilization. Five years later, he was paged for follow-up and stated that there had been no complaint regarding his right shoulder without any recurrence and 3D-CT demonstrated perfect bony union and remodelling of the glenoid (Fig. 34.3).

Arthroscopic Treatment: Surgical Technique

Regardless of the severity of glenoid bone loss, arthroscopic bony Bankart repair is indicated if a bone fragment is present on 3D-CT [4, 5, 19]. Since the majority of shoulders with a

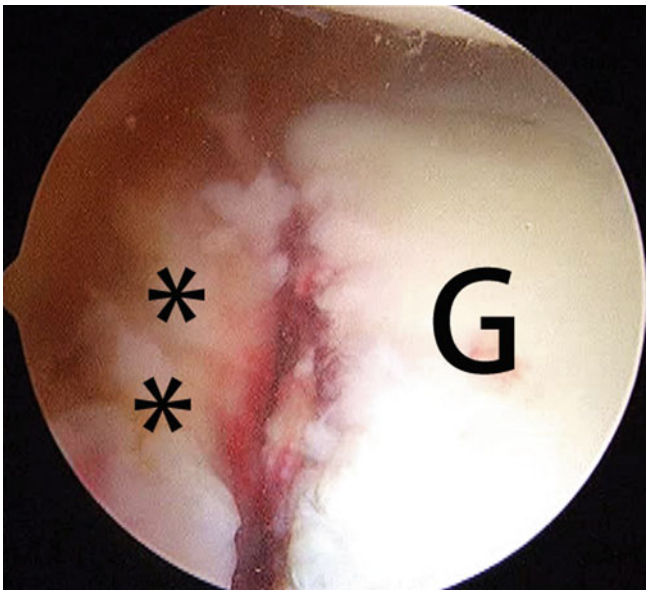


Fig. 34.4 Arthroscopic appearance of a bony Bankart lesion viewed from the anterior portal. The *asterisk* indicates the bony fragment embedded in the surrounding soft tissue (*G* glenoid)

large glenoid bone loss retains bony fragment at the anteroinferior glenoid neck [14, 15], this procedure is applicable to most shoulders with glenoid bone loss. Normally, in shoulders with bony Bankart lesion, the fragment is medially displaced and partly united to the glenoid neck, and also the fragment is firmly connected to the adjacent labrum or soft tissue (Fig. 34.4). Therefore, the majority of bony fragment associated with a bony Bankart lesion can be easily separated from the glenoid neck using standard straight or curved rasps. The gap between the fragment and original glenoid is well demarcated in most shoulders; if otherwise, careful palpation or preoperative 3D-CT greatly helps surgeons to delineate the gap [14, 15].

Patient Positioning

All patients are seated in the beach-chair position under general anesthesia and joint laxity is assessed by examination of both shoulders prior to surgical intervention.

Portals

A 4-mm arthroscope is introduced through a standard posterior portal and a diagnostic arthroscopy is performed. Then an anterior portal is created just superior to the subscapularis tendon and just lateral to the conjoined tendon using an outside-in technique, in order to facilitate instrument insertion without cannulas [20]. In addition, an anterosuperior portal is established at the anterosuperior margin of the rotator interval utilizing an outside-in technique. This becomes the second working portal. In shoulders with superior labral detachment, a lateral acromial portal, established just lateral to the midpoint of the acromion through the muscle-tendon junction of the infraspinatus, is used instead of the anterosuperior portal.

Diagnostic arthroscopy is first performed from a standard posterior portal. Then, arthroscope is switched to the anterior portal to evaluate capsular integrity and confirm bony Bankart lesion.

Step-by-Step Procedure (Box 34.1)

Box 34.1: Tips and Tricks

- Clearly define the gap between the fragment and the glenoid neck; otherwise, you may break the fragment or native glenoid during mobilization of the fragment along with the capsulolabral complex. In order to avoid this:
  - Recognize the size, shape, and location of the bony fragment using preoperative 3D-CT prior to surgery.
  - Delineate the gap using a radio-frequency instrument before inserting a rasp.
- Use penetrating instrument properly when penetrating the bony fragment; otherwise, you cannot penetrate the fragment nicely or may break the instruments. In order to avoid this:
  - Reduce and stabilize a bony fragment by grasping the labrum adjacent to the superior portion of the fragment with a grasper introduced from the anterosuperior portal.
  - Aim the blade of the bone penetrating tools perpendicular to the fragment.
  - After catching the bony fragment by the tip of the penetrating instrument, push the fragment to the neck of the glenoid, and then penetrate it by rotating the bone penetrator with a force perpendicular to the glenoid neck.

Mobilization of the Complex

After diagnostic arthroscopy from the anterior portal, arthroscope is again switched to the posterior portal. Then, separation and mobilization of the labroligamentous complex together with the bony fragment from the glenoid neck is performed using an elevator, straight and curved rasps, scissors, shavers, and a radio-frequency probe. All of these instrument tools are inserted through a cannulaless anterior

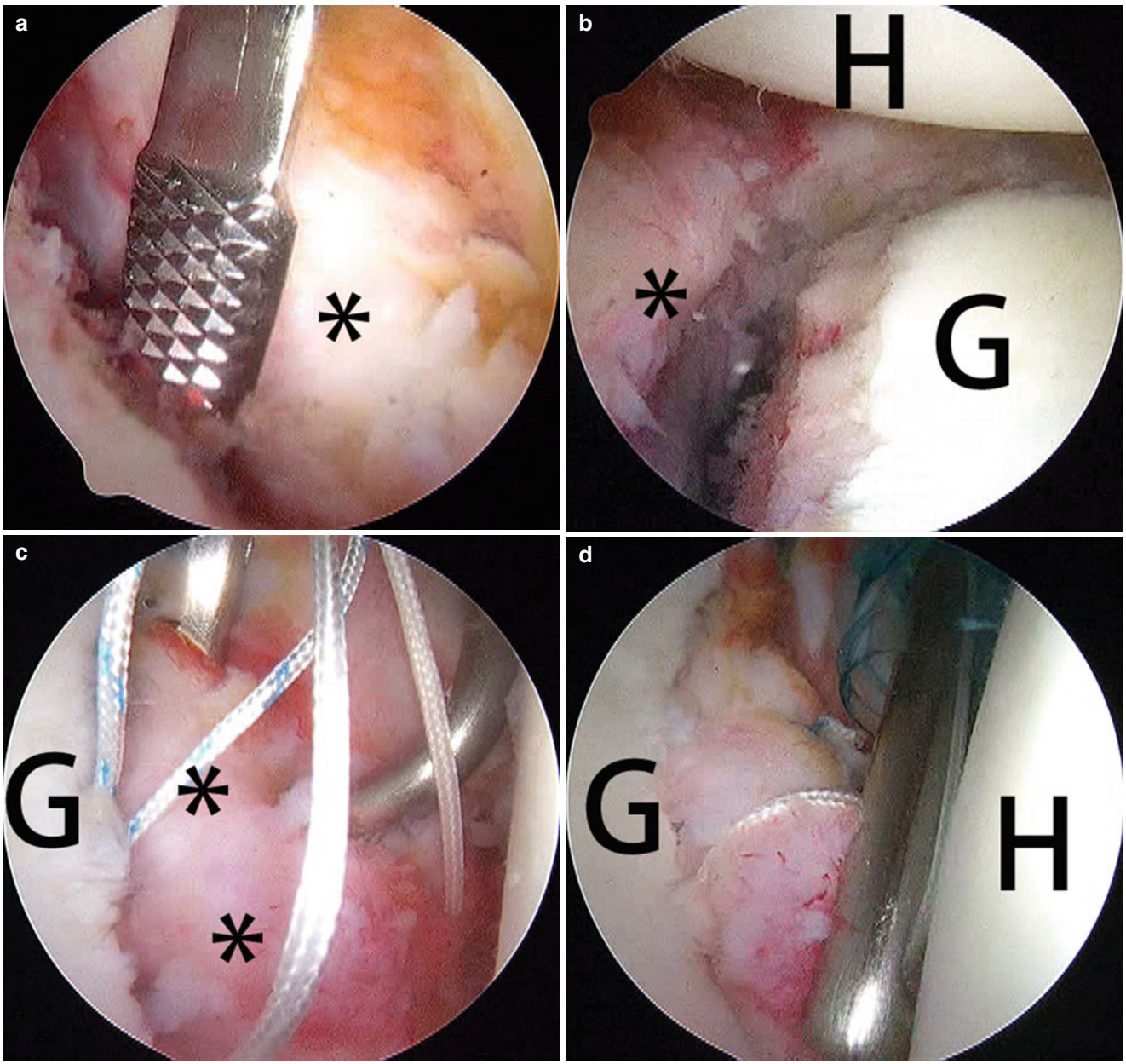


Fig. 34.5 Surgical procedures. (**a**) Bony fragment and adjacent labrum is separated from the glenoid neck using a rasp. (**b**) Arthroscopic view after complete separation and mobilization of the complex, viewing from the anterior portal. Articular cartilage at the margin of the inferior glenoid face was removed. (**c**) A grasper inserted through the anterosuperior portal stabilizes the bony fragment by pulling the adjacent labrum and a bone penetrating instrument inserted though the anterior portal is trying to penetrate the fragment through the surrounding soft tissue. (**d**) Knot tying after suture placement to the fragment (the *asterisk* indicates the bony fragment embedded in the surrounding soft tissue. *H* humeral head, *G* glenoid)

portal. This step is a vital part of this procedure. First, a straight rasp is inserted from the anterior portal and is placed in the small gap between the fragment and the glenoid neck. Then, the gap is expanded by tapping the handle of the rasp (Fig. 34.5a). After separating the fragment from the glenoid neck, the mobilization of the labroligamentous complex is performed up to the 7:30 position in the right shoulder until the complex and the fragment become completely free in exactly the same way as one would mobilize a Bankart lesion without a bony fragment using the instruments previously described. Once mobilization of the fragment and the complex is completed, preparation of the glenoid is performed by removing scar tissues from the glenoid neck and exposing the bony surface using a shaver and an abrader. Further, articular cartilage on the edge of the glenoid is also removed to promote tissue healing after repair (Fig. 34.5b). Normally,

the separation of the fragment from the neck can be readily accomplished using only elevators and rasps. If the separation of the fragment is difficult and the fragment is united firmly, a small-sized chisel can be introduced from the anterior portal to separate it from the glenoid neck.

Repair of Inferior Labrum Adjacent to the Osseous Fragment

The following procedure is very important in order to obtain optimal fragment reduction and provide proper tension to the inferior glenohumeral ligament. The first suture anchor loaded with #2 high-strength suture is inserted on the surface of the glenoid at the 6 o'clock position using a drill guide introduced through the cannulaless anterior portal. Because this portal has no cannula, the angle of approach of the guide can be adjusted easily allowing optimization of the angle to the glenoid [13]. After the first anchor insertion, a looped #2-0 nylon suture is placed into the labrum at the 6:30 position using a low-profile 7-mm Caspari Punch™ (Conmed Linvatec, Largo, FL, USA) or a Suture Hook™ (Conmed Linvatec). A suture relay is then performed intra-articularly [13]. The second anchor is inserted into the face of the glenoid at the 4:40 position, followed by the suture placement in the labrum adjacent to the inferior side of the bony fragment using the same technique. After completion of the suture placement of the two inferior anchors, knot tying is performed using a self-locking sliding knot through a cannula inserted through the anterior portal. To accomplish secure knot tying, the complex, together with the fragment, is held upward and laterally on the glenoid surface by a grasper introduced through the anterosuperior portal to reduce tensile force on the suture.

Osseous Fragment and Superior Labrum Repair

The next step is the suturing of the osseous fragment itself, either by passing the suture through the fragment or by penetrating it using bone penetrating tools such as a Bone Stitcher™ (Smith & Nephew, Andover, MA, USA), which is an originally developed bone penetrator with a stiff shaft and large handle (Fig. 34.6), or by passing suture around the fragment using a Suture Hook™ or Suture Leader™ (DePuy Mitek, Raynham, MA, USA) and Bone Stitcher™ [5, 14, 15]. It is very important to characterize the fragment shape and size preoperatively by 3D-CT evaluation to decide whether passing through or passing around the fragment is most appropriate [5, 14, 15]. This procedure is facilitated when the bony fragment is reduced and stabilized by grasping the labrum adjacent to the superior portion of the fragment with a grasper introduced from the anterosuperior portal (Fig. 34.5c). Although the number of suture anchors utilized is dependent on the size and shape of the osseous fragment, normally one or two suture anchors are used for stabilizing the bony fragment [5, 14, 15]. Knot tying is performed after placing the sutures through the fragment (Fig. 34.5d). The final step is to suture the labrum adjacent to the superior side of fragment to augment the stability of the entire complex (Fig. 34.7). Normally four suture anchors with simple sutures are used to reconstruct the entire labroligamentous complex (Fig. 34.8).

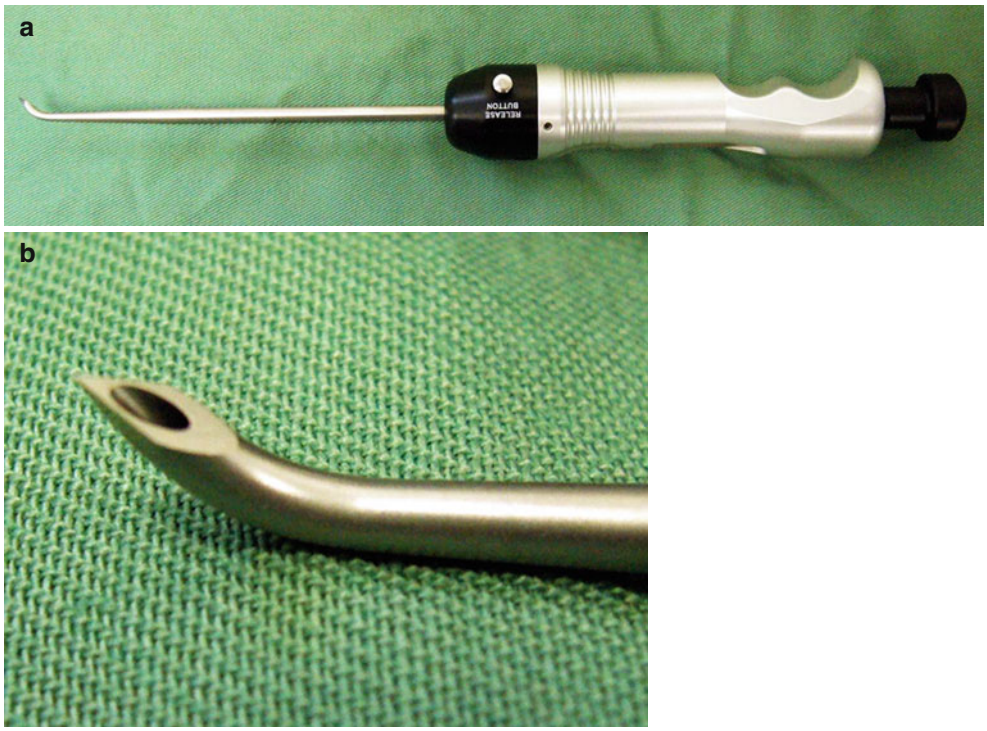


Fig. 34.6 Bone Stitcher™ (Smith & Nephew, Andover, MA). (**a**) This device has a large handle and a very sharp tip (**b**) with obturator

Management of the Associated Pathology

In shoulders with a capsular tear, a capsular repair utilizing two to three side-to-side stitches is performed prior to the bony Bankart repair. Furthermore, in shoulders with a superior labral detachment, arthroscopic reattachment is performed, after the Bony Bankart repair is completed, utilizing a lateral acromial portal instead of the anterosuperior portal.

Repair Augmentation

The rotator interval closure and/or Hill-Sachs Remplissage [21] is performed as an augmentation in patients with relatively high-risk shoulders, such as contact athletes, young and lax individuals, and those with a large Hill-Sachs lesion. In those patients, the rotator interval is closed by suturing the superior margin of the subscapularis tendon to the superior glenohumeral ligament with the arm held at the side and in maximum external rotation using #2 high-strength sutures [14, 15, 22].

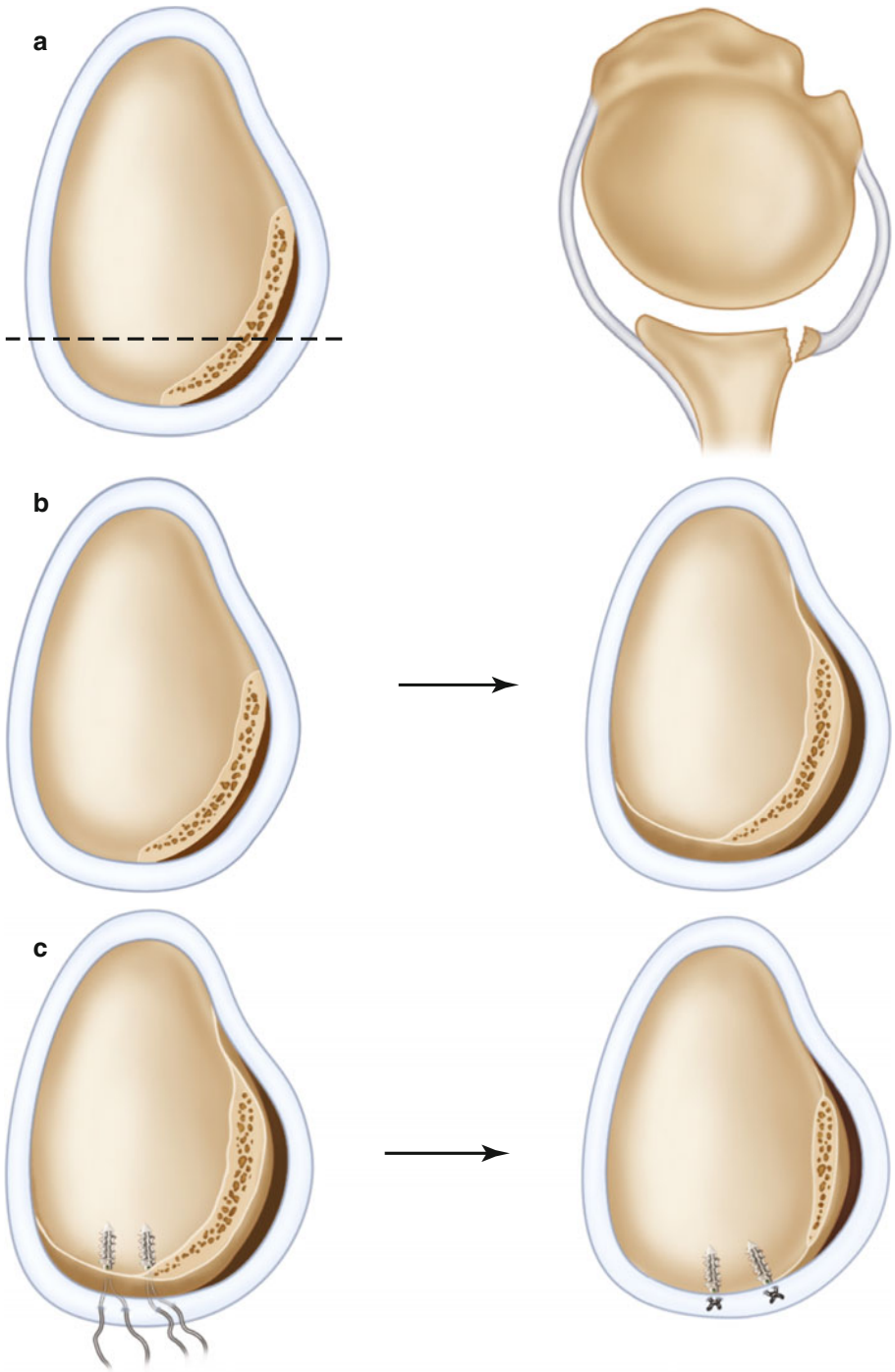
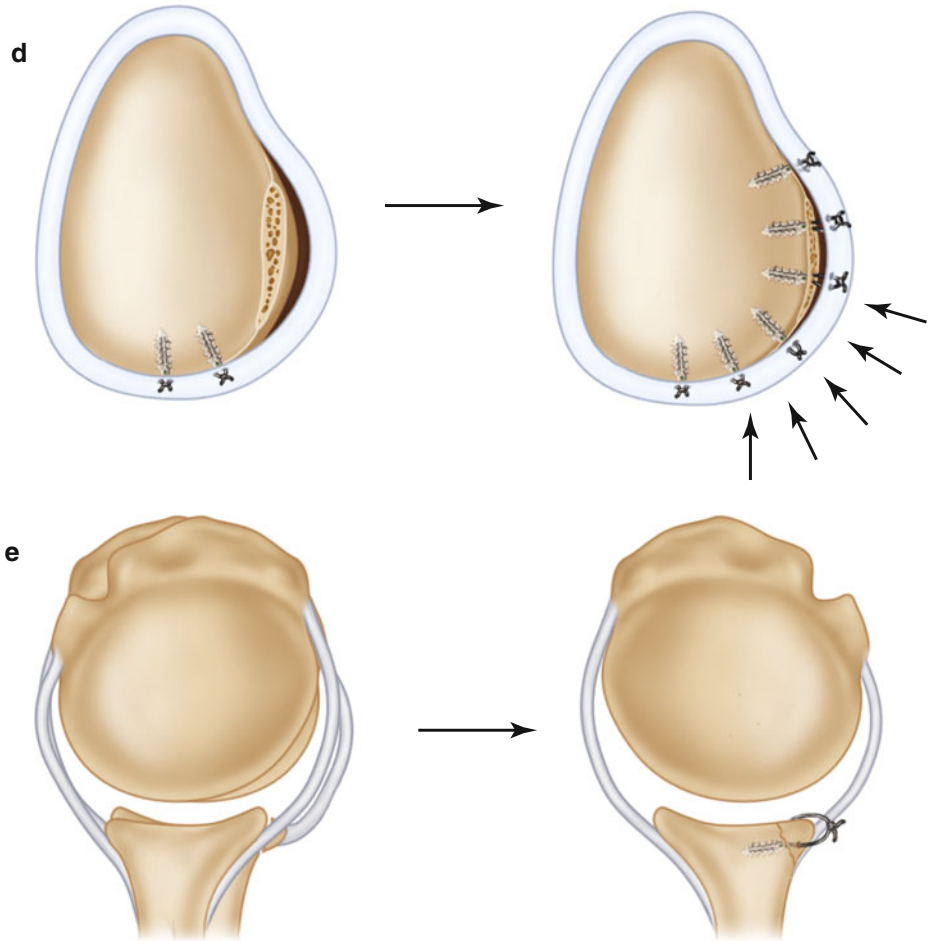


Fig. 34.7 Schematic drawings of entire surgical procedures. (**a**) In shoulders with bony Bankart lesion, the fragment is normally medially displaced and partly united to the glenoid neck, and also the fragment is firmly connected to the adjacent labrum or soft tissue (the *dotted line* indicates the plane of the axial section). (**b**) After separation of the fragment and labrum from the glenoid neck, the mobilization of the labroligamentous complex is performed up to the 7:30 position in the right shoulder until the complex and the fragment become completely free. In addition, articular cartilage on the edge of the glenoid is also removed. (**c**) Two suture anchors are inserted to the face of the inferior glenoid and the inferior labrum was first reduced. Thanks to this procedure, the bony fragment was automatically brought upward, and therefore, handling of the fragment becomes easier. (**d**) Bony fragment is stabilized by pulling the adjacent labrum with a grasper inserted through the anterosuperior portal. Then, a bone penetrating instrument is inserted though the anterior portal and sutures are placed to the fragment. (**e**) Knot tying provides not just fragment reduction but proper tensioning to the entire inferior glenohumeral ligament (The *gray area* on the glenoid indicates the area where articular cartilage is removed. The *dark area* on the labrum side indicates a bony fragment inside the soft tissue.)

Fig. 34.7 (continued)



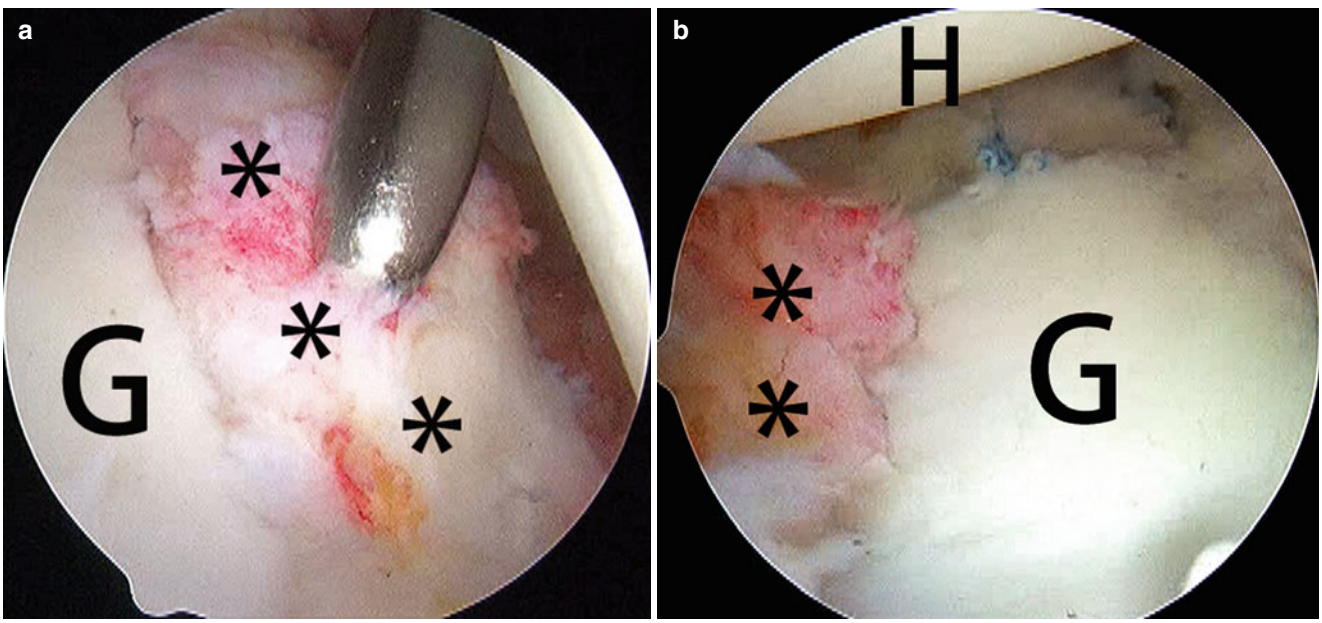


Fig. 34.8 Arthroscopic appearance after completing bony Bankart repair, viewing from the posterior portal (**a**) and the anterior portal (**b**) (The *asterisk* indicates the bony fragment embedded in the surrounding soft tissue. *H* humeral head, *G* glenoid)

Postoperative Care

The shoulder is immobilized for 3 weeks using a sling (Ultra Sling II, Donjoy, Carlsbad, CA). After immobilization, passive and active-assisted exercises are initiated for forward flexion and external rotation avoiding pain. After 6 weeks, patients begin strengthening exercises of the rotator cuff and scapular stabilizers. Three months after the operation, they are permitted to practice noncontact sports. Full return to throwing or contact sports is allowed after 6 months according to each individual's functional recovery. Excessive mechanical stress to the reconstructed site within 3 months after surgery may cause anchor/suture failure. In order to avoid this, patients should be instructed to not be too active until 3 months after surgery.

Summary

Prevalence of a bony Bankart lesion is as high as 50 % in recurrent anterior glenohumeral instability and the most of shoulders with large glenoid defect retains a bony fragment at the anteroinferior glenoid neck. In addition, since a bony Bankart lesion is acute or chronic avulsion type glenoid rim fracture, normally the fragment and labrum junction is intact even in chronic cases. Therefore, although sometimes technically demanding, arthroscopic bony Bankart repair is technically feasible regardless of fragment or glenoid defect size by incorporating the fragment into labrum and soft tissue repair. This technique can obviate the need for bone grafting in patients with a large glenoid defect and a bony fragment.

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Management of Hill-Sachs Lesions

35

Michael J. O'Brien and Felix H. Savoie III

Introduction

The Hill-Sachs defect is a compression fracture of the posterolateral humeral head that is associated with anterior shoulder dislocation or recurrent instability. At the time of the shoulder dislocation, the soft cancellous bone of the humeral head is impressed against the hard cortical bone of the anterior glenoid rim, creating a compression fracture in the humeral head. This can be visualized on an internal rotation radiograph of the shoulder, as first described by Hill and Sachs in 1940 [1].

Larger lesions with advanced bone loss are more likely to engage, resulting in shoulder instability at lower arm abduction angles. If these lesions are not addressed at the time of soft tissue labral repair, a higher failure rate and recurrent instability will ensue.

The arthroscopic management of these defects includes transfer of the infraspinatus and posterior capsule into the defect, known as the "remplissage" procedure, or bone grafting of the defect with allograft humeral head. Arthroscopic remplissage is a safe, effective technique for the management of humeral head bone loss at the time of surgical stabilization.

Epidemiology

The true incidence of Hill-Sachs lesions is unknown. While reported to occur between 40 and 90 % with an initial dislocation event [2–5], the incidence may be high as 100 % with recurrent instability [5].

Reverse Hill-Sachs lesions are located on the anterosuperior humeral head and are associated with posterior shoulder dislocations. The incidence of this type is difficult to quantify, as posterior dislocations are much less common, although the reverse lesion may occur in up to 86 % of posterior instability cases [4]. Posterior shoulder dislocations are uncommon, and reverse Hill-Sachs lesions are rare.

Pathophysiology

Hill-Sachs lesions most commonly occur during anterior glenohumeral instability episodes. The shoulder typically is in an abducted, externally rotated position. As the humeral head is forced anteriorly, the capsule, glenohumeral ligaments, and glenoid labrum are stretched and likely torn. As the humeral head translates farther anteriorly, a compression fracture occurs along the posterior-superior-lateral aspect of the humeral head as it impacts the anterior glenoid rim. In cases of recurrent anterior shoulder instability, the static restraints to glenohumeral translation (capsuloligamentous structures and labrum) become increasingly attenuated. This makes it easier for the relatively softer cancellous bone of the humeral head to sustain continued damage as it makes repeated contact with the harder cortical bone of the anterior glenoid rim.

The impression fracture is likely small with the first-time dislocation and may be missed on initial radiographs. With each subsequent dislocation, the compression fracture enlarges, becoming more evident on follow-up radiographs. A growing body of literature supports the notion that recurrent instability of the shoulder leads to progressive bone loss, both on the glenoid and the humeral head. Bone loss on either side of the joint may lead to recurrent instability at lower arm abduction angles and feelings of instability with activities of daily living. Shoulder dislocations can occur with less force, such as while sleeping at night or with the arm adducted at the side. A substantial amount of literature describes the well-established relationship between anterior glenoid bone loss and recurrent instability [6–9]. However,

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little published data exists on the role and management of bone defects on the humeral side [10].

Hill-Sachs lesions are typically described as engaging or non-engaging. An engaging Hill-Sachs lesion, as described by Palmer and Widen [11] and Burkhart and De Beer [6], occurs when the humeral head defect engages the rim of the glenoid while the shoulder is in a position of athletic function, with 90° of abduction and 0–135° of external rotation. In an engaging lesion, the long axis of the humeral head defect is oriented parallel to, and engages with, the anterior glenoid rim when the shoulder is in abduction and external rotation. When the defect is not parallel with the rim of the glenoid and thus does not engage with it in a position of function, the lesion is referred to as non-engaging [6].

Hill-Sachs lesions rarely occur in isolation. They most frequently occur in conjunction with an anterior capsulolabral avulsion (Bankart lesion) [3]. Other common coexisting injuries are humeral avulsion (HAGL) and mid-capsular tears, floating anterior capsule, anterior glenohumeral ligamentous pathology, and glenoid bone loss (i.e., bony Bankart lesion) [12]. Anteroinferior glenoid bone loss may ultimately become large enough to create a glenoid with the appearance of an inverted pear [6], which is associated with recurrent anterior shoulder instability. Optimal management requires close evaluation because bone loss in the shoulder is frequently a bipolar phenomenon that results in failure of surgery aimed at correction of the soft tissue defects alone.

History

The patient with a Hill-Sachs defect will uniformly have a history of shoulder instability, whether a single dislocation or recurrent instability events. It is important to inquire about the initial dislocation event, the mechanism of injury, and the position of the arm (i.e., abduction and external rotation) and if the shoulder self-reduced or required reduction with sedation. Inquiry should also be made regarding the number of subsequent dislocations and the ease with which the shoulder dislocates and reduces.

The ease of shoulder dislocation, number of dislocation events, and arm position all provide information on the stability of the shoulder. Shoulders that dislocate easily with activities of daily living and those that dislocate in lower arm abduction angles are more likely to have bone loss, both on the anterior glenoid and humeral head. The patient with glenoid bone loss or an engaging Hill-Sachs defect may describe a sensation of "catching" as the humeral head falls outside the glenoid track during arm movement. The patient may actually describe that "the shoulder falls out" when leaning forward during activities. These scenarios should lead the examiner to suspect bone loss and, when corroborated with physical examination, may lead the examiner to order advanced imaging of the shoulder.

Clinical Examination

Examination of the shoulder begins with inspection. The overall posture of the patient should be noted (i.e., sitting up straight or slouching forward), as well as the attitude of the shoulder and whether the shoulder and upper arm are held in a position of protraction or retraction. Fullness in the anterior shoulder may represent an anterior dislocation, while an arm fixed in internal rotation may represent a locked posterior dislocation. Musculature should be inspected for atrophy, specifically the deltoid and supraspinatus fossa. Assessment can be made for generalized ligamentous laxity. The shoulder should be palpated and any tender spots noted. Tenderness over the acromioclavicular joint may indicate acromioclavicular separation, and coracoid process tenderness may indicate tightness of the pectoralis minor and shoulder protraction.

Both active and passive range of motion (ROM) should be determined. During passive ROM, care should be taken to observe any apprehension by the patient, especially as the arm approaches the position of abduction and external rotation. The examiner places one hand on the patient's shoulder to stabilize the scapula, while grasping the patient's forearm with the other hand. Beginning with the patient's arm adducted to the side, the arm is externally rotated and brought into increasing abduction angles. With an unstable shoulder, apprehension will be evident in the position of abduction and external rotation. Crepitus may be palpable in this position with a labral tear and anterior shoulder instability. Apprehension and instability at lower arm abduction angles indicate severe glenohumeral instability and raise the suspicion of bone loss.

Rotator cuff strength must be assessed, especially in patients over the age of 40. In this patient population, shoulder instability may result in subscapularis rupture or avulsion of the supraspinatus and infraspinatus. Proximal biceps lesions may also occur, resulting in positive biceps provocative maneuvers.

Provocative maneuvers may reproduce a patient's pain and confirm the diagnosis of instability. The load and shift test can be performed either supine or sitting upright. If supine, the scapula is stabilized by the exam table; if sitting, the scapula is stabilized by the examiner's hand. The humeral head is then translated across the glenohumeral joint; crepitus and pain are indicative of a glenoid labral tear. The jerk test and posterior load and shift test evaluate for posterior

glenohumeral instability. Instability can be further confirmed clinically by the Jobe's apprehension and relocation tests.

Imaging

Several imaging studies can be useful for the diagnosis of glenohumeral instability with bone loss. Imaging always begins with a full series of shoulder radiographs, including an anteroposterior (AP) radiograph of the glenohumeral joint, a scapular Y or outlet view, and an axillary view. Irregularities on initial radiographs may raise the suspicion of bone loss. Bone loss on the anteroinferior glenoid can be evident on the AP and axillary projections by loss of the normal bony contour of the anterior glenoid. Bone loss on the posterolateral humeral head, as seen with a Hill-Sachs defect, can be visualized on an AP radiograph with the arm in internal rotation. Several other radiographs are useful in the evaluation of bone loss specifically. The West Point axillary view is very useful in the evaluation of glenoid bone loss; the Stryker Notch view is specific in evaluating for Hill-Sachs defects [13] and is particularly useful because the internal rotation of the humeral head brings the posterolateral bone defect into view.

Computed tomography (CT) is a superior imaging modality for evaluating bone loss. Three-dimensional CT, in particular, helps to quantify the size and location of bone defects on the humeral head and glenoid [14]. Digital subtraction of the humeral head allows for precise determination of bone loss on the glenoid [15]. Magnetic resonance imaging (MRI) and MRI arthrography can provide useful information on the presence of tears of the capsulolabral tissue. These images are invaluable in preoperative planning for surgical stabilization, as the decision can be made whether bone grafting of the defects will be necessary. Hill-Sachs defects that occupy less than 30 % of the humeral head can be managed effectively with an arthroscopic remplissage; defects greater than 30 % of the humeral head may require open bone grafting with humeral head allograft [14, 16, 17].

Treatment: Indications and Contraindications

Indications for surgery include shoulder instability with bone loss that causes pain and lack of function in patients that have failed appropriate conservative management. Contraindications include active infection and patients who are habitual dislocaters. Severe bone loss may be a relative contraindication to arthroscopic repair, as it may require an open bone grafting procedure.

Decision-Making Algorithm

Shoulder Instability

1. No bone loss → Arthroscopic Bankart repair
2. Bone loss
   (a) Glenoid loss <25 %, Hill-Sachs <30 %
   → Arthroscopic Bankart repair + remplissage
   (b) Glenoid loss <25 %, Hill-Sachs >30 %
   → Soft tissue Bankart repair + open bone grafting of Hill-Sachs
   (c) Glenoid loss >25 %, +/– Hill-Sachs >30 %
   → Latarjet or open bone grafting procedure

Remplissage is performed in patients with moderate to large Hill-Sachs defects associated with glenoid defects of less than 25 %. Patients with larger glenoid defects may require conversion to an open bone grafting procedure, such as the Latarjet procedure.

Clinical Case/Example

The case presented is that of an 18-year-old, right-hand dominant male high school senior with right shoulder recurrent instability. The patient initially dislocated his shoulder while making a tackle during a football game 1 year prior to presentation. This initial traumatic dislocation was reduced at the emergency department under sedation, and he was managed conservatively by the treating physician. He sustained his second dislocation at the beginning of his senior season of football and was reduced on the field by the athletic trainer. At that time, as the starting middle linebacker for his team, he elected to forego surgery in order to play his senior season of football. He was able to complete the season.

Upon presentation to the orthopedic clinic, he reports he sustained approximately four additional dislocations during the football season. With each dislocation event, he was able to reduce the shoulder on his own. He reported subluxations several times per week, including several occasions in his sleep. He avoided provocative positions of abduction and external rotation for feelings of instability. He only reported pain with instability episodes.

On examination, he held his shoulders retracted, and there was no muscle atrophy. Passive ROM of the right shoulder produced apprehension in external rotation above 90° of abduction. In this position, load and shift testing translated the humeral head over the rim of the glenoid with crepitus. Jobe's apprehension and relocation tests were positive. There was no posterior or inferior instability detected on exam.

An AP radiograph of the shoulder revealed loss of contour of the anteroinferior glenoid, but there was no visible Hill-Sachs defect. MRI (Fig. 35.1) confirmed an anteroinferior capsuloligamentous deficiency consistent with Bankart tear,

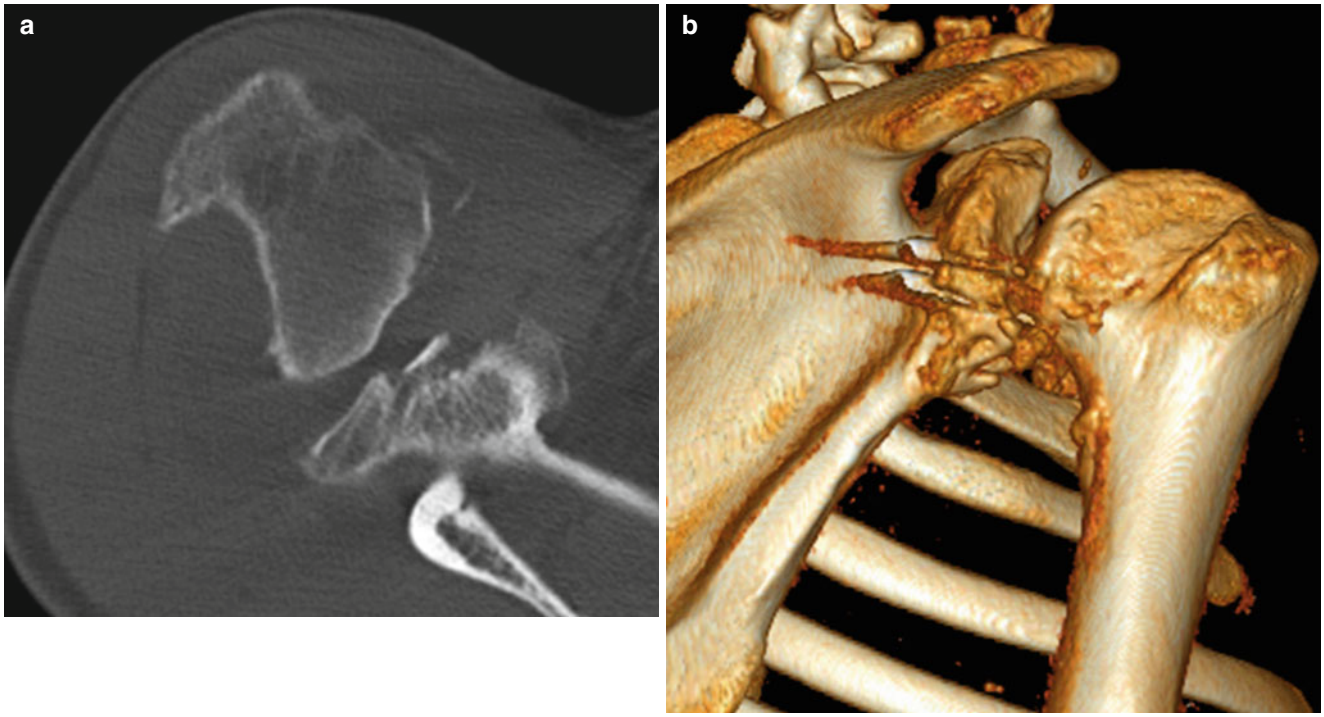

Fig. 35.1 CT scan (**a**) and 3D CT (**b**) demonstrating a large Hill-Sachs defect. The 3D CT is very helpful in determining the position and size of the defect

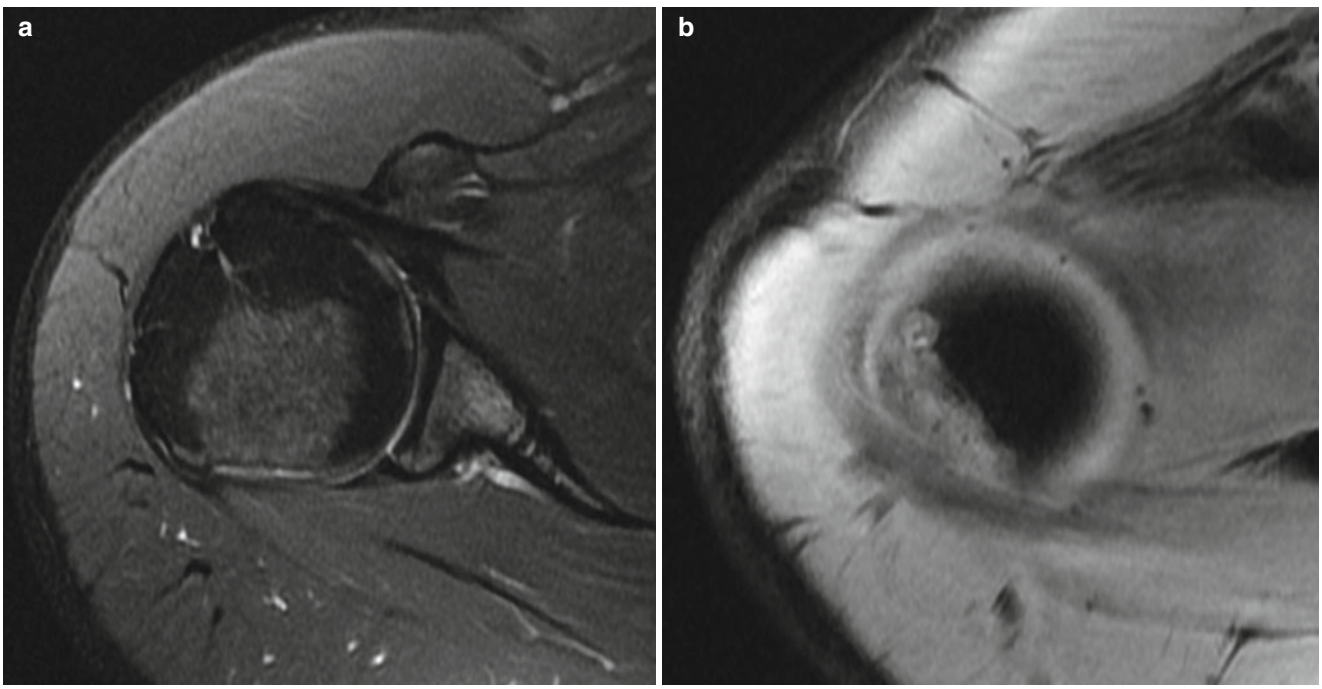

Fig. 35.2 Axial MRI cuts of a right shoulder in a football player with recurrent instability, showing (**a**) the anterior capsulolabral disruption and (**b**) the Hill-Sachs defect

with the labrum healed to the medial glenoid neck. Minimal bone loss (approximately 10 %) was noted on the glenoid; however, a moderate-sized Hill-Sachs defect was noted, occupying approximately 20 % of the humeral head (Fig. 35.2).

The patient had pain, recurrent instability, and lack of overhead function and failed a period of conservative treatment. For these reasons, surgery was recommended for a right shoulder stabilization procedure. Due to the minimal

(10 %) glenoid bone loss and a moderate (20 %) Hill-Sachs defect, the proposed surgery consisted of an arthroscopic soft tissue Bankart labral repair with remplissage to address the humeral head bone loss.

Arthroscopic Treatment: Surgical Technique

Patient Positioning

The surgery can be performed either in the beach-chair or lateral decubitus position. The author's preferred surgical technique is with the patient under general anesthesia in the lateral decubitus position on a beanbag, reclining 30° posterior.

At the beginning of the case, examination under anesthesia is essential, allowing the surgeon to evaluate for anterior, posterior, and inferior instability. This is paramount to identifying the direction and degree of instability at different arm positions. With the pathology identified, a surgical plan can be determined prior to making skin incisions.

The use of an arm suspensory device and ten pounds of traction is standard. This provides distraction across the glenohumeral joint to assist with visualization and allows equal access to the anterior and posterior compartments, as well as the rotator cuff. Traction can be removed so the arm can be taken through a full ROM to determine if a Hill-Sachs lesion is engaging or non-engaging.

Portals

Standard arthroscopic portals are utilized. A standard posterior portal is placed in the raphe of the infraspinatus. Anterosuperior and anteroinferior portals are created and utilized for the repair of the anterior labrum. An accessory posterolateral portal may be created, if necessary, for completion of the remplissage or bone grafting of the humeral head. Anterior portals are created using an outside-in technique with a spinal needle. While the entry sites of the anterior portals are relatively close inside the shoulder joint, it is important to keep the skin incisions as far apart as possible. This creates more working space on the outside of the shoulder and limits collision of the arthroscopic instruments with the arthroscope.

With the arthroscope in the posterior portal, the anteroinferior portal is created first and should be placed at the level of the upper border of the subscapularis. Utilized as the primary portal for repair of the glenoid labrum, it can be placed such that the cannula can lever the subscapularis inferiorly, allowing more exposure to the inferior glenoid. This routinely allows access to the 5:30 position on the anteroinferior glenoid for repair of the labrum.

An anterosuperior portal is established next, as the viewing portal for the arthroscope. This portal is established directly above the biceps tendon, allowing the surgeon to work on either side of the biceps. For the majority of the case, the arthroscope is placed in the anterosuperior portal, providing full visualization of the entire glenohumeral joint, with access to both anterior and posterior compartments through one viewing portal. Cannulas are placed in the anteroinferior and posterior portals for anchor placement, suture shuttling, and knot tying.

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

Diagnostic arthroscopy begins with the arthroscope in the posterior portal, from which all anterior structures can be assessed. As with all orthopedic procedures, it is important to develop a standard routine for diagnostic arthroscopy. This portion of the procedure should be performed in the same order every time. All important anatomic structures will be assessed and no pathology will be missed.

With every instability case, it is imperative to evaluate the biceps tendon, biceps anchor, and superior labrum. The biceps anchor and superior labrum should be probed to evaluate for a superior labrum anteroposterior (SLAP) tear. Placing a probe over top of the biceps tendon can pull the tendon into the joint, permitting inspection of the upper portion of the tendon that lies in the bicipital groove and assessment for any subluxation of the biceps out of the groove. The subscapularis is examined for tears; a 70° arthroscope can be utilized if any concern exists for partial tearing. The supraspinatus and infraspinatus are also inspected from the posterior portal to assess for partial or full-thickness tears. The Hill-Sachs defect will be visualized and its size assessed.

The anterior glenoid labrum is closely inspected for the presence of a Bankart tear. The labrum can be probed to evaluate for instability. Each glenohumeral ligament (superior, middle, and inferior) can be inspected directly. The arthroscope is driven into the axillary pouch to assess for the presence of loose bodies. The capsule is inspected for any tearing off the humerus. In this way, a humeral avulsion of the glenohumeral ligament (HAGL) can be diagnosed and addressed accordingly.

The Hill-Sachs defect is assessed to determine the size and whether the lesion is engaging or non-engaging. With the arthroscope in the posterior portal, the arm is taken out of traction and brought into the athletic position of abduction and external rotation. In the presence of a large Hill-Sachs lesion, the defect will engage the anterior glenoid rim and the humeral head will fall off the track of the glenoid, subluxating anteriorly. This dynamic assessment confirms the

presence of an engaging lesion, which must be addressed at the time of surgery for a successful outcome.

Diagnostic arthroscopy continues with the arthroscope in the anterosuperior portal. This provides the surgeon a 360° view of the glenohumeral joint. The anterior, posterior, and inferior labrum can be visualized and assessed for tearing and instability. The presence of glenoid bone loss can be assessed using a graduated probe placed across the face of the glenoid from the posterior portal. The length from the anterior rim to bare area is measured and compared to the length from the posterior rim to the bare area. Amount of glenoid bone loss, if present, can be determined. If significant anterior glenoid bone loss is present, the decision may be made to proceed with a bone grafting procedure.

Finally, the Hill-Sachs defect can again be visualized from the anterosuperior portal. Assessment to determine the presence of an engaging lesion can be performed while viewing from the posterior or anterosuperior portal, depending on surgeon preference.

Given the bone loss on the anterior glenoid and humeral head, it is paramount to determine if the Hill-Sachs lesion is clinically significant based on the size of the lesion and whether it engages the glenoid and causes glenohumeral instability. Historically, lesions involving less than 20 % of the humeral head articular surface are rarely of clinical significance, whereas lesions greater than 40 % are nearly always clinically significant and cause recurrent instability [10, 16]. Failure to recognize significant bone loss in conjunction with a soft tissue Bankart repair can lead to failure of the surgical repair and recurrent instability. Burkhart and De Beer [6] found a 67 % failure rate of soft tissue labral repair in the presence of significant bone loss. Boileau [7] also found a high failure rate of soft tissue labral repair when the bone loss was not addressed. Recurrent instability after surgical repair is devastating for the patient after completing months of rehabilitation.

Large Hill-Sachs lesions with bipolar bone loss may engage the glenoid at low arm abduction angles, leading to glenohumeral instability with activities of daily living. Therefore, it is vital to take the arm through a functional ROM and perform a dynamic assessment of glenohumeral stability at the time of surgery. If any Hill-Sachs lesion is present, the surgeon must determine if the defect is clinically significant and if it engages the glenoid. Once diagnosed, the lesion can be addressed arthroscopically.

Step-by-Step Procedure (Box 35.1)

Arthroscopic Bankart Repair with Remplissage

1. *Prepare the labrum*. After completion of the diagnostic arthroscopy and identification of all pathology, the repair can be performed. The case begins with preparation of the glenoid labrum. With the arthroscope in the anterosuperior portal, an arthroscopic elevator is introduced through the anteroinferior portal. The elevator is used to release the torn labrum from the medial glenoid neck and elevate it away from the bony attachment throughout the tear down to the 6:00 position. It is important to elevate the labrum until muscle fibers of the subscapularis are visualized, completely mobilizing the labrum for repair. A motorized shaver is then introduced to remove all fibrous debris between the labrum and the bony glenoid. The hood of the shaver is placed against the labrum, protecting it from iatrogenic damage. The shaver is also used to prepare the medial glenoid neck and anterior glenoid rim by roughening the bony surface to facilitate healing of the labrum.

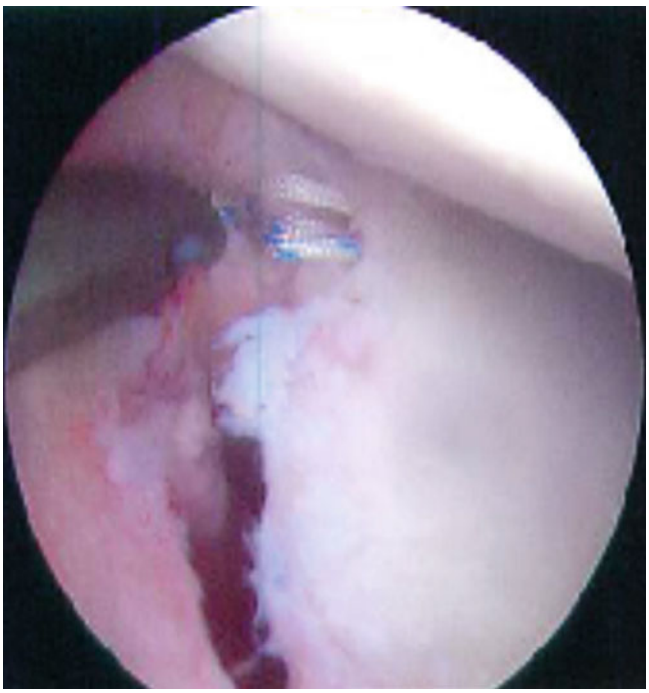

Fig. 35.3 Arthroscopic image demonstrating placement of the first glenoid anchor at the 5 o'clock position

2. *First glenoid anchor placement*. The first glenoid anchor is placed prior to addressing the Hill-Sachs defect. This facilitates reaching the inferior aspect of the glenoid. If the Hill-Sachs defect is addressed first and a remplissage is performed, the space inside the glenohumeral joint is reduced, and reaching the inferior glenoid for anchor placement can be challenging.

   The first glenoid anchor is placed, typically at the 5:00 position (Fig. 35.3) using double-loaded suture anchors. Either the anteroinferior cannula can be used to lever the subscapularis down or the anchor drill guide can be placed trans-tendon through the subscapularis to reach the 5:00 position. A percutaneous suture passer is used to pass both sutures in vertical mattress fashion. The first

suture is placed at 6:30, and the second at 5:30. Both sutures are subsequently tied. This repairs the anteroinferior labrum back to the glenoid rim while also completing an anterosuperior capsular shift.

3. *Prepare the Hill-Sachs defect*. The arthroscope remains in the anterosuperior portal, and a motorized shaver is placed through the posterior portal. The base of the Hill-Sachs defect is prepared by using the shaver to remove all fibrous tissue until bleeding bone is encountered.
4. *Perform the remplissage*. An arthroscopic remplissage is performed to transfer the posterior capsule and infraspinatus tendon into the Hill-Sachs defect. This transforms the engaging Hill-Sachs lesion to a non-engaging lesion. One or two double-loaded rotator cuff anchors are placed directly into the Hill-Sachs defect. It is important to remember that the defect represents a compression fracture and that the bone is usually very hard, necessitating over-tapping to prevent anchor fracture. The anchors can either be placed directly through the posterior cannula or percutaneous through an accessory posterolateral portal.

   Next, the posterior cannula is slightly backed out into the subacromial space, so the tip of the cannula lies just outside the posterior capsule and infraspinatus tendon. A percutaneous suture passer is used to retrieve the sutures through the posterior capsule and infraspinatus tendon. Typically, two mattress sutures will be placed for each anchor. A sliding knot is used for each suture and the sutures are tied blindly in the subacromial space (Fig. 35.4). Tying the sutures transfers the posterior capsule and infraspinatus tendon into the Hill-Sachs defect, converting the Hill-Sachs defect into a non-engaging lesion. The remplissage is now performed, prior to completing the Bankart repair. Completing the Bankart repair before addressing the Hill-Sachs defect will tighten the glenohumeral joint and translate the humeral head posteriorly, making visualization of the Hill-Sachs defect difficult.
5. *Complete the Bankart repair*. A double-loaded suture anchor is placed at the 3:00 position. Vertical mattress sutures are placed at 4:00 and 3:00 and tied. A third double-loaded anchor is placed at the 1:00 position. Vertical mattress sutures are placed at 2:00 and 1:00 and tied. This completes the Bankart labral repair.

Box 35.1: Tips and Tricks

- The lateral decubitus position offers excellent visualization of the entire glenohumeral joint. Placing the arthroscope in the anterosuperior portal provides a view of both the anterior and posterior compartments along with the Hill-Sachs defect and provides complete access to all areas of the joint, facilitating reparative procedures in the anterior and posterior shoulder. The Hill-Sachs defect can easily be addressed from this vantage point.
- Placing the first glenoid anchor before completing the remplissage facilitates the labral repair. Completing the remplissage first decreases the volume of the glenohumeral joint, and access to the anteroinferior glenoid may be difficult. Likewise, if the labral repair is completed prior to addressing the Hill-Sachs defect, visualization of the posterolateral humeral head may be obscured. Completing the procedure in the order given allows the surgeon to work efficiently in both compartments of the shoulder, addressing all pathology and completing the repair in a timely fashion.
- Alternatively, the Hill-Sachs defect can be prepared as the first step of the procedure without tying the sutures. The lesion is debrided, with anchors placed and sutures passed. The posterior cannula is then placed alongside the remplissage sutures, while the anterior labrum is repaired. As the final step, the remplissage sutures are tied to complete the repair.
- Using the posterior cannula to pass and tie the sutures facilitates the remplissage. By slowly backing the cannula out of the posterior glenohumeral joint, perfect placement in the subacromial space is achieved. The sutures are tied, securing the posterior capsule and infraspinatus into the Hill-Sachs defect, with the knots safely in the subacromial space. This avoids placing the arthroscope in the subacromial space to retrieve the sutures, which may take time and could potentially damage the sutures.

Open Bone Grafting of Hill-Sachs Defect

For large Hill-Sachs defects occupying greater than 30 % of the humeral head articular surface, an open bone grafting procedure may be necessary [14, 17]. This procedure may be performed in the beach-chair position through an anterior approach utilizing the deltopectoral interval or in the lateral decubitus position via a split between the middle and posterior deltoid muscles. In the anterior deltopectoral approach, a subscapularis tenotomy is used to gain access to the glenohumeral joint.

1. By bringing the arm into extreme external rotation, the Hill-Sachs defect is visualized. The base of the defect is prepared, either with a small curette to decorticate the bony bed or a small sagittal saw to make fresh bone cuts. The defect is measured with a ruler, determining both the anteroposterior and medial-lateral dimensions.

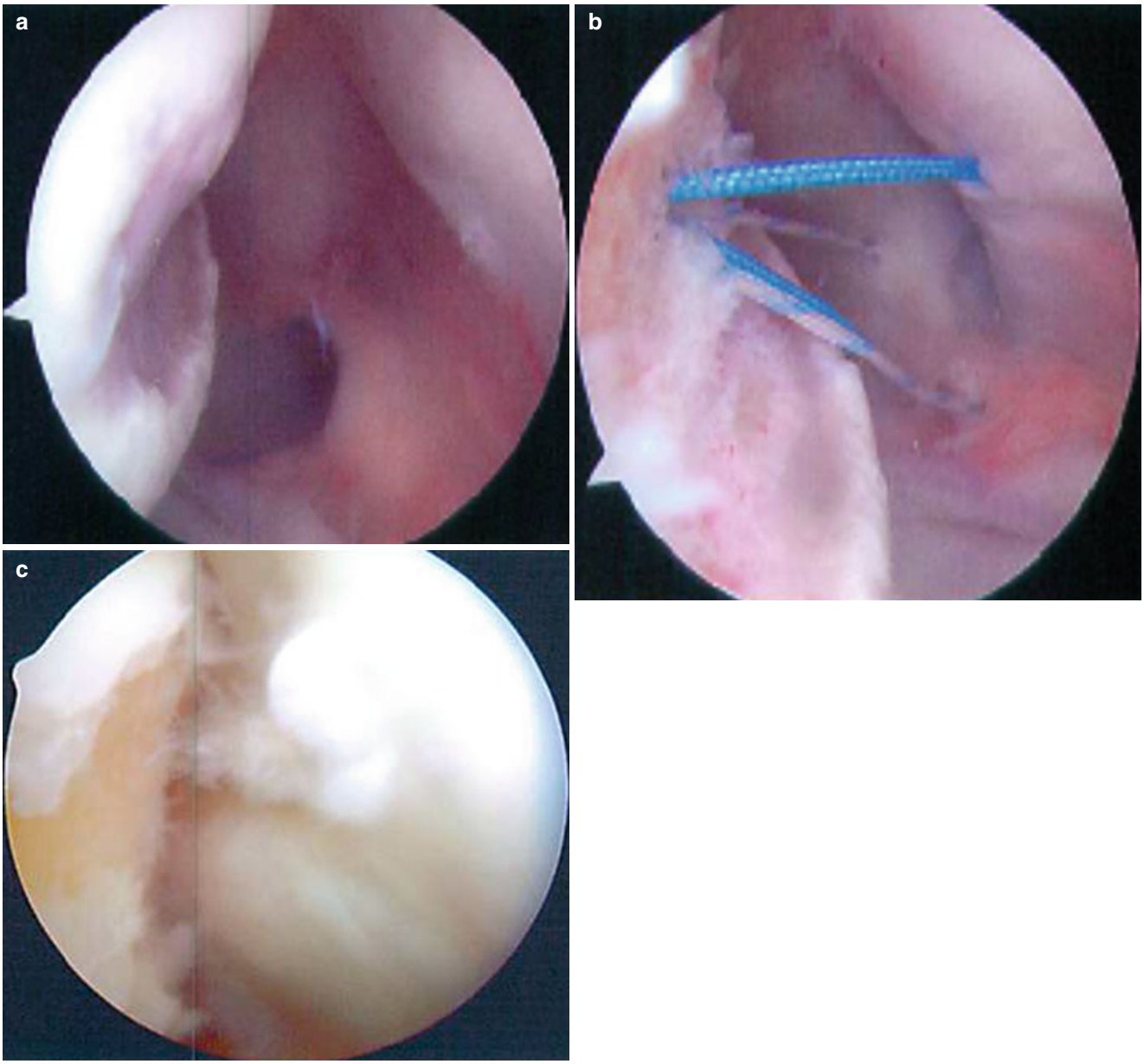

Fig. 35.4 Arthroscopic images demonstrating the remplissage. Once the defect is visualized (**a**), it is debrided with a shaver and a suture anchor is placed in the center of the defect (**b**). The cannula is backed into the subacromial space and mattress sutures are passed through the infraspinatus and posterior capsule (**c**). Once the sutures are tied, the infraspinatus and posterior capsule are transferred into the Hill-Sachs defect, completing the remplissage

2. On the back table, a fresh-frozen allograft humeral head is selected. The dimensions of the native lesion are marked on the posterolateral aspect of the allograft humeral head corresponding with the location of the Hill-Sachs defect. A sagittal saw is used to make the bone cuts, producing a wedge of allograft bone to fill the defect in the native humeral head.
3. The bone graft is then placed into the patient's shoulder, filling the humeral head defect. The graft may require fine contouring to match the fit of the native humeral head. The graft is secured into position with two cannulated headless compression screws (Fig. 35.5). Care should be taken to sink the headless screws 2 mm below the articular surface to avoid contact with the glenoid. The shoulder is reduced and taken through a full ROM to ensure smooth rotation with no crepitus or grinding. The subscapularis tenotomy is repaired and the deltopectoral interval is closed.

In the posterior deltoid split approach, the arm is left in traction. The interval between the posterior and lateral deltoid is split, allowing direct visualization of the infraspinatus. The raphe between the upper and lower infraspinatus is split, allowing direct visualization of the humeral head defect. The defect is sized, the allograft placed, and the infraspinatus closed. The arthroscopic Bankart repair can then be performed.

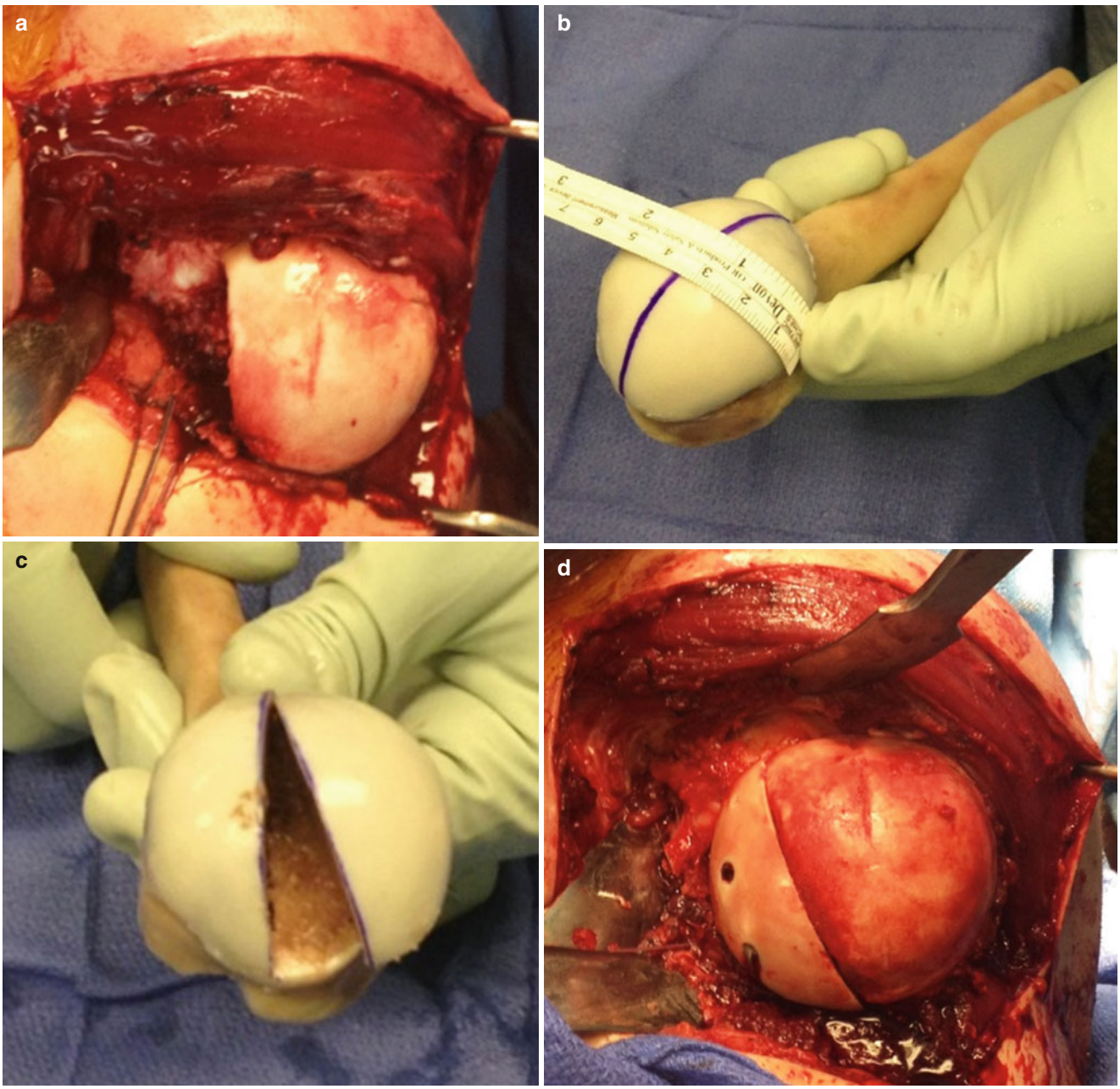

Fig. 35.5 Open bone grafting of a Hill-Sachs defect. The Hill-Sachs defect is exposed (**a**) by externally rotating the arm. The defect is measured, and the corresponding measurements are marked on the allograft humeral head (**b**). The humeral head cut is made with a small sagittal saw (**c**). The allograft wedge is then secured to the native humeral head with cannulated headless compression screws (**d**)

Postoperative Care

An abduction sling is utilized for 4 weeks postoperatively. Patients are allowed to remove the sling to shower, dress, and perform elbow ROM exercises. Patients are encouraged to begin posture training and scapular retraction exercises. At 4 weeks, the sling is discontinued. Formal physical therapy is initiated for passive and active-assisted ROM. Rotator cuff and periscapular strengthening is initiated at 12 weeks. Full return to sport is allowed at 5 months postoperatively.

Literature Review

"Remplissage," French for "to fill in," is an arthroscopic technique to fill a Hill-Sachs defect with local capsulotendinous soft tissue. It is a modification of the Connolly procedure [18], an open procedure transferring the infraspinatus tendon and a small portion of the greater tuberosity into the humeral head defect. In 2008, Eugene Wolf [19] described an arthroscopic modification, performing a posterior capsulodesis and infraspinatus tenodesis with fixation of the tissue

to the surface of the Hill-Sachs defect. This successfully fills the humeral defect by converting it from an engaging, intra-articular defect into extra-articular lesions. The goal is to prevent engagement of the lesion with the anterior glenoid.

In 2009, the technique was modified by Koo et al. [20], who described a double-pulley suture technique using two anchors to insert the infraspinatus tendon into the entire Hill-Sachs defect. This modification created a broader footprint of fixation and tying the sutures over rather than through the infraspinatus tendon allowed for a more anatomic, tissue-preserving approach.

Advantages of the remplissage technique include the ability to address the humeral head bone loss entirely arthroscopically and to perform concomitant procedures and a fast recovery time. Additionally, this approach has none of the risks and morbidity associated with open bone grafting procedures. Potential disadvantages include decreased postoperative ROM and sequelae of a nonanatomic repair construct.

In 2008, Deutsch and Kroll [21] described a case of significant postoperative loss of external rotation following remplissage. Motion was improved following arthroscopic release of the infraspinatus tenodesis. The authors proposed that the infraspinatus tendon and posterior capsular tissue created a mechanical block to motion, limiting external rotation.

Limitations to motion have been corroborated by other studies, yet the clinical significance is unknown. Elkinson et al. [22] evaluated shoulder stability and ROM following remplissage in a cadaveric model. The addition of remplissage to Bankart repair resulted in a statistically significant reduction in shoulder internal-external ROM in adduction (15.1°), but not in abduction (7.7°). Remplissage provided little additional benefit in specimens with a 15 % Hill-Sachs defect, but was effective in preventing engagement and recurrent instability in specimens with a 30 % Hill-Sachs defect.

Giles et al. [23] compared remplissage to humeral head allograft and partial resurfacing arthroplasty in cadaveric specimens with 30 and 45 % Hill-Sachs defects. Remplissage effectively prevented engagement in all specimens, but caused a greater reduction in ROM compared to allograft reconstruction and resurfacing arthroplasty.

Elkinson et al. [24] compared three remplissage techniques in a cadaveric model to assess the effect on shoulder stability: anchors in the Hill-Sachs defect valley, anchors in the humeral head rim, and anchors in the valley with medial suture placement. All remplissage techniques enhanced shoulder stability but restricted ROM and increased joint stiffness. Medial suture placement resulted in the greatest joint stiffness.

Stiffness observed in a cadaveric model may be more severe than that observed clinically. Boileau et al. [25] noted a loss of external rotation of 8° in adduction and 9° in abduction compared to the contralateral side in 47 patients after Bankart repair with remplissage. Ninety-eight percent of patients had a stable shoulder, 90 % were able to return to playing sports postoperatively, and 68 % returned to the previous level of sport, including overhead athletes. Park et al. [26] found no subjective complaints of decreased ROM in 20 patients after Bankart repair with remplissage. Three patients (15 %) did report a subsequent instability episode, but none required additional surgery. Nourissat et al. [27] also found no significant statistical difference in ROM between arthroscopic Bankart repair alone and Bankart repair plus remplissage at 2-year follow-up, with a difference in external rotation at the side of 4° and in abduction of 3°.

Summary

Several options exist for the treatment of large Hill-Sachs defects in patients with recurrent shoulder instability. Arthroscopic remplissage is a safe, effective technique for the management of bone loss on the humeral head. Postoperative reductions in external rotation may occur, but these differences may not be clinically significant. It is paramount for the orthopedic surgeon to recognize engaging Hill-Sachs defects and treat them accordingly. Failure to recognize bone loss at the time of operative stabilization may result in failure of soft tissue reconstruction surgery.

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Glenoid Bone Loss: Arthroscopic Bone Grafting

36

Ettore Taverna, Guido Garavaglia, and Henri Ufenast

Introduction

The etiology of anteroinferior glenohumeral instability is multifactorial. Successful treatment of this condition requires to identify and repair all clinically significant lesions which may be causing shoulder instability [1, 2].

An erosion of the glenoid is quite a common phenomenon in chronic anterior shoulder instability [3–6] (Fig. 36.1). Together with a Hill-Sachs lesion, a glenoid bone lesion often coexists in anterior shoulder dislocation (Fig. 36.2).

Prevalence of fracture or erosion of the anteroinferior part of the glenoid rim in shoulders with recurrent anterior dislocation has been reported up to 90 % of cases [3, 4]. The lesions are caused by impaction of the posterior superior aspect of the humeral head on the anteroinferior glenoid rim during an episode of dislocation.

Biomechanical studies have found an inverse relationship between the size of the glenoid defect and the stability of the shoulder: the larger the defect, the less stable the shoulder. The stability of the shoulder progressively decreases as the size of the osseous defect increases [7–9]. Furthermore, clinical studies confirmed that a relationship does exist between the extent of the glenoid bone loss and the results of the treatment of recurrent anterior shoulder instability. Particularly, severe bony lesions (i.e., large Hill-Sachs lesions and/or glenoid bone loss) are associated with failure of the arthroscopic treatment of shoulder instability and constituted in the recent past the real limit of the arthroscopic approach [7, 10].

Treatments for patients with recurrent anterior shoulder instability have been grouped into two surgical categories: soft tissue repair and bone grafting. In cases involving bone loss from the humeral or the glenoid aspect of the shoulder, a bone graft procedure may be indicated [11–14].

Many authors recommend coracoid transfer if the glenoid rim deficiency involves 25 % of the anteroposterior diameter of the glenoid. Others suggest that measures to restore the arc of glenoid concavity may be beneficial, in terms of both stability and motion, for patients who have a glenoid defect greater than 20 % of the glenoid length [5, 7, 10]. The relationship between the size of the glenoid defect and the clinical outcome continues to be investigated. To date, the exact size of the glenoid bone loss that contraindicates an arthroscopic soft tissue repair is still unknown [8].

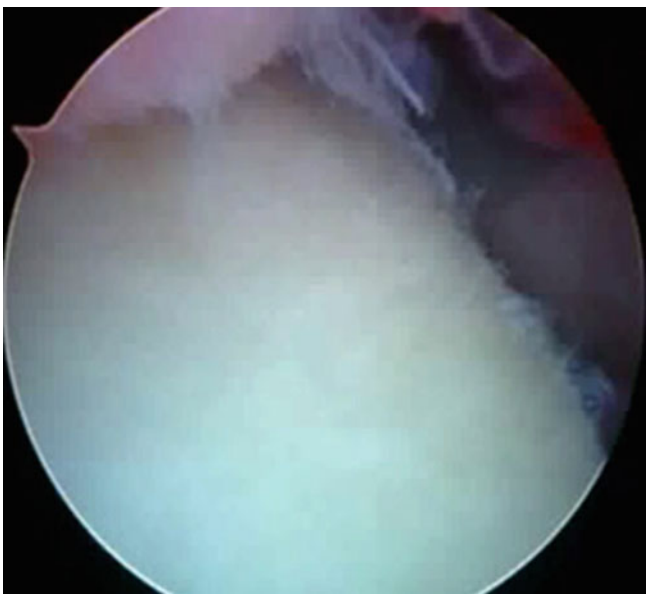

Fig. 36.1 Arthroscopic vision of an anterior glenoid bone loss

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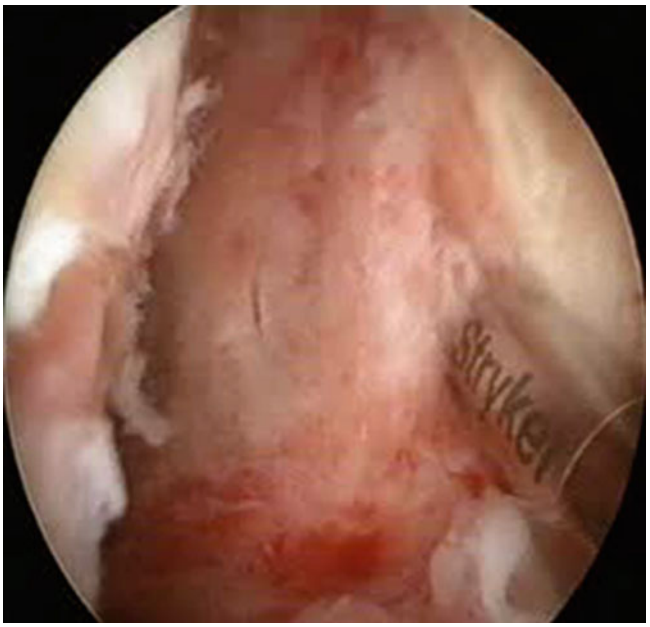

Fig. 36.2 Arthroscopic view of a bony Bankart lesion

Optimally, the arthroscopic surgeon should be able to address all clinically relevant lesions, including bony defects, in all cases by incorporating techniques that allow restoration of both the anatomy and biomechanical function of damaged structures. If not properly addressed, plastic deformation of the capsule and the ligaments and or hyperlaxity could constitute another cause of failure of the arthroscopic treatment [8].

Recently, some authors described techniques for arthroscopic positioning of a bone graft at the anteroinferior aspect of the glenoid [15–17].

History

Patients presenting with glenohumeral anterior instability with bony lesions report various mechanisms of injury. These mechanisms often include individual whose arm is taken into forcible external rotation when abducted 90°. A similar pattern can result from a fall on outstretched abducted arm. Direct distraction forces on the capsule and ligaments can be the mechanism of the injury.

The amount of force required to create the instability is important. Atraumatic or minimally traumatic events lead generally to subluxation. In patients who sustain dislocation, the force provoking the injury is generally greater and more likely shows capsule and ligaments tear and associated bony lesions. Individuals who required reduction by a physician at the first episode of dislocation are more likely to have soft tissue injuries, but with recurrent episodes of dislocation, bony defects are more and more common [3, 5].

Clinical Examination

Symptomatic instability is most often diagnosed by history and confirmed by physical examination.

Shoulder apprehension tests in all directions should be done to confirm the clinical diagnosis. The operative approach is aimed at the direction of instability.

Numerous instability tests have been described. We always perform the drawer test. The examiner stabilizes the scapula with one hand and grasps the humeral head with the other hand. Anterior and posterior stresses are applied; the amount of translation and pain must be recorded. In case of significant glenoid bone loss, the anterior translation can determine a dislocation of the shoulder. The apprehension and relocation tests are also often positive in patients with glenoid bone deficits. With the patient supine, the arm is taken into abduction and external rotation and an anterior stress is applied until the patient's apprehension is reproduced. Posterior stress is then applied by pressing against the humeral head with reduction of the anterior subluxation and immediate decrease of the patient's apprehension.

Imaging

Standard radiograph views in the instability series include anteroposterior views done in three rotations (internal, neutral, and external). Presence of a Hill-Sachs lesion could be noted for each rotation (present or absent). If present on the external view, its location is more superior on the humeral head. Glenoid lesions are noted with distinction between an avulsion fracture and a loss of the anteroinferior sclerotic contour in an AP view (Fig. 36.3) or using a glenoid profile

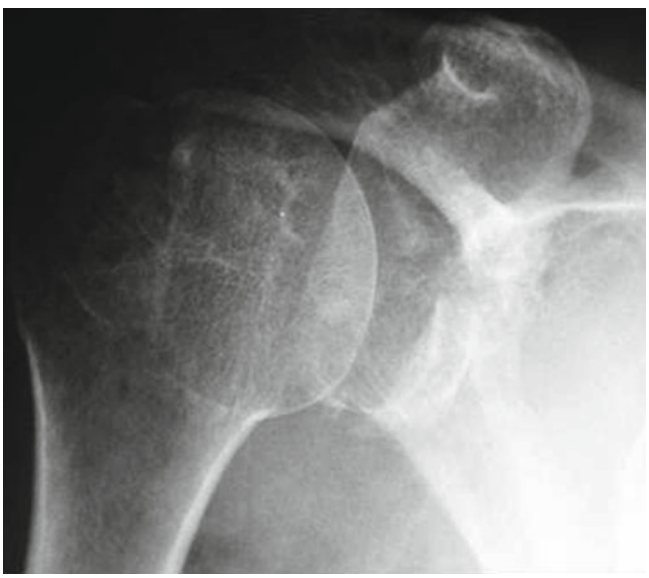

Fig. 36.3 X-ray AP view: fracture and loss of the anteroinferior sclerotic contour of the glenoid

view with a contralateral comparison view as described by Bernageau [3, 18]. Disruption of the anterior osseous triangle compared to the contralateral shoulder is classified into three groups:

- Fractures are defined as an abnormality of the anterior glenoid rim characterized by a visible osseous fracture fragment.
- The "cliff" sign is defined as a loss of the normal anterior triangle without a visible osseous fracture fragment.
- The "blunted angle" sign is defined as a rounding off of the normally sharp anterior angle of the triangle [3, 18].

If a bone defect in the glenoid is shown with X-ray projections, it is important to determine the area and the percent of the bone loss. In the past, the shape of the inferior glenoid has been described as a circle; using this circle method with a 3D CT, spiral CT, and MRI, it is possible to measure the bone defect of the glenoid [4, 5, 18–22].

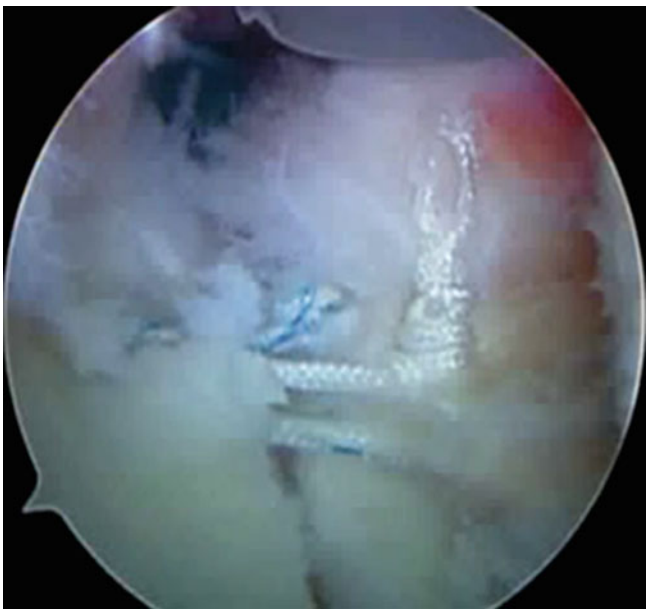

Fig. 36.4 Arthroscopic bony Bankart repair

Treatment: Indications and Contraindications

Recurrence of instability represents the major complication of anterior shoulder stabilization. Currently, most surgeons use suture anchor techniques for arthroscopic soft tissue stabilization because of more reproducible results [1]. However, even with recent technical advances, a recurrence rate of between 5 and 20 % still persists [8, 10]. The best way would be to preoperatively identify patients whose risk factors preclude arthroscopic stabilization. Numerous prognostic factors have been reported in the literature. Younger patients are at risk, but no clear limit is proved. Athletes who practice contact or collision sports have higher recurrence rates after standard arthroscopic stabilization.

Possibly, patients with significant glenoid bone loss, given the unacceptably high rate of recurrence after arthroscopic soft tissue repair, are good candidates for arthroscopic "bony procedures" [5, 8, 10, 15].

Decision-Making Algorithm

Treatment algorithms depend on many factors, but size and type (fragment or erosion) of the bone defect of the glenoid are paramount. If a mobile bone fragment is associated with the labrum lesion, then the possibility for an arthroscopic reattachment does exist (Fig. 36.4), despite the size of the fragment. In presence of bone erosion, no precise guidelines do exist. If the percent of bone loss is greater than 20 % of the area of the intact contralateral glenoid, a bone grafting procedure, either open or arthroscopic, to fill the defect and restore the glenoid arc is recommended by most authors [5, 7, 8, 10–12, 15–17]. If the missing area of the glenoid is less than 10 % and there are no patulous soft tissues, an arthroscopic soft tissue reconstruction is certainly an option for restoring the stability of the joint. If the bone loss is between 10 and 20 %, other factors should be considered. Certainly, a coexisting Hill-Sachs lesion could constitute an indication for a bony procedure. Table 36.1 outlines the treatment options based upon these factors.

Table 36.1 Algorithm for selecting appropriate procedure for anterior glenohumeral instability

Glenoid bone loss	Decision-making algorithms in shoulder instability	
>20 %	Bone grafting procedure	
<10 %	Soft tissue procedure	
>10 % < 20 %	Coexisting Hill-Sachs lesion	Bone grafting procedure
	ISIS score >6	Bone grafting procedure
	No Hill-Sachs – ISIS score <6	Soft tissue procedure

In addition to an accurate assessment of the possible presence of bone defect preoperatively, other risk factors that could preclude arthroscopic soft tissue stabilization must be verified [23]. If the instability severity index score (ISIS) [24] is more than 6 points, an isolated soft tissue reconstruction could be insufficient for stabilizing the shoulder (Table 36.2).

In summary, the preoperative careful assessment of bony lesions, the ISIS scoring system, physical examination, and history may help the surgeon to select patients who will benefit from an arthroscopic anterior soft tissue stabilization and those who will not.

Table 36.2 The ISIS score

	Prognostic factors		Points
Questionnaire	Age at surgery	≤20 years old	2
		>20 years old	0
	Degree of sport practice (preop)	Competition	2
		recreational or no sports	0
	Type of sports (preop)	Contact or forced ABD-ER	1
		Other	0
Exam	Shoulder hyperlaxity	Shoulder hyperlaxity	1
		Normal laxity	0
AP X-ray	Hill-Sachs on AP X-ray	In external rotation	2
		Not visible in ER	0
	Glenoid loss of contour on AP X-ray	Loss of contour	2
		No lesion	0
Total			10 pts

Surgical Technique: The Arthroscopic Bone Block Procedure

The arthroscopic anterior bone block procedure described by Taverna [17] combines an arthroscopic Bankart repair with the transfer of the tip of the iliac crest graft that is passed through a cannula placed in the rotator interval and fixed on the glenoid rim under the equator (Fig. 36.5). The efficacy of this procedure is related to the bone block effect provided by the tricortical bone graft that increases the size of the glenoid surface and the concavity recreation provided by the labral repair and capsular and ligaments shift and repair. The goal of the procedure is to restore the normal anatomy of the unstable shoulder with bone defects.

Bone Graft Harvesting

With the patient in supine position, we harvest a tricortical bone graft measuring 1 cm by 2 cm from the iliac crest. With a 1-mm Kirschner wire, we create two holes at 0.5 cm from the ends.

Patient Positioning

The technique allows for patient positioning in the beach chair or lateral decubitus position. Draping allows readied access to the posterior and anterior aspects of the shoulder girdle.

Portals

A high posterior lateral viewing portal is preferred for initial glenohumeral inspection so as to allow a subsequent posterior vector guide and double barrel guide sleeve to be inserted

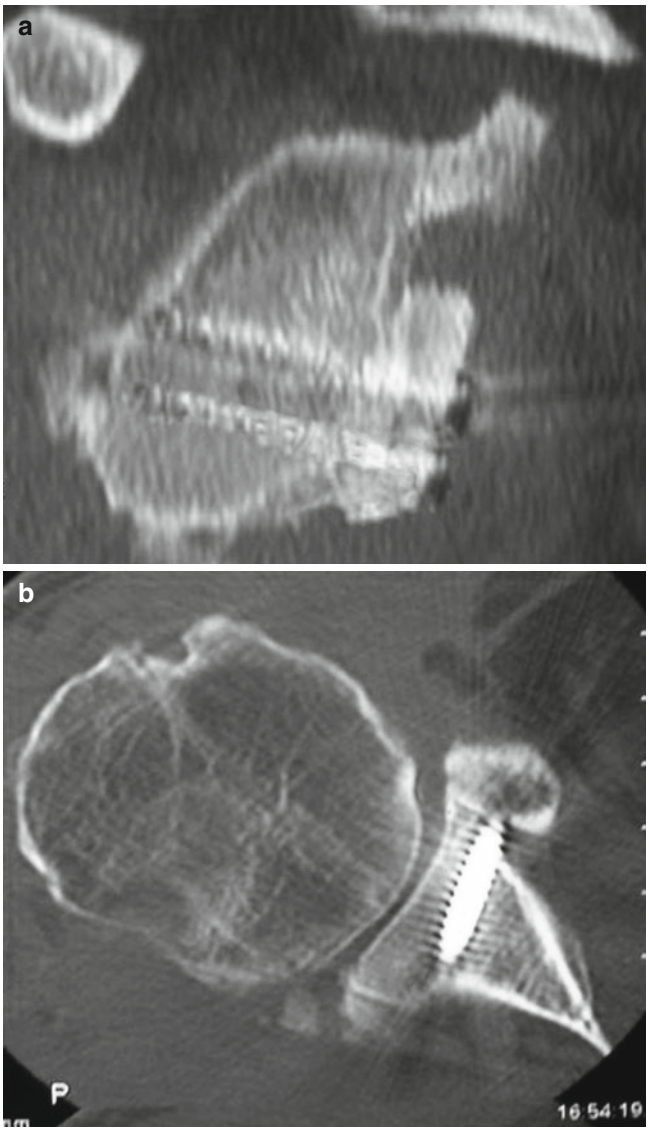

Fig. 36.5 (**a**) CT imaging of the bone block procedure. The bone graft is positioned to the anteroinferior margin of the glenoid. (**b**) CT scan at 1-year follow-up after a bone block procedure

posteriorly into the joint. Standard anterosuperior and anteroinferior portals are made utilizing 6- and 8-mm clear cannulas.

Step-by-Step Procedure

Following joint inspection, the anterior labrum and capsule are detached from the anterior aspect of the glenoid, utilizing the combination of an elevator, shaver, and radio-frequency device. The labrocapsular complex is freed to view the subscapularis muscle fibers. Once detached, a traction suture is placed into the labrocapsular tissue. The anterior glenoid bone defect is then smoothed with a motorized burr. A spinal needle is then inserted posteriorly and rested against the face of the glenoid south of the equator. A second posterior portal is created and the drill guide is inserted posteriorly and its arm placed flush along the face of the glenoid with its hook passed over the edge of the glenoid (Fig. 36.6). Each drill sleeve of the guide is then advanced until it is flushed to the posterior glenoid neck, and two cannulated drill pins are inserted and advanced by power until exiting parallel to one another 4 mm below the cortical edge of the anterior glenoid (Fig. 36.7). It is important to identify the cortex from the articular cartilage to ensure proper depth placement of the cannulated guide pins. A pair of specially designed flexible yet stiff guide wires is inserted through the cannulated drills and retrieved out the anteroinferior cannula (Fig. 36.8). Care is taken to prevent any twisting within the cannula during removal and later bone block passage. A tricortical iliac crest bone graft previously harvested and prepared in 20-mm length × 8 mm × 8 mm is prepared with 2.3-mm drill holes

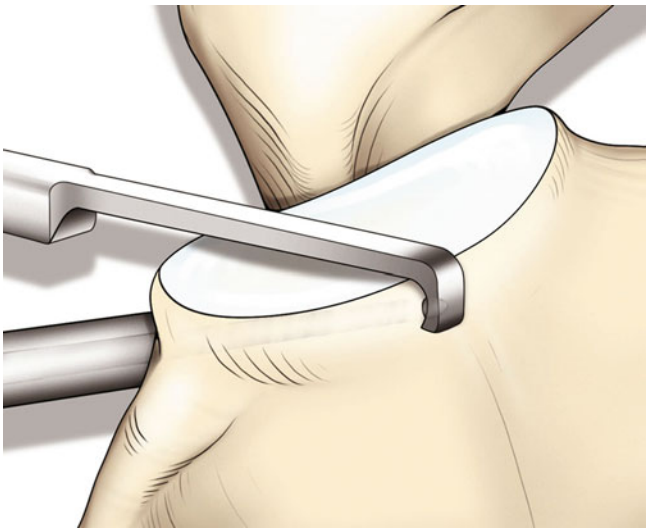

Fig. 36.6 Drill guide is inserted posteriorly and its arm placed flush along the face of the glenoid with its hook passed over the edge of the glenoid below the glenoid equator

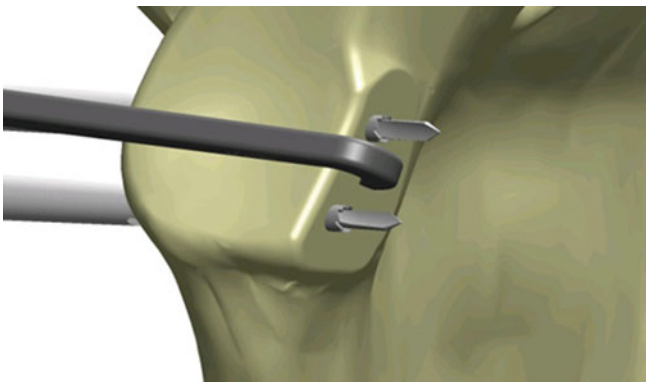

Fig. 36.7 Each drill sleeve of the guide is advanced until exiting parallel to one another 4 mm below the cortical edge of the anterior glenoid

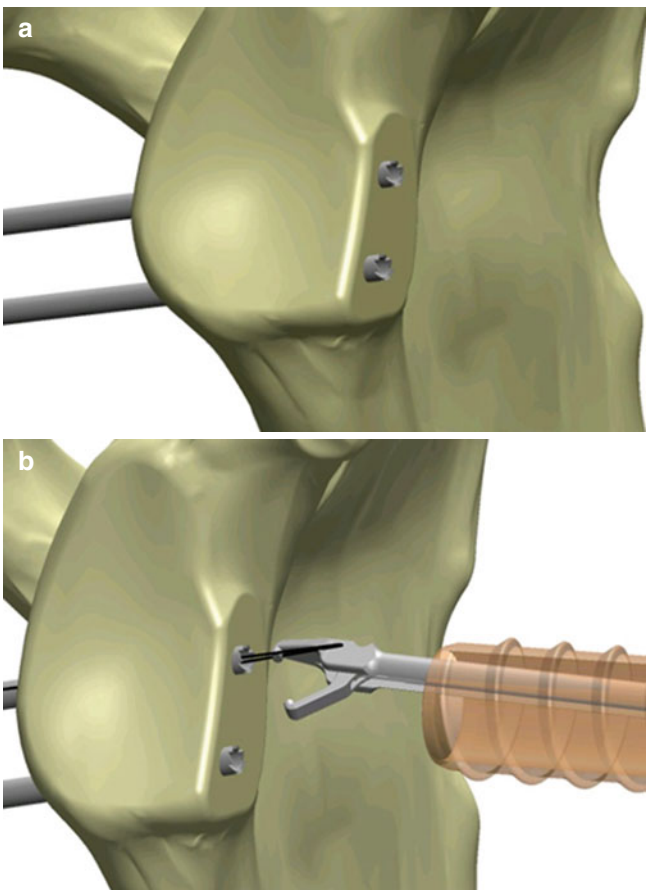

Fig. 36.8 (**a**) Cannulated sleeve position. (**b**) Flexible guide wires are inserted through the cannulated drill sleeves and retrieved out the anteroinferior cannula

placed 5 mm from each leading edge of the bone block. The bone graft is then placed over the guide wires and secured with two guide-wire stops (Fig. 36.9). The guide wires enter the bone block on the cancellous surface exiting on the single side of the cortex. The bone block is then inserted into the anteroinferior cannula, and while advancing, the guide wires

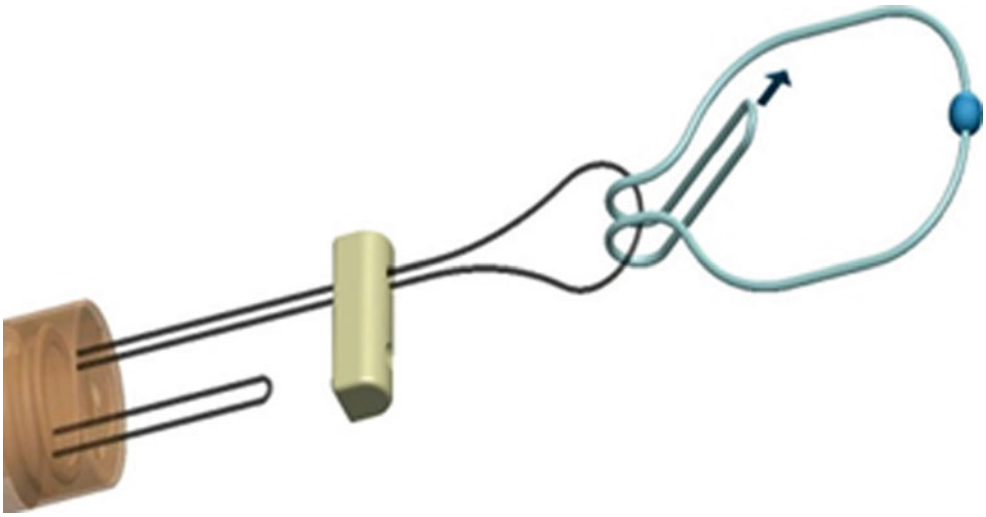

Fig. 36.9 The bone graft is then placed over the guide wires and secured with two guide-wire stops

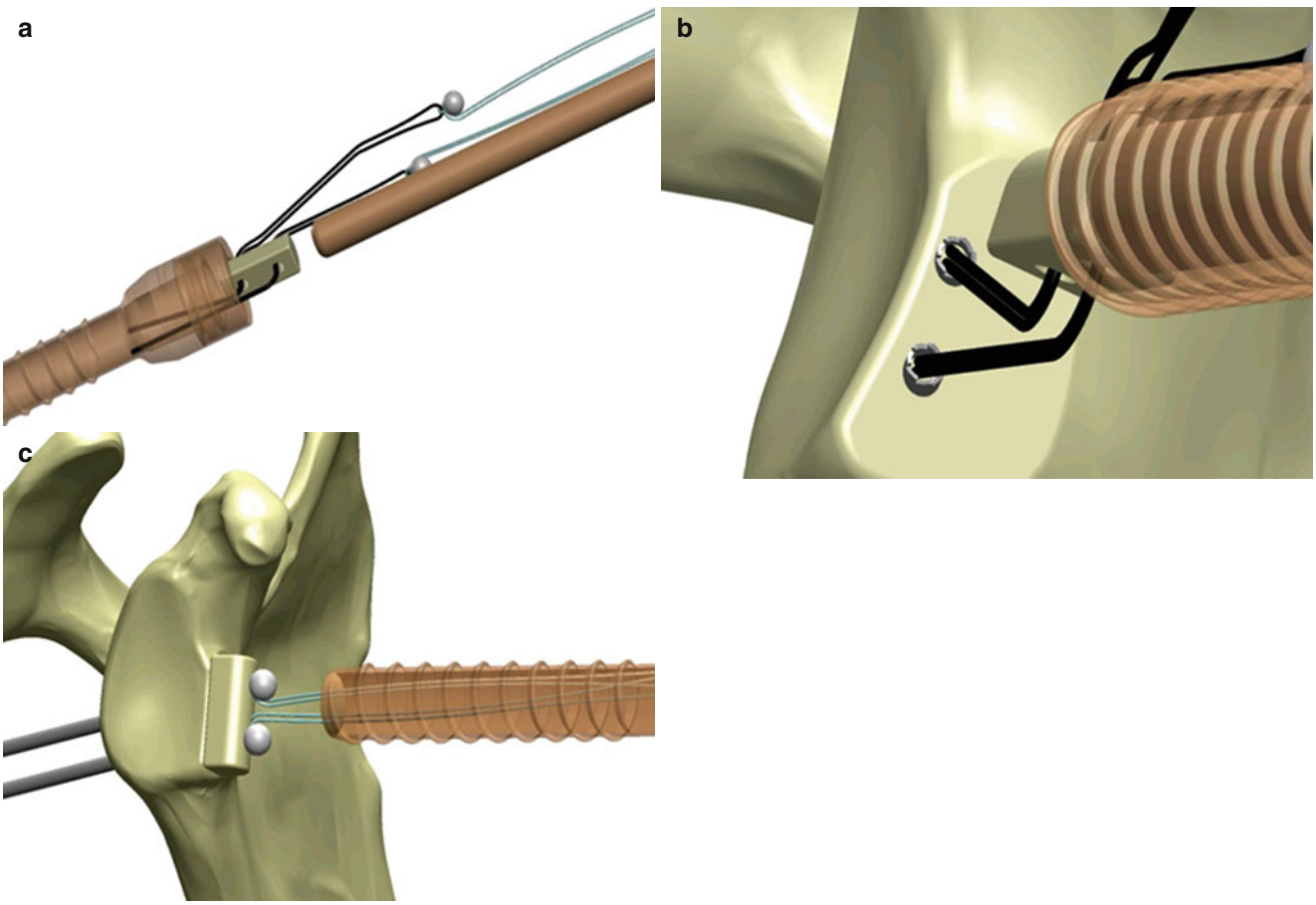

Fig. 36.10 (**a**) The bone graft is then inserted into the anteroinferior cannula. (**b**) Graft is passed into the cannula. (**c**) Graft into position along the anterior aspect of the glenoid

posteriorly are passed into position along the anterior aspect glenoid (Fig. 36.10). A cannula is inserted through a subscapularis portal, and subsequently, each guide wire is removed, exposing the stiffened portion of the wire. In sequence two, 3.5-mm cannulated metallic screws are inserted over the guide wires and cannula and advanced until fixation of the bone block is achieved (Fig. 36.11). Stability of the graft is assessed with an arthroscopic probe.

Three suture anchors are then placed along the glenoid rim at 3, 4, and 5 o'clock positions and then anteroinferior

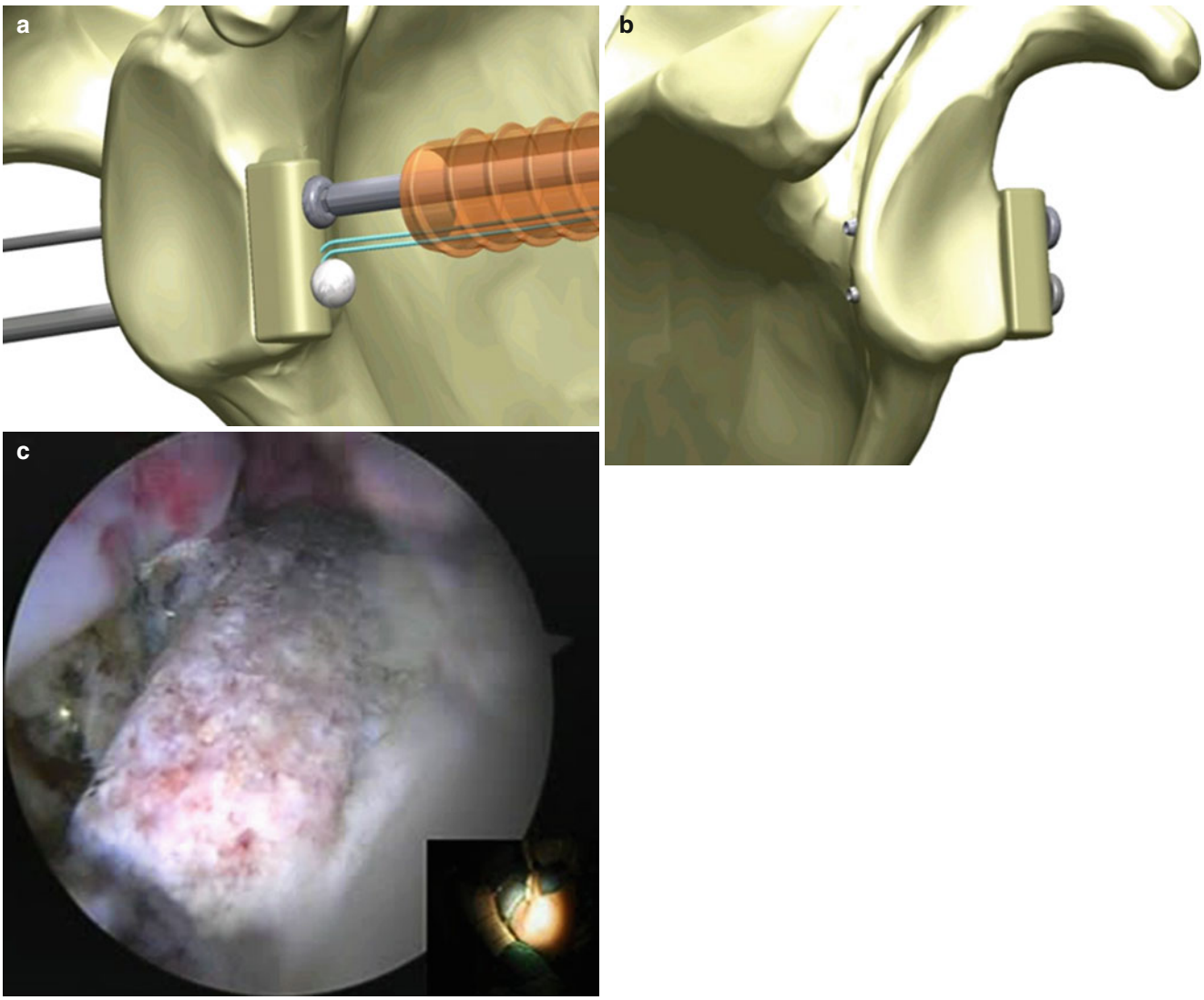

Fig. 36.11 (**a**) Two 3.5-mm cannulated metallic screws are inserted over the guide wires and cannula. (**b**) The screws are advanced until fixation of the bone block is performed. (**c**) Arthroscopic view of bone graft positioning

capsuloligamentous shift and anterior labrum reattachment are performed. In this way, the graft becomes an extra-articular platform (Fig. 36.12). The advantages of this procedure are the associated repair of the glenoid labrum and tensioning and shift of capsule and ligaments. As for the Bristow-Latarjet procedure, the bone block is placed in an extra-articular position, preventing synovial fluid from coming in contact with the bone graft, and avoids the potential contact between the humeral head and the bone block with the screw, which can cause pain and glenohumeral osteoarthritis. This is an anatomic procedure which restores normal glenohumeral anatomy, by increasing bony surface of the glenoid and recreating normal insertion of the labrum, ligaments, and capsule. Compared to the Bristow-Latarjet procedure, the damage to the subscapularis fibers is minimal. The weakest point of this procedure is the impossibility to address instability due to glenoid bone defects combined with inconsistency of the labrum-capsule-ligaments complex. In this case, the dynamic musculotendinous sling effect created by the conjoint tendon passing over the inferior part of the subscapularis of the Bristow-Latarjet procedure is mandatory.

Postoperative Care

Postoperatively, we recommend to keep the patients in a sling for a period of 3 weeks. After the immobilization, there is no limitation in passive motion and the patients are allowed to a full recovering in elevation and external rotation. After complete wound healing is obtained, swimming pool active exercises are recommended and resumed working activities are allowed. Progressive strengthening exercises are started

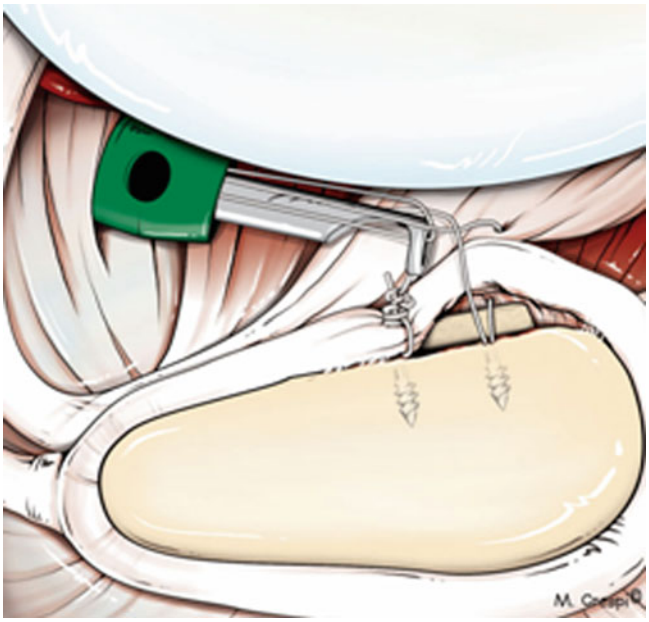

Fig. 36.12 Soft tissue repair is performed with anchors placed along the anterior glenoid rim

after 6–8 weeks. Return to overhead and contact sports is generally allowed after 4–6 months after surgery.

Summary

The effect of a glenoid defect on shoulder stability continues to be investigated. Biomechanical studies have found an inverse relationship between the size of the glenoid defect and the stability of the shoulder. However, there is no consensus on when and how bony procedures are needed to restore glenohumeral stability, and further studies are needed to determine the amount of bone loss that significantly affects the recurrence rate of an isolated soft tissue repair in an unstable shoulder. Generally, a bony erosion that narrows the inferior half of the glenoid to a width that is less than that of the superior half of the glenoid (the inverted-pear configuration) is certainly considered a contraindication for arthroscopic soft tissue repair. Therefore, such lesions require bone grafting of the anteroinferior glenoid.

The goal of an arthroscopic procedure for stabilizing a glenohumeral joint should involve restoration of the arc of glenoid concavity and when it is possible along with labrum repair and capsule and ligamentous tension. However, arthroscopic management of glenoid bone loss continues to be a complex surgical procedure. The above-described technique addresses many challenges associated with previously described procedures.

Placement of the bone block along the anterior glenoid followed by a traditional Bankart repair ensures extra-articular bone buttress support of the anterior portion of the glenoid face with preservation of the capsule-labral complex.

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Arthroscopic Latarjet Procedure

37

Laurent Lafosse, Vito Bongiorno,
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The arthroscopic Latarjet procedure (ALP) is the natural evolution of a long-term validated open technique. Thus combining the advantage of any minimally invasive technique with the possibility to treat other concurrent pathologies, the ALP has been developed and ameliorated during the last 10 years.

Anterior Shoulder Instability Lesions

The term "anterior shoulder instability" is commonly used to define symptoms related to pathological anteroinferior displacement of the glenohumeral joint. However, anterior shoulder instability can be due to a number of underlying soft tissue lesions, ranging from simple Bankart lesion to more complex capsulolabral lesions, such as anterior labrum-ligamentous periosteal sleeve avulsion (ALPSA), complex injuries of the labrum (Detrisac types II and IV), or humeral avulsion of glenohumeral ligaments (HAGL) [1–3]. Therefore, it is essential to be more precise in the analysis of anterior shoulder instability pattern in order to assess the criteria for selecting the most appropriate method of repair.

According to Walch et al. [4], we can define three major groups of patients:

- Group I: Dislocation (at least one full dislocation which needed reduction by another person than the patient)
- Group II: Subluxation (shoulder never fully dislocated but clear feeling by the patient of anterior shoulder displacement confirmed by the physical exam)
- Group III: Unstable painful shoulder (the patient complains about shoulder pain and the surgeon determines the origin of the pain as shoulder instability, as labrum detachment).

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In the most frequent cases of instability by dislocation (group I), the inferior glenohumeral ligament (IGHL) is always involved, and most of the times, soft tissues are severely injured (ligament or labrum tears, or HAGL lesion). This group showed the higher failure rate after soft tissue repair. This high failure rate seems to be rather predictable, as the classic arthroscopic Bankart procedure only consists of a simple reattachment of the glenoid labrum and IGHL. This treatment does not completely address the complex damage to the capsule-labral-ligamentous structures. In addition, bony lesions of the humeral head and glenoid can occur at the time of the dislocation, such as the Hill-Sachs lesion at the humeral side and/or glenoid rim fractures, which produce permanent loss of the bone stock, thus further impairing residual joint stability.

Different Therapeutic Options: Why a Coracoid Transfer?

At a certain extent of soft tissue and/or bony lesions, it clearly appears that an isolated soft tissue procedure is not sufficient. The most successfully used bone block procedure addressing bony lesions is the open coracoid transfer procedure (Latarjet or modified Bristow procedure) [5, 6].

Initial description of the Bristow procedure was a simple translation into the subscapularis muscle of the conjoined tendon by sawing the bony chip of the distal part of the coracoid. The modified Bristow procedure uses a larger fragment of the coracoid tip, which is fixed to the anterior glenoid neck with a single screw [7]. In the Latarjet procedure, half of the coracoid is fixed in a flat position using the advantage of congruence between the curvature of the anterior glenoid and the undersurface of the coracoid. The larger size of the bone block allows a double-screw fixation, thus providing better rotational stability and compression of the graft as well as restoring a larger portion of the missing glenoid surface. The ligamentoplasty effect is created by crossing the conjoined tendon over the inferior part of the subscapularis

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tendon, which is slightly tensioned in a more inferior and posterior direction. This reinforces the anterior restraints by creating a dynamic tension of the inferior capsule and the subscapularis, especially in external rotation, as originally described by Patte [8, 9].

By restoring or augmenting the glenoid bony contour, engagement of a Hill-Sachs lesion is prevented (except in very large, medial, and deep Hill-Sachs lesions that may need an arthroscopic remplissage). Actually, the subscapularis muscle is simply split horizontally and not superiorly detached with an L-shaped incision, as initially described.

An isolated transfer of the conjoined tendon to the glenoid neck over the subscapularis tendon has been described to replace the sling of the torn glenohumeral ligaments, and recently, some reports focused on the arthroscopic transfer of the conjoined tendon. However, these procedures do not address the inferior ligament weakness and/or the glenoid bone loss [10].

Latarjet (or the modified Bristow) is the most successful procedure because it combines a bony procedure with a ligamentoplasty provided by the conjoined tendon transfer through the subscapularis muscle. Moreover, high initial fixation strength of the coracoid allows a very early rehabilitation, without any postoperative immobilization in most of the cases. This is better achieved with the Latarjet procedure performed with a double-screw fixation rather than the modified Bristow (single-screw fixation).

Why an Arthroscopic Bone Block Graft?

There are several reasons to perform a bone block procedure arthroscopically:

- Placement of the bone graft is more accurate as it is placed under arthroscopic control.
- The open surgery does not allow to deal with associated intra-articular lesions like SLAP lesions, posterior labral tears, or posterior bone loss, which is fully repairable by an all-arthroscopic technique by soft tissue reconstruction or double (anterior and posterior) bone graft. It is impossible to deal with double instability by a single open approach.
- Open treatment for posterior instability is a very difficult and invasive surgery, for both soft tissue repair and bone graft technique. The arthroscopic iliac crest fixation is definitely much easier, more precise, and accurate when performed arthroscopically.
- If during an arthroscopic surgery planned for Bankart repair examination shows that soft tissues are not repairable (i.e., HAGL lesion and torn ligaments), conversion to open surgery is not compulsory to perform the Latarjet procedure.
- Revision for coracoid transfer failure by iliac crest transfer is less aggressive and allows extensive dissection and accurate release with good results when performed arthroscopically.
- Like in other joints, arthroscopic technique provides less soft tissue damage, easier and faster healing and recovery, earlier return to sport activities, less postoperative pain, and better cosmetic result.

Indications for Latarjet and Modified Bristow Procedure

Glenoid Bone Loss

Many authors reported failure of soft tissue repair due to glenoid bone loss [11]. The mechanical consequences of the anteroinferior glenoid erosion have been proved by biomechanics studies and assessed by different radiological, CT scan, and arthroscopic (inverted pear) studies [12]. In some cases, the bony fragment can be reduced and fixed arthroscopically with suture anchors [13]. However, in chronic cases, the fragment is smaller than missing glenoid contour and bone healing is not always achieved. Therefore, reattachment of bony fragments should be performed in the acute setting [14].

Humeral Bone Loss

Location and depth of the Hill-Sachs lesion varies according to the cases (small and superficial to deep and medially extended) and can be responsible for persistent instability, even in cases of sufficient glenoid bone reattachment. Assessment of the size and location of Hill-Sachs lesion is difficult and can be accomplished on conventional X-ray in internal rotation and 2D or 3D CT scans.

A "Remplissage" technique by fixation of the infraspinatus tendon into the bone defect has been described with satisfactory results, but external rotation is limited and long-term results are not reported [15].

Combination of Glenoid and Humeral Bone Loss

Combined bone loss on the glenoid and humeral head is frequent and responsible for major instabilities. This can be assessed with specific radiographic views and CT but is definitively confirmed by dynamic visualization during arthroscopy.

Irreparable Soft Tissues Damage

The HAGL (humeral avulsion of the glenohumeral ligament) lesion can be diagnosed with MRI or CT arthrography, but in most of the cases, it is discovered during arthroscopy.

Different techniques of capsular repair on the humeral side with suture anchors are possible depending on the lesion pattern, but results are disappointing essentially due to stiffness after repair [3, 16].

In cases of long-lasting recurrent instability, it is uncommon to encounter a simple detachment of the labrum-capsule-ligament complex, where simple glenoid reattachment provides a sufficient repair. However, if quality and the strength of soft tissues are not restored, stability cannot be restored.

Revision of Failed Bankart Repair

After an open or arthroscopic Bankart repair, failure can be easily diagnosed when recurrence of dislocation does occur. When regained stability is enough for a sedentary lifestyle, the patient cannot return to full sport activities but does not always complain of persistent instability. This explains some good results reported in short-term follow-up studies of arthroscopic Bankart repair [12, 17]. However, most of the times, patients are limited by a permanent minor instability, which can be painful and evolving towards more symptomatic instability and/or arthritis over time (5–7 years after surgery). In these cases, the lesion was not completely repaired: the glenoid is often eroded, the labrum-capsule-ligament complex is definitely damaged, and there is a real need of bone supply associated with a ligamentoplasty.

Patients at Risk

Comparative studies on the outcome of the Latarjet procedure and soft tissue repairs showed that results achieved with the Latarjet procedure are significantly better in terms of stability [17].

Indeed, stability is the most relevant outcome in shoulder instability repair and represents a crucial safety issue in some patients who have a high risk of recurrence due to their work (i.e., manual workers) or sport activities, such as throwing, or contact and collision sports (i.e., Judo and football). The Latarjet provides a supraphysiologic stability and is a great solution in these cases. Furthermore, as recovery time after Latarjet procedure is very short, manual workers and high-level athletes may benefit of early return to full stability and function of the shoulder. Finally, Latarjet procedure should be considered as first option in patients who require early recovery of external rotation after surgery.

One or Two Screws? Bristow or Latarjet?

Some surgeons believe the one-screw modified Bristow procedure is easier and faster than the double-screw Latarjet procedure. However, it is indisputable that the contact area of the bone graft is smaller with the modified Bristow. This creates the following concerns:

- The technique is unsuitable for large bone defects.
- Primary fixation strength is small.
- Risk of nonunion is high.

Indeed, good results have been reported with both techniques and there is no strong evidence to support the superiority of the Latarjet procedure. Accordingly, certain cases may benefit from either Bristow or Latarjet. Only a prospective randomized study with long-term follow-up could validate the appropriate indication of each technique.

Why a Specific Instrumentation and Specific Implants?

We developed an arthroscopic technique based on the same principles as the open Latarjet procedure. We performed the first full arthroscopic Latarjet in 2003 and worked on developing a full set of specific instruments which allow open and arthroscopic management of both Latarjet and modified Bristow procedure. The development of this specific instrumentation made the procedure standardized and reliable. We describe the different steps of the procedure for both open and arthroscopic technique. The main differences and advantages of both approaches are described [18].

Surgical Techniques

Open Technique

Today, there is no specific instrumentation for open technique; the coracoid is cut, prepared, and fixed with great difficulties to the anterior border of the glenoid through the subscapularis split as there is no simple way to get the screw fixing the coracoid into a predrilled glenoid hole. Fixation is achieved on a deep and poorly exposed area, and visibility is worsened once the glenoid is drilled because of increased bleeding; drill hole on the glenoid is also difficult to find with the screw already passed though the coracoid, which is difficult to manipulate. The instrumentation provides several advantages:

- Distance between the 2 holes is fixed and reliable.
- The use of a cannula eases manipulation and placement of the coracoid.
- Fixation of the coracoid to the glenoid neck can be temporarily achieved with a K-wire and easily modified; this makes the fixation more accurate and reproducible.
- Control of coracoid rotation during the first screw placement is provided by a second K-wire.
- Final fixation is increased with top-hat washers, which fits with the cannulated screws.

Arthroscopic Technique: Modified Bristow Procedure

The Bristow standing up position single-screw fixation is the easiest way to start.

Once the subscapularis has been split, a long cannulated screw is introduced into the inferior portal through a single cannula and fixed onto the coracoid along its anteroposterior axes. The coracoid is cut proximal to the fixation and moved to the glenoid for its final fixation using a long K-wire introduced through the cannulated screw.

Arthroscopic Latarjet Procedure (Box 37.1)

The Latarjet laying position with double-screw fixation needs a double cannula which is fixed to the coracoid by two steps: coracoid preparation including drilling and cut is managed through the superior portal; fixation to the cannula is managed through the medial portal.

Most of the surgeons who contributed to the development of the arthroscopic Latarjet started preparing arthroscopically the coracoid and then switched to open technique in order to manage the fixation. Step by step, they progressed doing more steps arthroscopically and less open. The swelling is the most important inconvenience of this method but is not a big issue after enough experience. A staged approach is still a useful strategy for surgeons who begin to perform this technique.

Box 37.1: Tips and Tricks

- Correct placement of the graft is crucial. Therefore, after joint assessment, we mark the graft position on the glenoid rim using an electrocautery (a burr could also be used). This must be done with the scope in the *A* portal (standard posterior) in order to have a perpendicular view of the anterior glenoid rim. If one tries to mark the glenoid later in the procedure through a superior or lateral portal, it is not possible to judge the placement of the graft correctly due to a too sharp angle of view (due to the 30° inclination of the scope). Make a clearly visible mark, as it might wash off during the procedure and become difficult to find at the time of graft placement.
- Inadequate placement of the *M* portal will make the subscapular split and correct graft placement very difficult. Usually, when misplaced, the *M* portal is too lateral. We use a switching stick through the *A* portal and align it with the glenoid plane. Looking from above the shoulder, the switching stick gives the precise location in which the *M* portal should be placed. Make sure not to advance the stick anterior of the subscapular muscle as the brachial plexus can be injured.
- Before creation of the "ring" around the coracoid, adequate release of the coracoid from its surrounding soft tissue must be performed, especially along the inferior and medial aspects of the coracoid in order not to have any impinging soft tissue during graft placement. A switching stick through the *D* portal can lift the deltoid muscle and help to create more space. One must also take care to release the conjoined tendon from its adhering tissue on the lateral and anterior side, thus creating a free graft easy to manipulate. Care must be taken not to harm the musculocutaneous nerve when liberating the conjoined tendon.
- After the graft is harvested, we use the burr to remove any remaining bone spurs and adjust the radius of curvature of its inferior aspect in order to improve matching with the glenoid neck contour. Maintaining the graft with the cannula and using the burr without suction reduce the risk of injuries to the surrounding neurovascular structures.
- Coracoid graft must be placed flush to the glenoid articular margin. Furthermore, screws do not have to interfere with the humeral head. Therefore, we take care to place the K-wires along the medial third of the coracoid, so that the screws will be placed medial to the glenoid surface and with a maximum distance from the humeral head.
- The subscapularis split must be placed in the lower third of the muscle in order not to restrict external rotation. We found that a too high subscapularis split limits external rotation.
- Scapula retraction is helpful to place the graft and screws parallel to the glenoid, thus avoiding proud placement. We use a Healix anchor introducer and screw it into the base of the coracoid through the *J* portal. The device can be used as a joy stick that allows easy retraction of the scapula backward and facilitates correct screw placement. Make sure not to use traction when performing an arthroscopic Latarjet procedure as it will pull forward the scapula and inevitably increase the angle between the glenoid surface and the screws.
- Assessment of graft placement in the axial plane (medial-lateral direction) is difficult. At this aim, we place a switching stick through the *A* portal over the glenoid surface and pointed towards the graft. This allows accurate assessment of graft placement,

as a too lateral graft will engage the tip of the switching stick. Placement can be adjusted by using the stick to push the graft more medially. If the graft is already fixed with pins, remove the too lateral one and turn the graft slightly around the correct one. This allows medial adjustment without losing the correct height. To ensure the graft is not placed too low (distal), drill the inferior screw hole first to feel whether or not the drill is in the glenoid bone.

Patient Preparation and Joint Evaluation

Interscalene block is done under ultrasound guidance and general anesthesia is then performed. A cerebral ultrasound Doppler is used to monitor brain oxygen rate in order to maintain low mean arterial blood pressure.

The patient is positioned in the beach-chair position and landmarks are drawn over the skin including a line showing the direction of the joint. Standard posterior portal (*A*) for the scope is made in the soft spot and intra-articular evaluation is performed. Joint assessment includes dynamic examination of instability, glenoid bone defects, humeral head bone defects (Hill-Sachs lesion), and HAGL lesions (Fig. 37.1).

Joint and Coracoid Preparation

An anterior portal (*E*) through the rotator interval is done. Electrocautery and shaver are inserted via the anterior portal in order to prepare the anteroinferior glenoid: removal of the glenoid labrum from 2 to 5 o'clock (right shoulder), anterior

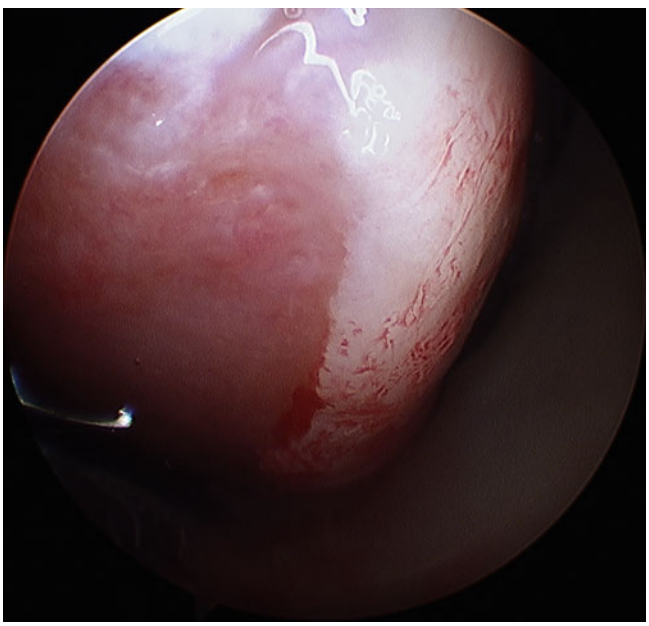

Fig. 37.1 Left shoulder: engaging Hill-Sachs lesion

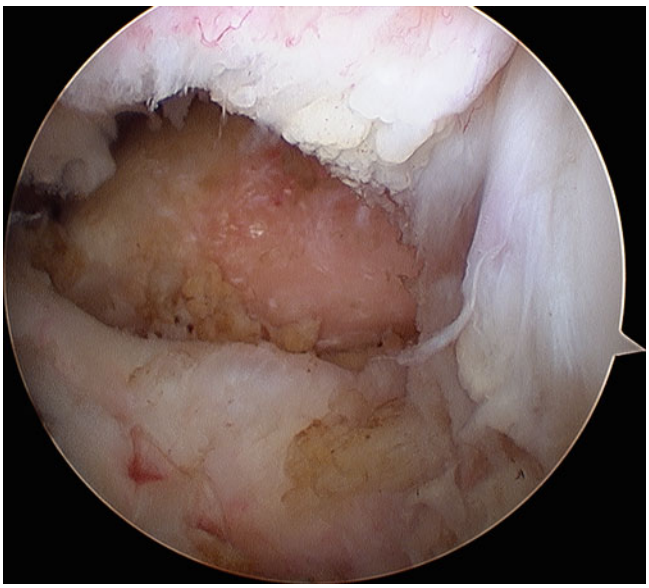

Fig. 37.2 Opening of the rotator interval (RI) and exposure of the lateral coracoid process

capsulectomy until exposure of the fibers of the subscapularis muscle, and marking on the anterior border of the glenoid cartilage for final graft placement. Subsequently, the rotator interval is opened to expose the coracoid and conjoined tendon, and appropriate care should be taken when releasing the coracoacromial ligament to avoid damage to the conjoined tendon (Fig. 37.2).

Anterolateral portal (*D*) is now created guided by a spinal needle. This portal is used to perform the following steps around the coracoid: complete opening of the rotator interval, detachment of the coracoacromial ligament from the lateral side, cleaning of the superior surface, and removal of the bursa from the inferior surface in order to expose the anterior and posterior aspect of subscapularis muscle. The fascia of the lateral conjoint tendon (coracobrachialis) is also opened down to the tendon of the pectoralis major. Glenoid neck is slightly abraded with a 4 mm burr through this portal in order to create a flat surface to promote healing of the graft (Fig. 37.3).

Coracoid Harvesting

With scope in the anterolateral portal (*D*), we now create the inferior axillary (*I*) facing the coracoid process, the inferolateral portal (*J*) located between *D* and *I* portals, and the medial portal (*M*) (Fig. 37.4). The medial portal is located using the switching stick coming from the back (*A* portal) to check the direction and level of the portal on the chest wall. The switching stick is not pulled through the subscapularis in order to preserve the axillary nerve and is removed after the portal is done.

Coracoid exposure is completed with the scope in the *I* portal by resecting the pectoralis minor tendon using the electrocautery from the *M* portal. It is important to detach the tendon close to the medial aspect of the coracoid in order to

protect the plexus and musculocutaneous nerve (Fig. 37.5). A switching stick is placed in *D* portal to lift up the deltoid and create additional room during this step.

The tip of the coracoid is identified with a long K-wire and a spinal needle is used to assess the correct position and create a portal above the coracoid (*H*). This portal is used to drill and perform osteotomy of the coracoid process. Remember that the clavicle, cephalic vein, and the head of the patient can hinder this step (Fig. 37.6). Two K-wires are drilled using the Coracoid Drill Guide, approximately 5 mm proximal to the tip of the coracoid and between the middle and medial thirds of the process. Once the K-wires are inserted, the drill guide is removed and the position checked using multiple views of the coracoid process (scope in I and J portals). The two holes are then drilled and tapped. A "top-hat" washer is inserted into each hole using the K-wire as a guide. A burr is now used to create a "stress riser ring" around the coracoid to ensure the osteotomy does not propagate into the proximal hole of the coracoid (Fig. 37.7). Once the ring is completed, the osteotomy is performed from the *H* portal using a curved osteotome. The coracoid is then free in the anterior space of the shoulder, giving excellent exposure to split the subscapularis.

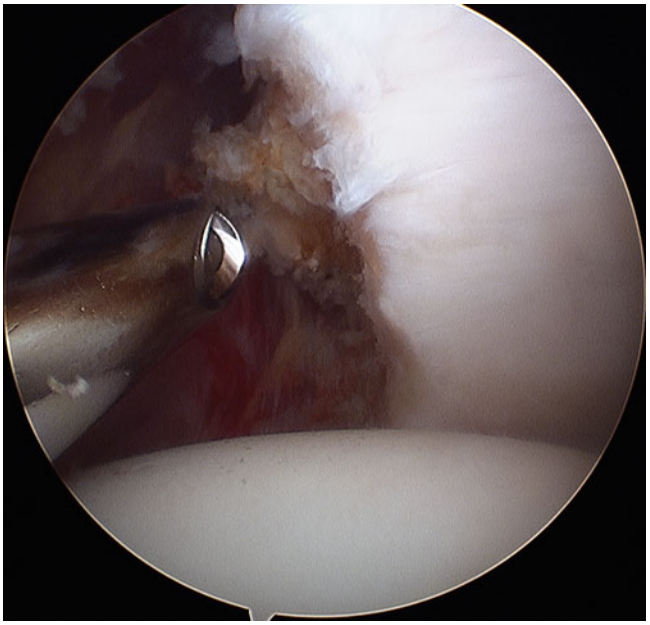

Fig. 37.3 Anteroinferior glenoid neck preparation and removal of the bony fragment. The scope is in the *D* portal through the RI

Subscapularis Split

The switching stick is inserted from the *A* portal and used to identify the level of the subscapularis split between the middle and inferior thirds of the tendon (check the markers prepared at the beginning of the procedure). Capsule has been previously removed so switching stick can be gently pushed through the subscapularis fibers and visualized from the anterior side of the muscle to avoid plexus injuries (axillary nerve is just in front of the subscapularis). Visualizing the subscapularis via the *I* and *J* (anterior view) portals ensures the split is in the correct location. The split is created using electrocautery (via *M* portal) from the tip of the switching stick coming out from the anterior side of the subscapularis fibers, laterally

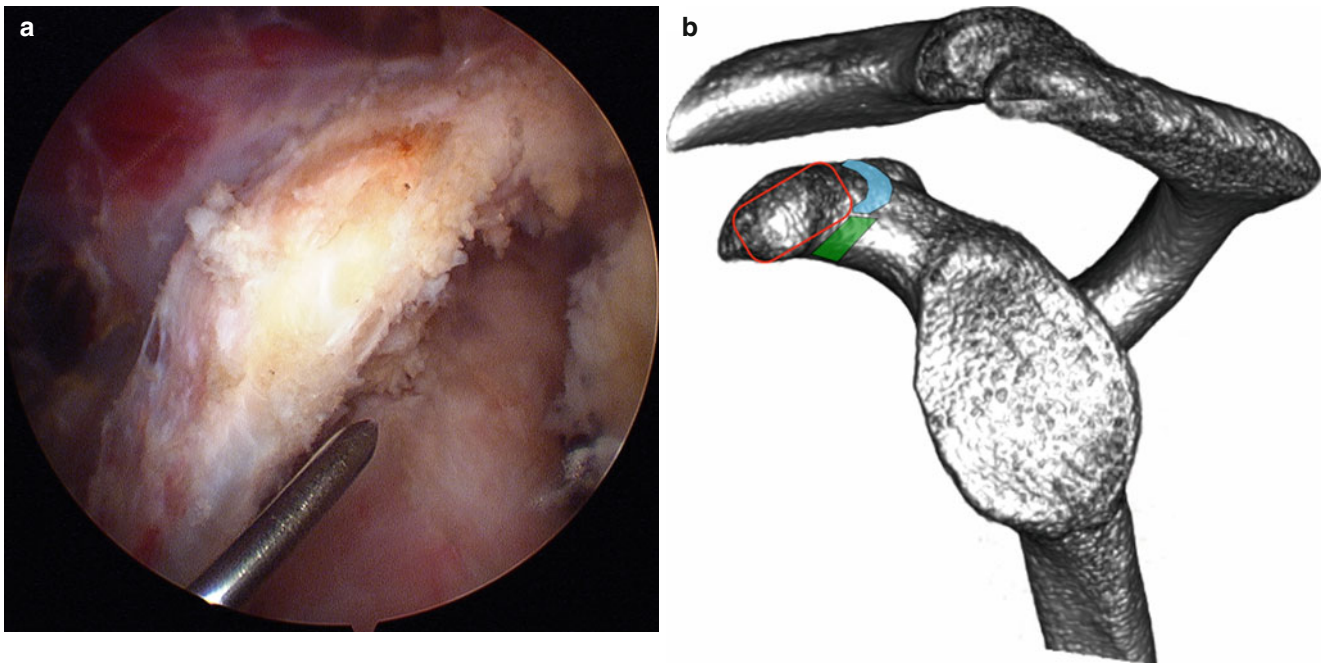

Fig. 37.4 (**a**) *D* portal visualization: a needle is placed to set the *I* portal, in line with the axis of the coracoid process. Pectoralis minor tendon is still attached. (**b**) Shoulder lateral view (*D* portal): note the orientation of the coracoid process and the stress-rising landmarks

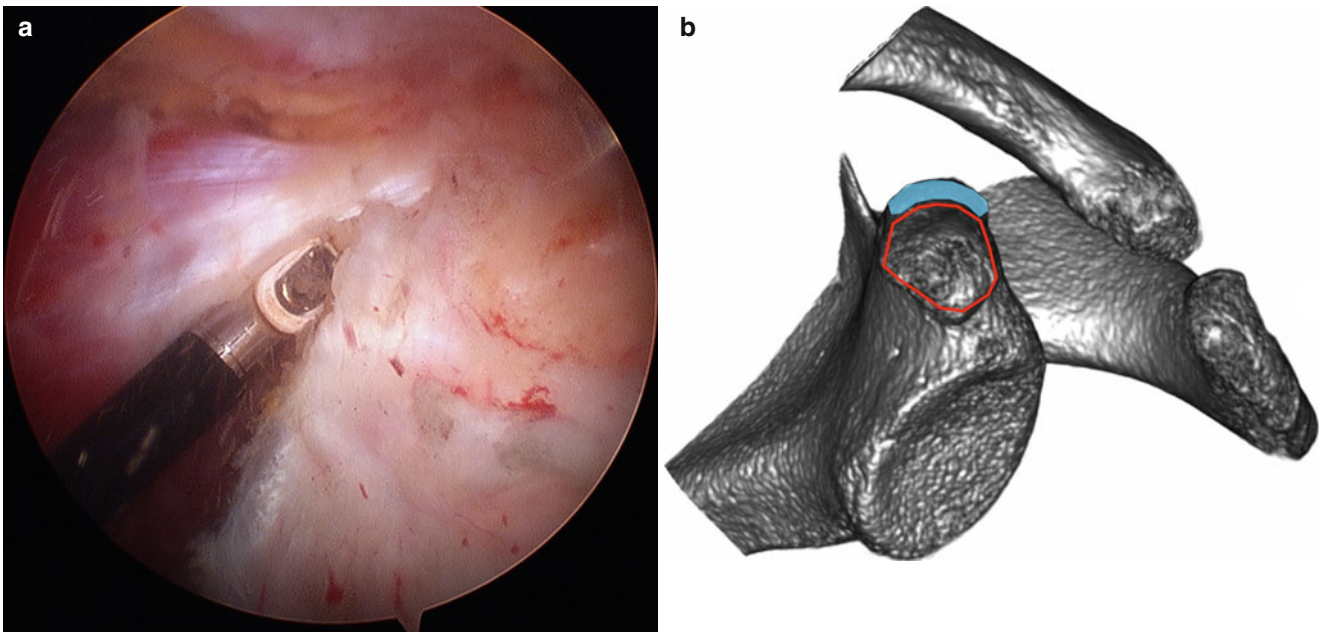

Fig. 37.5 (**a**) *I* portal view: pectoralis minor detachment at the junction with the medial conjoined tendon. (**b**) Coracoid view from the *I* portal: note that the scope is oriented at around 45° in order to be in line with the coracoid and conjoined tendon fibers. This is to reduce the risk of wrong placement of the k-wires in the medial-lateral plane since the coracoid is round shaped

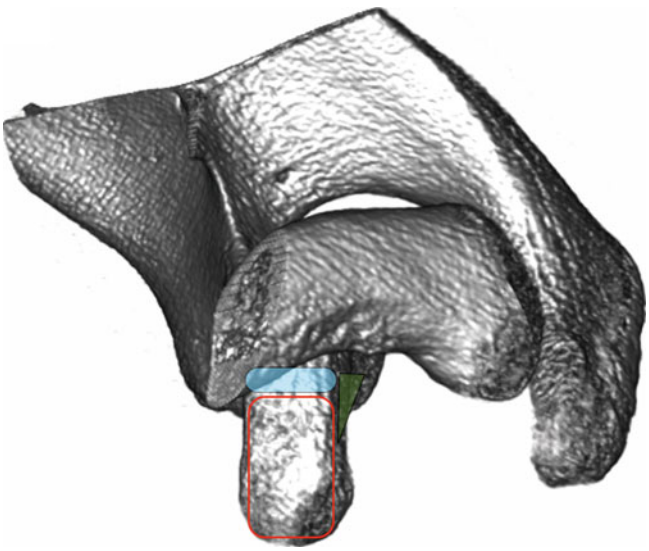

Fig. 37.6 Coracoid superior view: the clavicle is on the way for the osteotomy

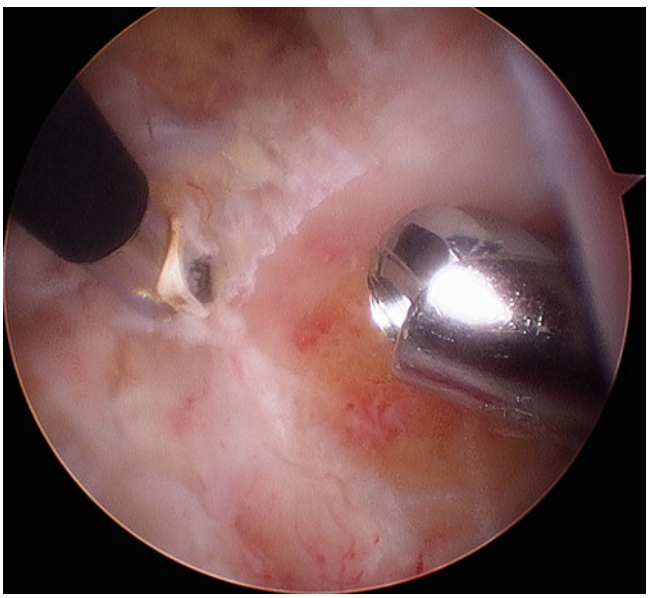

Fig. 37.7 Coracoid inferior stress riser to avoid a "spike" creating conflict with the glenoid neck (scope in the *I* portal)

to the insertion onto the lesser tuberosity. The split is bluntly completed (Fig. 37.8). (Rotate the arm internally and externally to better view and expose the muscle and tendon.)

Coracoid Transfer

The coracoid is then retrieved and firmly fixed to the cannula (in the *M* portal) using two long cannulated screws passed into the bone through the top hats. The coracoid is completely mobilized removing any remaining soft tissue attachments along the medial aspect. Graft preparation is completed by gently burring the inferior aspect and removing any spur that remains at the base of the harvested coracoid so that it will match the glenoid neck. The arm is then placed in internal rotation and forward flexion in order to reduce tension to the conjoined tendon and open the subscapularis split, so facilitating correct placement of the graft.

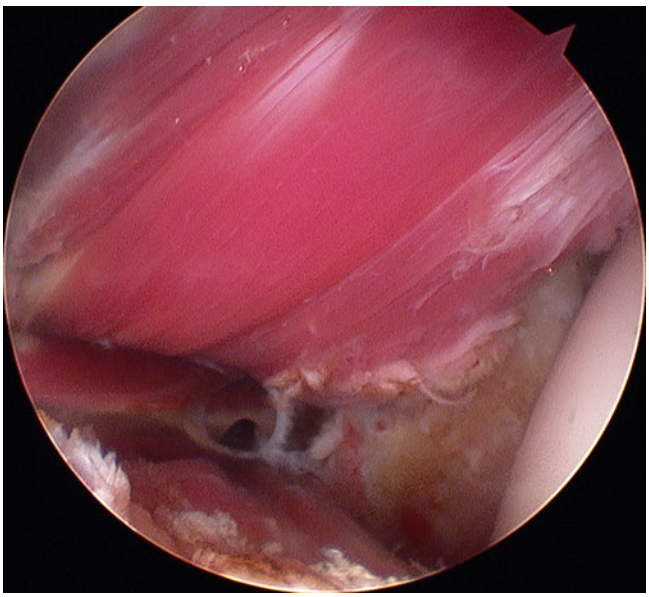

Fig. 37.8 Subscapularis blunt split and glenoid exposure

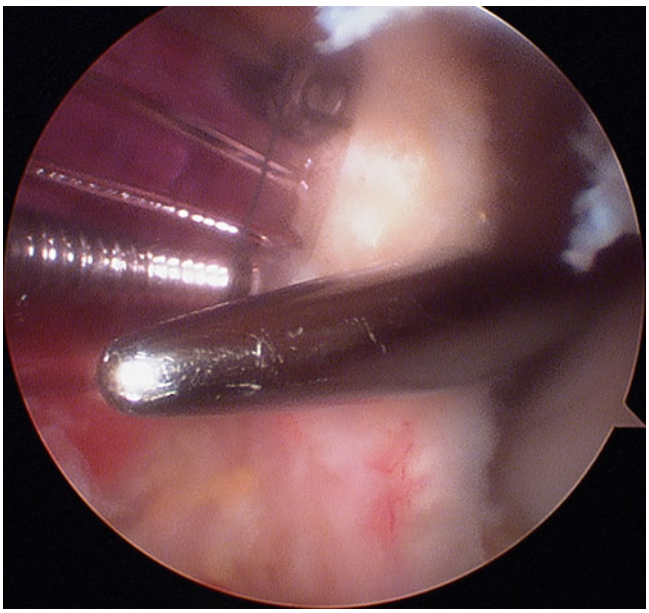

Fig. 37.9 Graft placement and medial-lateral check with a switching stick from the back to avoid proud placement of the graft

The cannula is used to manipulate and place the graft onto the glenoid neck through the subscapularis split. The switching stick from the back is used to open the split and allow passage of the graft. Graft positioning is also checked with the switching stick in the A portal. This ensures the graft is not prominent (Fig. 37.9).

Coracoid Fixation

Before coracoid fixation scapula has to be retracted posteriorly. This can be easily done by inserting an Healix 5,5 tap screw (DePuy Mitek; Raynham, MA) in the site of the coracoid osteotomy through the J portal and using it as a joy stick to control the scapula.

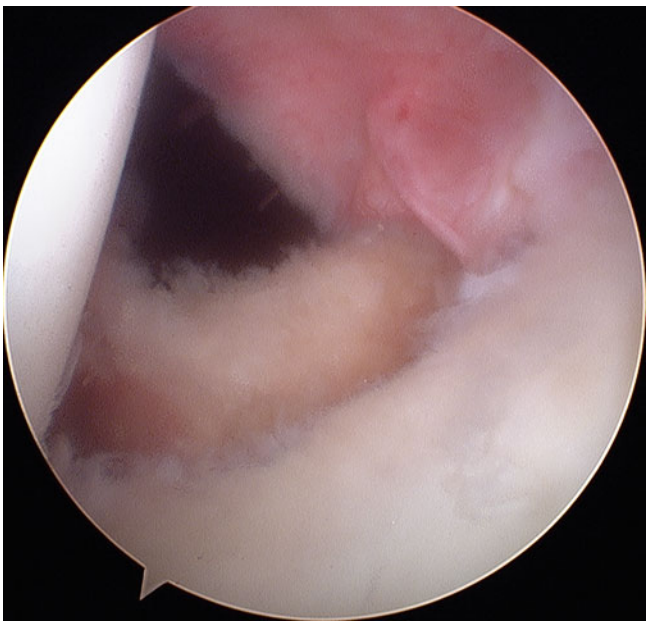

Fig. 37.10 Final check from the back (*A* portal)

With the graft in position (check for the landmarks prepared at the beginning of the procedure), two long K-wires are inserted into the cannulated screws, through the graft and glenoid, and then out of the posterior aspect of the shoulder. The K-wires are firmly held posteriorly with clamps to ensure they remain in place during drilling and fixation of the graft. K-wires in the posterior side of the shoulder are slightly divergent from the switching stick and have to be parallel to each other (if not, remove and replace one to avoid divergent screw placement).

The graft position is checked from the anterior I and posterior A portals. Ideal placement is between 3 and 5 o'clock (right shoulder). The inferior hole is drilled first to ensure good positioning of the screw in the bone. The cannulated screw is removed and the hole drilled with a specific cannulated drill. The drill is advanced slowly and screw length measurement is taken off the drill bit when the posterior glenoid cortex is felt. The inferior screw is then inserted and the sequence is repeated for the superior screw.

The screws are then alternatively tightened to ensure compression of the graft onto the glenoid neck. Overtightening may break or medialize the graft and should be avoided. The K-wires are then removed posteriorly prior to removal of the cannula anteriorly. This ensures the K-wires do not damage the brachial plexus during removal. The graft is finally checked and any prominence can be corrected with the burr (Fig. 37.10). The "sling effect" can be visualized from either the I or J portals at the end of the procedure.

Summary

The arthroscopic Latarjet procedure is a safe, reliable, and reproducible procedure in the hands of experienced and skilled arthroscopic surgeons. Nevertheless, the technique is constantly evolving to improve intraoperative safety and pitfall prevention. We recommend heeding the above hints and tricks in order to achieve good results within a reasonable operative time.

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Failed Rotator Cuff Surgery

38

Patrick J. Denard and Stephen S. Burkhart

Epidemiology

The number of rotator cuff repairs performed annually has risen in recent years [1]. Fortunately, these repairs result in satisfactory outcomes in the majority of cases [2]. However, some patients have persistent pain or disability following rotator cuff repair. Structural failure of repair in particular remains common, particularly with massive tears where single-row (SR) fixation of rotator cuff has led to a re-tear rate of approximately 69 % [3]. While functional outcome is correlated with postoperative rotator cuff integrity [2, 4, 5], remarkably many patients do quite well despite structural failure [6, 7]. In patients with persistent symptoms and/or structural failure of the repair, a careful evaluation is required to determine first if any further treatment is appropriate and then second the type of intervention that will likely benefit the patient.

Causes of Failure

The causes of failure after rotator cuff repair are multifactorial. Causes of clinical failure include wrong or missed diagnosis, aggressive postoperative rehabilitation, postoperative stiffness, and structural failure of repair.

A wrong diagnosis on initial presentation can lead to unnecessary treatment of a rotator cuff tear. One must remember that approximately 50 % of individuals over the age of 60 have a rotator cuff tear [8, 9]. It is therefore incumbent upon the surgeon to determine whether a given rotator cuff tear is indeed symptomatic. The most common wrong diagnosis is likely C6 cervical radiculopathy which can manifest as shoulder pain and should be distinguished by the presence of dermatomal pain, numbness, weakness, neck pain, and abnormal reflexes.

In our opinion the most common missed diagnosis is failure to recognize a subscapularis tendon tear at the time of surgery. Arthroscopy has provided the ability to see the tendon from the articular side which has led to increased recognition of subscapularis tendon tears. Several reports have shown that subscapularis tears are present in nearly 30 % of all arthroscopic shoulder surgeries and approximately 50 % of rotator cuff repairs [10–13]. Unfortunately, preoperative magnetic resonance imaging (MRI) has a poor sensitivity for detecting subscapularis tears. In one report, for instance, only 31 % of subscapularis tears were recognized by the radiologist prior to surgery [14]. The key therefore is arthroscopic recognition which is improved with manipulation of the arm and the use of a 70° arthroscope as will be discussed subsequently.

Aggressive postoperative rehabilitation can lead to re-tearing despite a technically adequate repair. From the moment that the rotator cuff is repaired to bone, there is a race between healing and the strength of the fixation construct. Histological evaluation of rotator cuff healing in a primate model has shown that tendon maturation following repair of the rotator cuff requires 12–15 weeks [15]. Moreover, recent clinical studies have shown that the vast majority of structural failures occur in the first 12 weeks following a repair [5, 16]. Historically it was advised that early passive range of motion was necessary following rotator cuff repair in order to prevent stiffness. However, with an arthroscopic repair the incidence of clinically important postoperative stiffness is only 3 % [17]. With these factors in mind, it is important to weigh the risks and benefits of aggressive early range of motion following an arthroscopic rotator cuff repair (ARCR). Lee et al. recently reported a group of 64 ARCRs randomized to aggressive or limited passive range of motion exercises postoperatively [18]. Strikingly, the re-tear rate was 23 % in the aggressive rehab

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group compared to only 8 % in the limited rehab group. Cuff and Pupello also reported a higher re-tear rate with early passive range of motion compared to delay of range of motion for 6 weeks [19].

Postoperative stiffness can occur after a rotator cuff repair, particularly following an open repair. Namdari and Green reported on 345 rotator cuff repairs, the majority of which were performed with an open or mini-open technique, and observed that 13.6 % of patients were "clinically stiff" at 1 year [20]. Fortunately, most patients with postoperative stiffness have a healed rotator cuff [21, 22]. These patients therefore often only require a capsular release alone, and the outcome following an arthroscopic capsular release may be similar to the final functional outcome of patients who do not require a capsular release [21].

Structural failure of repair can be attributed to both intrinsic factors and extrinsic factors. Intrinsic patient factors include age [2, 4, 23], tear size [23, 24], and biologic failure despite strong fixation [23, 25]. Boileau et al. reported the rate of healing following ARCR was 95 % for patients less than 55 year of age, 75 % for patients 55–64 years of age, and only 43 % for patients over 65 years of age [4]. Similar results were reported by Harryman et al. in a series of open repairs [2].

Larger tear size is the greatest risk factor for re-tearing. Nho et al. reported that with every centimeter of increase in rotator cuff size, the odds of a postoperative tendon defect increases over 2 times [23]. They also reported that the likelihood of failure was nearly 9 times greater for multiple-tendon tears compared to single-tendon tears. Harryman et al. reported an 80 % healing rate for single-tendon full-thickness tears, which decreased to 57 % for two-tendon tears and to 32 % for three-tendon tears [2].

Biologic failure despite strong fixation is more difficult to quantify than tear size and age but also plays a role in the rate of rotator cuff healing. Goutallier et al. reported that grade 2 (more muscle than fat) fatty degeneration of the infraspinatus was associated with a higher tear recurrence following open rotator cuff repair with suture to bone [26]. In this series, the re-tear rate was 50 % with grade 2 fatty degeneration compared to only 10 % when the fatty degeneration was grade 1 or 0 (some or no fat streaks). On the other hand, Burkhart et al. reported that with arthroscopic suture anchor fixation of the rotator cuff, 86 % of massive tears with preoperative grade 3 (muscle equals fat) or greater fatty degeneration still achieve functional improvement [27]. Efforts to augment biologic healing have led to the development of platelet-rich plasma (PRP), rotator cuff patches, and several preclinical investigations of other biologic enhancements. As of this writing, however, the impact of these enhancements is unproven. Moreover, multivariate analysis has suggested that age and tear size may be more important than biologic factors in rotator cuff healing [23].

Extrinsic factors are under the surgeon's control and include surgeon volume, biomechanical failure due to inadequate strength of the repair construct or improper repair configuration for a given tear pattern (e.g., repairing a U-shaped or L-shaped tear as if it were a crescent tear), and aggressive postoperative rehabilitation (previously discussed) leading to failure of the repair construct.

Higher surgeon volume has been associated with lower complication rates following several surgical procedures including rotator cuff repair and shoulder replacement. In a recent study the need for revision within 1 year of a primary rotator cuff repair was found to be higher for surgeons who performed less than 3 rotator cuff repairs per month [1].

The introduction of suture anchors has transferred the weak link in rotator cuff repair from the bone to the tendon [28, 29]. One of the easiest ways to improve fixation of the tendon is to therefore increase contact area (i.e., footprint restoration). Double-row (DR) rotator cuff repairs have demonstrated improved fixation characteristics compared to SR rotator cuff repairs [30]. Most early clinical studies of non-suture-bridging DR repairs have not yet revealed a better functional outcome for the patient [31, 32]. Although concern has been raised about over-tensioning resulting in medial failure following DR repair [33], overall DR repairs are clearly associated with a lower rate of structural failure compared to SR repairs. In a systematic review of 1,252 rotator cuff repairs, the recurrence rate for tears $\leq$3 cm was 19 % following SR repair compared to only 7 % following DR repair [3]. For tears larger than 3 cm, the re-tear rate increased to 45 % following SR repair versus 26 % following DR repair. Moreover, in the early clinical studies, a traditional or triangular DR repair was performed which consists of two independent rows of fixation. Current DR repairs are suture bridging in which the medial and lateral rows are linked. These suture-bridging constructs exhibit even greater load-to-failure [34] and footprint restoration [35] compared to the traditional DR repair, and early clinical results are very encouraging [36]. Similar to improvements in DR, SR repair may be enhanced. The number of fixation points can be increased with triple-loaded anchors, and stitch configuration can be altered to limit cutout (e.g., anchor-based ripstop) [37–39].

Evaluation

When structural failure occurs, the surgeon must decide if further surgery is indicated. Structural failure does not always result in clinical failure. Many patients with partial healing of the cuff and a residual defect will be much better after surgery, and in these patients, surgical intervention is not indicated. In general, patients who still have disabling pain and weakness at 9–12 months after surgery should be

evaluated for possible revision repair. A detailed history, physical examination, and evaluation of imaging are necessary in these patients. Additionally, consideration should be given to further diagnostic testing if there is suspicion for infection or neurologic injury.

History

Similar to the evaluation of patients undergoing primary rotator cuff repair, the history is important to define the cause of pain and rule out non-shoulder pathology (e.g., cervical radiculopathy). Electrodiagnostic testing should be considered if there is the possibility of neurologic injury (e.g., axillary nerve palsy) or cervical radiculopathy. Intermittent pain that is activity related is suggestive of symptoms related to the rotator cuff, whereas constant pain and/or systemic symptoms should raise suspicion for postoperative infection. Erythrocyte sedimentation rate (ESR) and C-reactive protein levels are commonly used to evaluate for postoperative infection. Unfortunately, the sensitivities of these tests are only 60 and 50 %, respectively, in the setting of postoperative rotator cuff infection [40]. If there is strong suspicion for postoperative infection, consideration should be given to a joint aspiration for cell count, gram stain, and culture. Because the majority of these infections are caused by *Propionibacterium acnes*, cultures should be retained for a minimum of 7 days [40].

The patient's previous postoperative rehabilitation protocol should also be reviewed to determine if early aggressive motion or strengthening contributed to structural failure.

Clinical Examination

Previous surgical incisions should be inspected for signs of inflammation that are suggestive of infection. It is important to determine the integrity of the deltoid attachment, particularly if the previous repair was performed with an open technique. Djurasovic and colleagues reported that 78 % of patients with an intact deltoid had a satisfactory result following revision open repair, compared to only 57 % for patients with compromised deltoid integrity [41].

Active and passive range of motion are compared to assess for postoperative stiffness. Patients with stiffness without a recurrent tear can benefit from an isolated capsular release and subacromial lysis of adhesions [21]. Pseudoparalysis is defined by active elevation of less than 90° (due to an unstable glenohumeral fulcrum rather than pain) with full passive range of motion. While recovery of pseudoparalysis following primary arthroscopic repair is approximately 75–90 % [42, 43], in our experience recovery of pseudoparalysis following revision rotator cuff repair is less than 50 % [43].

In addition to standard strength testing, several physical exam tests can be used to define the pattern of tear and prognosis for recovery. The inability to maintain external rotation with the arm in 20° of abduction and maximal external rotation is considered a positive external rotation lag sign and has been reported to have a sensitivity of 65 % for detecting lesions extending into the infraspinatus tendon [44]. Walch et al. reported that both the inability to maintain external rotation with the arm at the side (dropping sign), and the inability to externally rotate the arm from a position of 90° abduction with the elbow flexed 90° (hornblower's sign), have a 100 % sensitivity for detecting grade 3 or 4 fatty degeneration of the infraspinatus and teres minor [45]. In one study the bear-hug test has been shown to have the highest sensitivity for detecting subscapularis tears [13]. Given the inaccuracy of MRI at detecting subscapularis tendon tears [14, 46], a positive bear-hug, belly-press, or lift-off test should alert the surgeon to a previously missed subscapularis tear.

Imaging

Plain radiographs are obtained to evaluate the glenohumeral joint space and for the presence of proximal migration and adaptive changes of the proximal humerus (i.e., femoralization) and undersurface of the acromion (i.e., acetabularization). Proximal migration alone is not a contraindication to repair and can be reversed following ARCR [47]. On the other hand, advanced adaptive rotator cuff arthropathy changes are a contraindication to repair. Radiographs should also be evaluated for other causes of pain (e.g., chondrolysis, loose anchors, acromial fracture).

Advanced imaging is used to assess tear pattern and rotator cuff muscle quality. Ultrasound is cost-effective and has less postoperative hardware artifact compared to MRI. However, it is operator dependent, less familiar to most orthopedic surgeons, and does not provide a thorough evaluation of the glenohumeral joint. Postoperative MRI is less accurate than MRI in the primary setting. In one study there was a 91 % sensitivity of MRI for detecting a recurrent rotator cuff tear, but the specificity was only 25 % [48]. In other words, MRI has the tendency to overdiagnose recurrent rotator cuff tears. In this study, MRI also demonstrated a poor ability to assess rotator cuff tear size. Compared to tear presence and size, MRI is likely more accurate in the postoperative setting for determining rotator cuff muscle quality. T1 parasagittal images should be inspected for the presence and severity of fatty degeneration, which correlates to some extent with prognosis. Goutallier et al. originally classified fatty degeneration into five categories: grade 0, no fatty deposit; grade 1, some fatty streaks; grade 2, more muscle

than fat; grade 3, muscle equals fat; and grade 4, less muscle than fat [26].

Another consideration in the preoperative imaging studies is to evaluate for the amount of the greater tuberosity that is available for additional suture anchor placement. If large numbers of anchors are already present or if cystic cavitation has occurred around the anchors, then the surgeon may have to remove some or all of the existing anchors and bone graft the defects.

Treatment: Indications, Contraindications, and Decision-Making Algorithm

Several studies have shown that the majority of individuals obtain functional improvement despite re-tear following rotator cuff repair [2, 6, 7]. The majority of such re-tears are smaller than the original tear, suggesting that partial healing can improve function. Jost et al. reported on the long-term outcome of 20 re-tears following an open repair [6]. At an average follow-up of 7.6 years, Constant scores did not demonstrate any significant deterioration compared to values at 3.2 years, and 95 % of the patients remained satisfied with their result. However, negative prognostic factors included a decrease in the acromiohumeral interval and progression of glenohumeral arthritis and fatty degeneration. Notably, the six patients with extension of the re-tear into the infraspinatus tendon had an age-adjusted Constant score of 75 % compared to 94 % for the patients with an intact infraspinatus tendon.

Revision repair is considered for individuals with persistent symptoms (9–12 months) despite nonoperative management in whom a comprehensive evaluation has ruled out infection or non-shoulder diagnoses and radiographs do not demonstrate advanced adaptive changes. Additionally, if postoperative stiffness alone is the cause of disability, we simply proceed with an arthroscopic capsular release. Although Goutallier et al. classified grade 2 as a turning point in prognosis for recovery [26], there is evidence from the primary repair literature that individuals with grade 3 and even grade 4 fatty degeneration can obtain improvement after ARCR with suture anchor fixation. Burkhart et al. reported that 100 % of patients with grade 3 fatty degeneration obtained functional improvement following ARCR [27]. For grade 4 degeneration, however, only 40 % of patients demonstrated substantial functional improvement. Therefore, for individuals with grade 4 fatty degeneration, the decision to attempt revision rotator cuff repair should be carefully considered. For a young individual without adaptive changes of the proximal humerus, a revision repair may be reasonable following proper counseling of the prognosis. The risk of complications from a revision ARCR is low, and many patients wish to attempt this following an informed discussion even if there is grade 3 or greater fatty degeneration. On the other hand, for patients over the age of 70 with grade 4 fatty degeneration and severe dysfunction, a reverse total shoulder arthroplasty may provide a more predictable functional outcome [49]. Latissimus dorsi transfer has been considered an option for young patients with an "irreparable" posterosuperior tear. However, in our experience this scenario rarely exists as most tears in young patients are repairable and even partial repairs function quite well. We therefore do not consider this nonanatomic procedure for our patients until we have at least attempted a revision repair.

Arthroscopic Treatment: Surgical Technique

Both open and arthroscopic techniques have been reported for revision rotator cuff repair. Additionally, a mini-open repair may be an option, but there are no published reports on this technique for revision rotator cuff repair. Our preference is an arthroscopic technique. Compared to an open or mini-open approach, an arthroscopic technique creates minimal trauma to the deltoid origin, allows a complete evaluation and treatment of the frequent concomitant pathology (e.g., biceps pathology or capsular thickening) within the glenohumeral joint, and provides a better evaluation of the tear pattern and its mobility. The major limitation of an arthroscopic approach is the high technical demand of the procedure. However, with any technique, revision rotator cuff repair is more difficult and time consuming than primary repair. The rotator cuff is often retracted and adhesed to the glenoid, coracoid, and undersurface of the acromion. As previously alluded, the emphasis for large and massive tears is to restore balanced force couples which means that repair of the subscapularis and infraspinatus tendons is paramount.

Patient Positioning

Following induction of general anesthesia, the patient is placed in the lateral decubitus position. The trunk is secured using a beanbag; anteriorly it is important to keep the edge of the beanbag clear from the operative site, particularly for repairing the subscapularis. An axillary roll under the nonoperative arm protects the brachial plexus. The legs are flexed and padded with pillows between and beneath the patient's legs. The thorax and legs are additionally secured to the operating room table using tape. A warming blanket helps maintain core body temperature, particularly for longer, more complex procedures. Following an exam under anesthesia, the operative site is prepped and draped in the standard fashion. The surgeon must ensure there is adequate exposure of the entire shoulder, particularly anteriorly and

posteriorly. The arm is suspended with a Star Sleeve Traction system (Arthrex, Inc., Naples, FL) and balanced suspension of 5–10 lb to maintain the arm in 30° abduction and 20° forward flexion. By varying the amount of abduction and rotation, an assistant opposite to the surgeon can maximize exposure and visualization.

Portals

Portal placement is critical to angle of approach, and for that reason, with the exception of the initial posterior portal, we use an 18-gauge spinal needle to precisely establish all portals in an outside-in fashion. The 3 standard portals that are used during ARCR are: posterior, anterior, and lateral subacromial. An anterosuperolateral portal is also required for subscapularis tendon repair. In addition to the most commonly used portals for arthroscopic repair, one should not hesitate to establish accessory portals for anchor placement or suture passage if the standard portals (combined with manipulation of the arm) do not afford a proper angle of approach. We commonly place anchors, for instance, through small percutaneous incisions to achieve the proper "deadman" angle [50].

Many surgeons create a posterior portal 1–2 cm inferior and 1–2 medial to the posterolateral corner of the acromion. We feel this location is too lateral and superior. During longer cases such as massive rotator cuff repairs, subcutaneous swelling will cause the skin incision to shift superiorly and laterally. We establish a posterior portal by palpating the soft spot of the glenohumeral joint and enter the joint at or just below the equator of the humeral head. The exact position varies from patient to patient but is approximately 4 cm inferior and 4 cm medial to the posterolateral corner of the acromion. This portal is used for initial glenohumeral arthroscopy, and the same skin puncture is used for viewing and working in the subacromial space.

The anterior portal is established using an outside-in technique just superior to the lateral half of the subscapularis tendon for diagnostic glenohumeral arthroscopy. This same skin puncture can be used as an anterior working portal and inflow subacromial portal and can be used during distal clavicle resection if indicated.

The lateral subacromial portal is approximately 4 cm lateral to the lateral aspect of the acromion, in line with the posterior border of the clavicle. One must ensure that the portal is parallel to the undersurface of the acromion. This portal serves as a viewing and working portal in the subacromial space.

The anterosuperolateral portal is created in the presence of a subscapularis tear or if it is necessary to perform a biceps tenodesis. It is established through the rotator interval just anterior to the supraspinatus tendon and directly above the long head of the biceps. The point of entry is approximately 1–2 cm lateral to the anterolateral corner of the acromion. Placement should allow a 5–10° angle of approach to the lesser tuberosity and should be parallel to the subscapularis tendon. This angle of approach is also ideal for performing arthroscopic coracoplasty.

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

Diagnostic arthroscopy is performed through a posterior viewing portal with an arthroscopic pump maintaining pressure at 60 mmHg. It is important to closely evaluate for a tear of the subscapularis tendon and perform a repair if the tendon is torn. Visualization of the subscapularis footprint requires a 70° arthroscope and can also be improved with internal rotation of the humerus as well as a posterior lever push by an assistant. In the case of a retracted tear, the upper border of the subscapularis can be located by the "comma sign" which is composed of portions of the superior glenohumeral ligament and the medial head of the coracohumeral ligament [51]. This tissue not only helps identify the subscapularis tendon but also connects the superolateral subscapularis tendon to the anterolateral supraspinatus tendon (Fig. 38.1). As such, subscapularis repair facilitates repair of the supraspinatus and in fact is critical because failure to repair the subscapularis will place the anterior supraspinatus tendon under greater tension, potentially leading to repair failure (Fig. 38.2) [52]. If the subscapularis tendon is not immediately visible at the front of the joint, it is probably retracted medially to the level of the glenoid margin. In rare circumstances the subscapularis is not immediately identifiable due to significant scarring. In this scenario, a window is created just anterior to the glenoid above the mid-glenoid notch, and dissection is carried medially to the base of the coracoid where the subscapularis can be reliably identified (Fig. 38.3).

A systematic dissection of the bony landmarks and excavation of the rotator cuff are required to identify the posterosuperior tear margins. Identifying the scapular spine will help the surgeon delineate the supraspinatus from the infraspinatus. While viewing through the lateral portal, the rotator cuff is identified. For tears adhesed to the undersurface of the acromion, one technique for excavating the cuff is to place a 4.5 mm shaver through the posterior portal, in a plane just below the acromion, and aiming just lateral to the scapular spine. When the scapular spine is palpated with the tip of the shaver, it is swept laterally, maintaining its plane just below the acromion, until the tip of the shaver blade penetrates through the fibrous tissue as it thins out laterally. This maneuver preserves whatever rotator cuff might have been encased

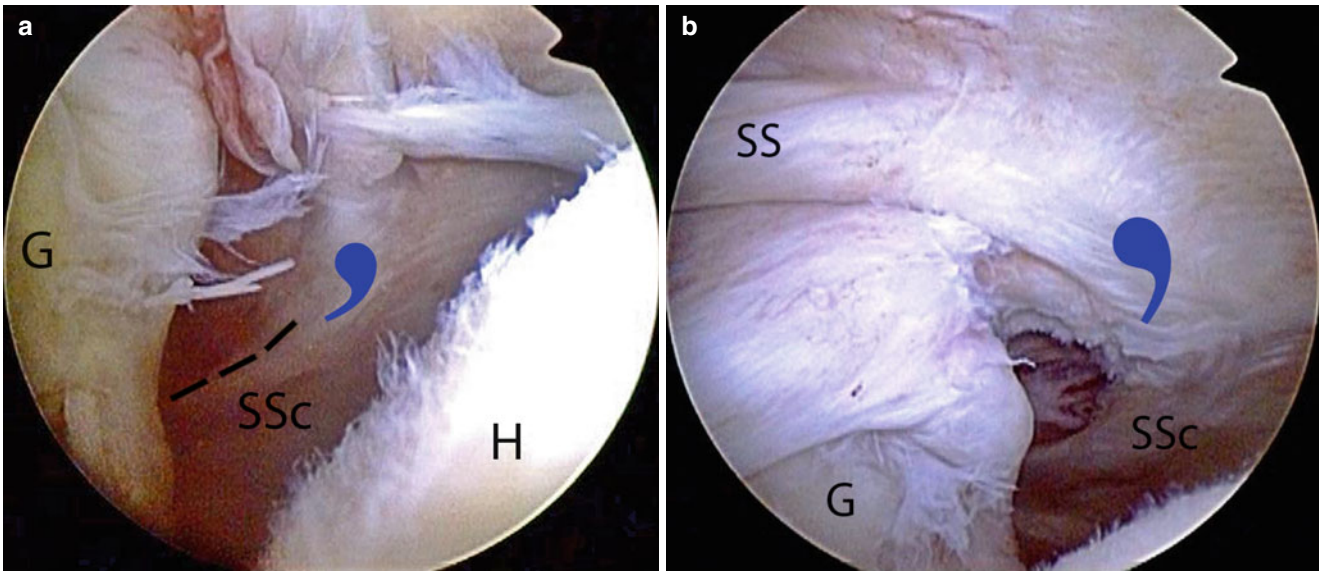

Fig. 38.1 (**a**) Right shoulder, posterior viewing portal, demonstrates the comma sign in an individual with a massive retracted rotator cuff tears. The comma sign leads to the superolateral border of the subscapularis tendon (*outlined by dashed black lines*). (**b**) Same shoulder demonstrates how the comma tissue connects the subscapularis and supraspinatus tendons. Subscapularis tendon repair will therefore facilitate supraspinatus tendon repair. *G* glenoid, *H* humerus, *SS* supraspinatus tendon, *SSc* subscapularis tendon, *blue comma symbol* comma sign (Reproduced with permission from Burkhart et al. [67])

within the scar tissue that had become adhesed to the acromion (Fig. 38.4). The shaver blade then completes the dissection of the lateral edge of this soft tissue envelope from the acromion. After the bony landmarks have been skeletonized, residual bursal leaders (which represent a *false cuff*) must be divided and debrided back to tendon (Fig. 38.5).

Step-by-Step Procedure

In most revision rotator cuff repair cases, we perform an arthroscopic biceps tenodesis or tenotomy. We address the biceps for two reasons. First, the biceps tendon has been shown to be an important source of pain in patients with primary rotator cuff tears [53] so we have a lower threshold to address the biceps in the revision setting. Second, many revision repairs involve the subscapularis tendon, and poorer outcomes have been associated with attempts to retain the biceps tendon in the setting of subscapularis repair [54]. The choice between tenotomy and tenodesis is based on functional demands and cosmetic concern, but we prefer tenodesis in most cases. Two half-racking #2 FiberWire (Arthrex Inc., Naples, FL) sutures are placed as traction sutures in the biceps tendon, and then a tenotomy is performed. The biceps is next exteriorized, secured with a whipstitch, sized, and left for later tenodesis. This sequence of biceps preparation allows the biceps to retract out of the way and allows greater access to the subscapularis insertion for subsequent repair, where attention is now turned.

Assessment of the subcoracoid space is an essential component of subscapularis repair. The coracoid tip is located while working through the anterosuperolateral portal and viewing through a posterior portal. At the level of the coracoid tip, a shaver is used to make a window in the rotator interval just superior to the subscapularis tendon. The medial sling of the biceps and the superior glenohumeral ligament should be preserved laterally. Once the coracoid tip is identified, the posterolateral aspect of the coracoid is skeletonized while viewing with a 70° arthroscope. The width of the coracohumeral interval is estimated by comparing it to the known width of a 5 mm shaver. An interval of less than 6 mm is considered stenotic and is an indication for coracoplasty which is performed with a high-speed burr [55, 56].

Next, a combination of electrocautery, ring curettes, shaver, and burr are used to prepare the lesser tuberosity bone bed to a bleeding base. Tears of the upper portion of the subscapularis require minimal mobilization and only one suture anchor. Our preferred technique in these cases is a knotless construct which uses a FiberTape suture (Arthrex, Inc., Naples, FL) placed through an anterosuperolateral portal and a BioComposite SwiveLock C anchor (Arthrex, Inc., Naples, FL) placed through an anterior portal [57]. For a retracted subscapularis tear, a 3-sided release of the subscapularis is required. An anterior release is achieved

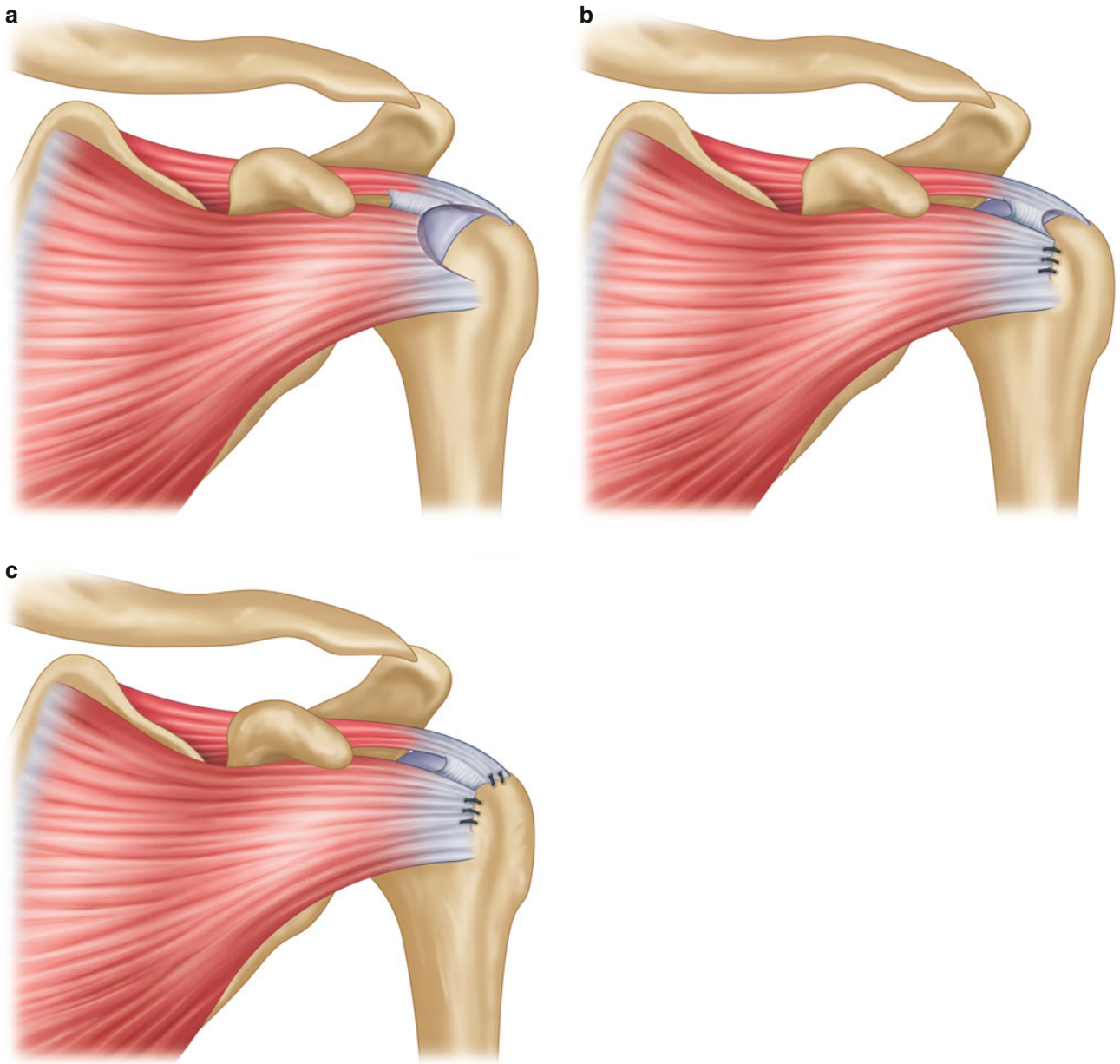

Fig. 38.2 Schematic of the relationship between subscapularis repair and supraspinatus repair. (**a**) Massive retracted and contracted tear of the subscapularis and supraspinatus tendons. (**b**) Repair of the subscapularis partially reduces the supraspinatus retraction. (**c**) Repair of the supraspinatus can then be accomplished with minimal tension (Reproduced with permission from Burkhart et al. [67])

by skeletonizing the posterolateral aspect of the coracoid. Release of the superior border of subscapularis from the coracoid base is bluntly achieved with a 30° arthroscopic elevator. The posterior release is achieved by freeing adhesions between the anterior glenoid neck and the posterior surface of the subscapularis tendon. Following mobilization the subscapularis is repaired to bone. For complete tears (i.e., full-thickness tears of 100 % of the subscapularis tendon), at least 2 anchors (BioComposite Corkscrew FT; Arthrex Inc., Naples, FL) are required, and a knotless technique should not be used. The inferior anchor is placed first, and sutures are passed in a mattress configuration with a retrograde or antegrade instrument, followed by placement of the superior anchor and suture passage. Sutures are then tied sequentially with a Surgeon's Sixth Finger Knot Pusher (Arthrex Inc., Naples, FL) from inferior to superior. The biceps tenodesis is then completed if indicated. It is also acceptable to medialize the subscapularis footprint up to 5–7 mm to maximize

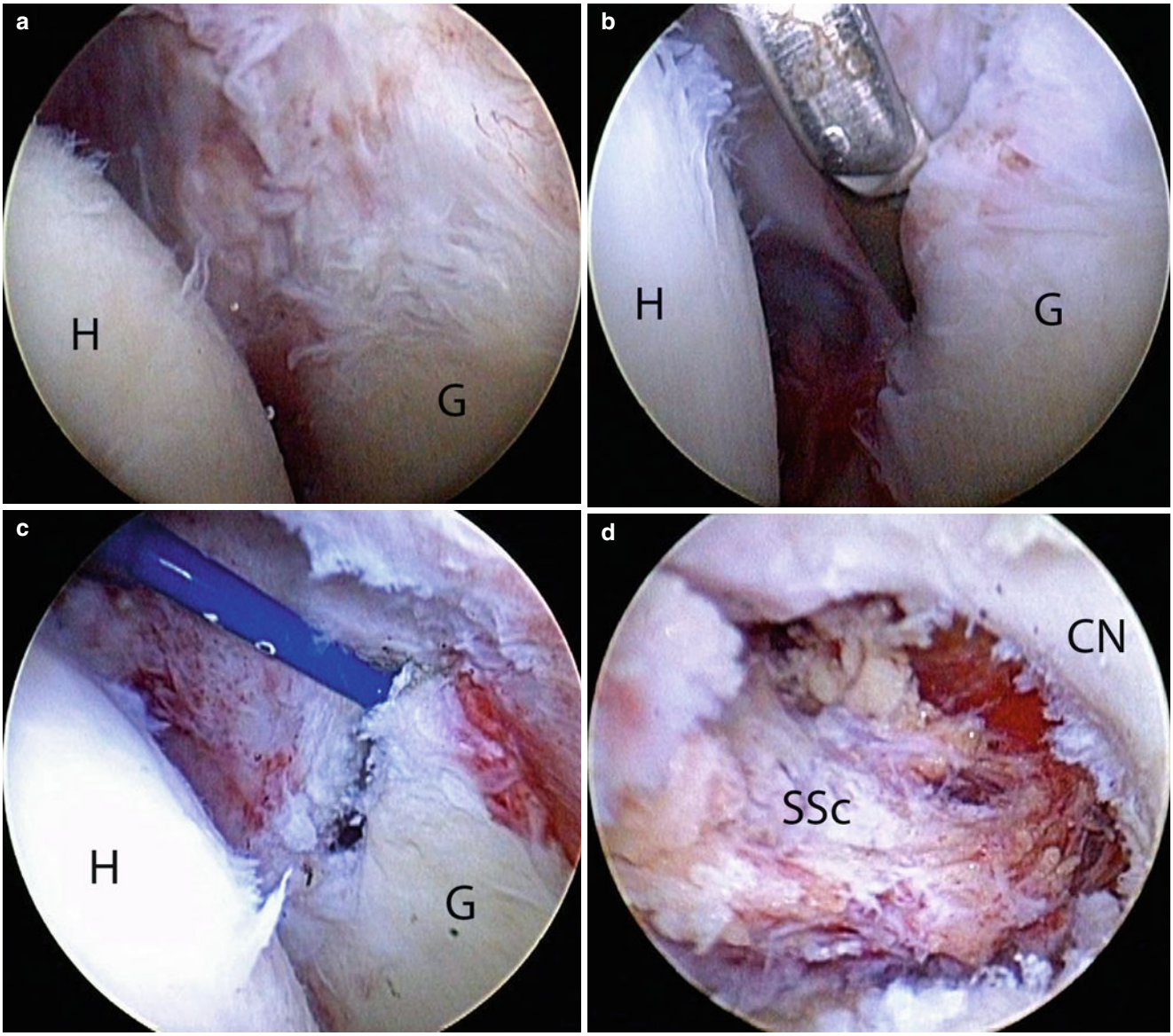

Fig. 38.3 Left shoulder, posterior viewing portal demonstrates excavation of retracted adhesed subscapularis tendon tear when the comma sign is not readily visible. (**a**) In this retracted adhesed tear, the comma sign cannot be visualized. (**b**) A window is created anterior to the glenoid above the mid-glenoid notch. (**c**) Dissection is carried medially with an electrocautery device. (**d**) View with a 70° arthroscope shows identification of the subscapularis tendon inferior to the coracoid neck. *CN* coracoid neck, *G* glenoid, *H* humerus, *SSc* subscapularis tendon (Reproduced with permission from Burkhart et al. [67])

tendon-to-bone contact and minimize resting muscle tension [58]. Attention is then turned to the posterosuperior rotator cuff.

After identification of the posterosuperior rotator cuff margins, the bone bed is prepared for repair. At this stage, remaining sutures or anchors from previous surgeries are removed as needed. Often previous anchors can be retained, and new anchors can be placed adjacently. However, anchor removal can sometimes be necessary either to remove a prominent anchor or to provide sufficient biologic surface for tendon-to-bone healing. If the inside of the anchor is visible and the type of implant is known, the inserter from that implant can be used to remove the anchor. Otherwise, an OATS harvester (Arthrex, Inc., Naples, FL) larger than the diameter of the anchor can be used. The harvester is impacted around the anchor then turned counterclockwise to remove the anchor. This technique will remove some additional bone surrounding the anchor which can be retained and replaced into the defect. Additionally, any remaining defect can be filled with an allograft compaction grafting technique using the same OATS harvester.

Next, an assessment is made of tear pattern and mobility. If there is insufficient mobility of the tendon to reach the bone bed, a slight amount of mobility can be gained with a capsular release beneath the supraspinatus and infraspinatus tendons.

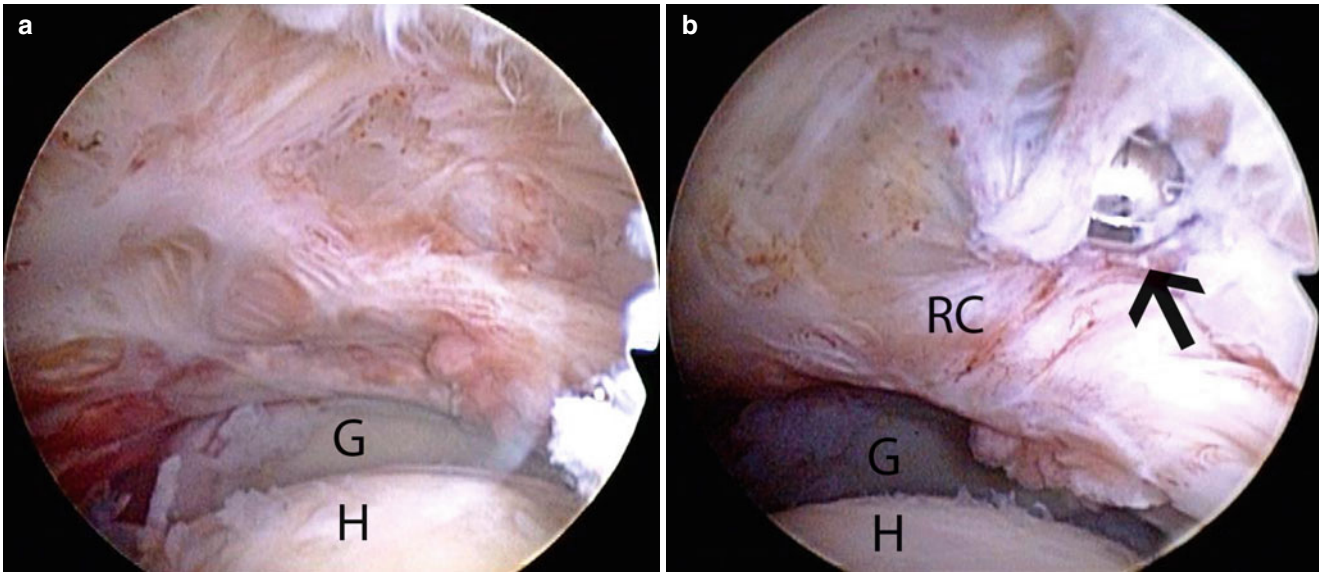

Fig. 38.4 (**a**) Right shoulder, lateral subacromial viewing portal demonstrating a massive contracted rotator cuff tear which is adhesed to the undersurface of the acromion. (**b**) A shaver (*black arrow*) is inserted through a posterior portal and used to "bounce off" the scapular spine, just lateral to the bone, and it is then swept laterally to penetrate the fibrous tissue laterally as it defines the plane about the rotator cuff. *G* glenoid, *H* humerus, *RC* rotator cuff (Reproduced with permission from Burkhart et al. [67])

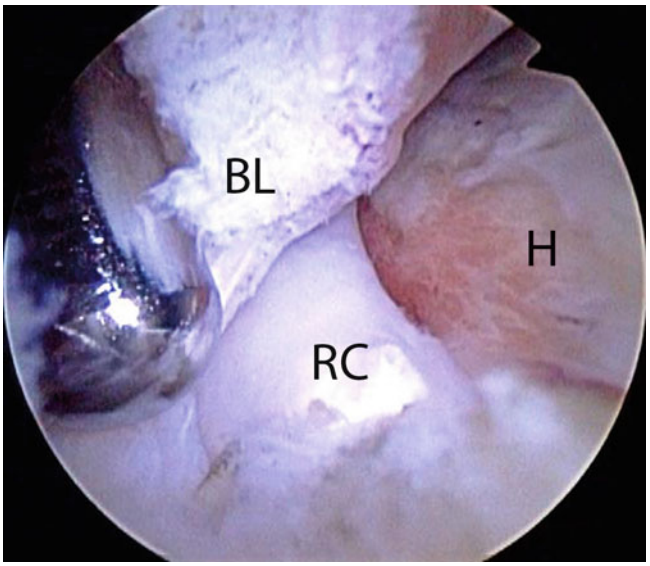

Fig. 38.5 Right shoulder, lateral subacromial viewing portal, demonstrates a bursal leader which is excised with a shaver to delineate the true rotator cuff margins. *BL* bursal leader, *H* humerus, *RC* rotator cuff (Reproduced with permission from Burkhart et al. [67])

If the capsular release fails to provide sufficient additional excursion, interval slides are required in order to obtain tendon-to-bone repair [41, 59–61]. In most cases, SR suture anchor repair is possible after interval slides, and in some cases the additional excursion is great enough to allow DR repair. However, if only partial repair is possible, as much of the tendon as possible is repaired to bone with emphasis on the infraspinatus. Additionally, a side-to-side repair of supraspinatus to infraspinatus may help to reestablish a crescent-shaped cable that can transmit a distributed load to the anterior and posterior anchor points of the partial repair.

Postoperative Care

Following arthroscopic revision rotator cuff repair, the shoulder is immobilized in a sling for 6 weeks. Active elbow flexion and extension are allowed without resistance, but no shoulder motion is permitted. At 6 weeks, the sling is removed and the patient begins passive overhead elevation and external rotation. Passive internal rotation is delayed until 4 months postoperatively because internal rotation places very high strains on the anterior half of a repaired supraspinatus tendon. At 4 months postoperatively, strengthening and passive internal rotation are allowed. Strengthening gradually progresses as tolerated, and full activities are allowed at 1 year postoperatively.

Literature Review

The results of open surgical management of failed rotator cuff repairs have been mixed in the few series reported in the literature. In 1984, DeOrio and Cofield reported the results of 27 revision repairs, 8 of which were massive tears [62]. Overall, only 17 % of patients obtained a good result, and among the massive tears only 12.5 % of patients achieved a good result.

The results led to the authors to recommend "an immediate glenohumeral arthrodesis" if the recurrent tear was massive in size. More recently, Djurasovic et al. reported more encouraging results in 80 revision rotator cuff repairs, 51 of which were classified as large or massive tears [41]. Overall, active elevation improved from 105° preoperatively to 130° postoperatively, and 69 % of patients achieved a satisfactory result. In large or massive tears, a satisfactory (excellent, good, or fair) functional outcome was obtained in 67 % of cases. A satisfactory result was associated with an intact deltoid origin, good-quality rotator cuff tissue (defined subjectively), preoperative active elevation of the arm above 90°, and only one prior procedure.

The results following arthroscopic revision rotator cuff repair have been encouraging. Lo and Burkhart reported on 14 cases, 11 involving massive tears, at a mean follow-up of nearly 2 years [63]. The mean postoperative University of California Los Angeles (UCLA) score was 29, good or excellent results were achieved in 64 % of patients, and 93 % of patients were satisfied with the procedure.

Keener et al. reported on 12 arthroscopic revision rotator cuff repairs with a mean follow-up of 33 months [64]. Nine of 21 patients had a single-tendon tear, 11 had a 2-tendon tear, and 1 had a 3-tendon tear. Unfortunately, it was not noted whether the tears were complete or not, making classification between non-massive and massive impossible. Following revision, American Shoulder and Elbow Surgeons (ASES) scores had improved from 40 to 73, and forward elevation had improved from 130° to 147°. Postoperative ultrasound performed on all patients demonstrated that 48 % percent of shoulders had an intact repair. Seventy percent of the single-tendon repairs were intact, compared with 27 % of the two-tendon tears ($p=0.05$). The average age of the 10 patients with an intact repair was 52 years, compared with 59 years for the 11 patients with a recurrent tear. Postoperative healing was associated with an improved functional outcome according to age-adjusted Constant scores (84 for healed vs. 69 for recurrent tears).

Piasecki et al. reported the results of 54 arthroscopic revision rotator cuff repairs [65]. Only 4 patients had a massive rotator cuff tear. At a mean of 31 months postoperative, ASES scores had improved from 44 to 68, and forward elevation had improved from 121° to 136°. An association was noted between poor outcome and female gender or active preoperative forward elevation less than 120° prior to revision.

Although tear size has been associated with the risk of recurrence following primary repair, size of tear is not a contraindication to revision repair. Recently Lädermann et al. reported on a repair cohort of 21 non-massive tears and 53 massive tears with a mean follow-up of 63 months [66]. Overall, the mean ASES score improved from 47 to 75, the mean UCLA score improved from 17 to 26, and 78 % of patients were satisfied with their surgery. No statistical difference was observed in the functional outcome between massive and non-massive repairs. Only 6 patients (8 %) required additional surgery within the follow-up period of over 5 years. Interestingly, there was functional improvement by ASES scores between the 1-year postoperative visit and final follow-up. This finding is important for several reasons. First, from a rehabilitation standpoint, revision repairs appear to take a longer time to reach full functional improvement than most primary repairs. This information is useful for the surgeon monitoring postoperative progress, as well as for counseling patients regarding the timeline for recovery. More importantly, this sustained improvement suggests that revision arthroscopic repair of recurrent tears is durable in the long term. Similar to the findings of Piasecki et al., female gender and preoperative forward elevation less than 135° were associated with a poorer result.

Summary

Structural failure is not uncommon following rotator cuff repair. Intrinsic and extrinsic factors contribute to failure of a repair. A careful evaluation is required to determine which patients will benefit from a revision repair since many patients will maintain functional improvement despite recurrence. When indicated, revision repair is technically challenging. Encouraging results have recently been reported for revision repair with arthroscopic techniques.

Box 38.1: Tips and Tricks

In many revision situations, there may be deficient tissue involving either the tendon or bone or both. One construct we have found particularly useful is a load-sharing ripstop repair which is useful when there is insufficient tendon length or mobility to achieve a suture-bridging repair [38, 39]. One or 2 suture tapes (FiberTape; Arthrex, Inc., Naples, FL) are first placed as inverted mattress stitches in the rotator cuff, 3 mm lateral to the musculotendinous junction. Then, 2 anchors are placed approximately 3–5 mm lateral to the articular margin as in a SR repair. The sutures from the medial anchors are then passed as simple stitches, medial to the suture tapes. Next, the suture tape limbs are secured laterally using 2 knotless anchors (BioComposite SwiveLock C; Arthrex, Inc., Naples, FL). Importantly, care is taken to secure the suture tape limbs so that they surround the free suture limbs from the medial anchors. Finally, the simple sutures from the medial anchors are tied. We have had early clinic success with this repair construct in the setting of very difficult cases. Remarkably, the majority of failures occurred via suture anchor failure, potentially re-transferring the weak link in rotator cuff repair back to bone fixation.

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Arthroscopic Suprascapular Nerve Release

39

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Epidemiology

Suprascapular neuropathy is a disorder characterized by shoulder pain and/or weakness of the supraspinatus and infraspinatus muscles of the rotator cuff. Suprascapular neuropathy may present with a variety of clinical symptoms including pain and weakness in forward flexion, abduction, and external rotation of the shoulder, muscle wasting, as well as sensory disturbances about the superior and posterior aspects of the shoulder. Diagnosis is usually suspected in patients presenting with pain in the superior, posterior, or posterolateral aspect of the shoulder and isolated atrophy and/or weakness of the supraspinatus and/or the infraspinatus. This condition can also be present and not produce any symptoms, such as in volleyball players who may develop marked atrophy of the infraspinatus and external rotation weakness from traction on the suprascapular nerve without any pain, loss of function, or reduced efficiency when competing [1]. Additionally, the frequent existence of concomitant shoulder disorders can make diagnosis of suprascapular neuropathy difficult.

Suprascapular neuropathy was described in the French literature as early as 1936 [2]. In 1952, Schilf reported the first evidence of compression of the suprascapular nerve [3], and Clein subsequently described open decompression of the suprascapular notch in 1975 [4]. Initially only sparse mention of the disorder was found in the published literature. The largest case series included 53 patients [5] and a meta-analysis published in 2002 describes 88 published reports of the disorder between 1959 and 2001 [6]. The true incidence of suprascapular neuropathy is unknown, but an increasing understanding of the disorder and refinements in diagnostic testing and imaging has subsequently heightened awareness and diagnosis.

Historically viewed as a diagnosis of exclusion, the true incidence of suprascapular neuropathy is unknown. While unproven, estimates describe an incidence of 1–2 % in all patients presenting with shoulder pain [7]. The incidence may be even higher as one study found an incidence of 4.3 % (92/937) electrodiagnostically confirmed suprascapular neuropathy in a shoulder referral practice [8].

Over the last decade, however, numerous studies revealed a variety of potential causes of suprascapular neuropathy. Specific at-risk individuals include overhead athletes such as professional volleyball players with a reported incidence of as high as 34 % [9], but many of these overhead athletes are often asymptomatic [1]. An association of suprascapular neuropathy and massive rotator cuff tears has also been established with an incidence of 8–27 % in this cohort of patients [10–12]. Compression can occur at the suprascapular or spinoglenoid notch from a space-occupying lesion such as a ganglion cyst or tumor [13–15], trauma to the neck or scapula [16–19], or the associated traction on the nerve caused by rotator cuff tears [12] and repetitive overhead activities [1, 9, 20–22]. The association between labral tears and paralabral cysts causing suprascapular neuropathy in both radiographic and clinical studies is well established [23–27].

Other, less common entities include viral neuritis (Parsonage-Turner syndrome) [28], intimal damage to the suprascapular artery leading to microemboli in the vasa nervorum [22], glenohumeral dislocation [17–19], fractures

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about the shoulder girdle [16, 29, 30], penetrating injury to the shoulder, and surgical procedures requiring a posterior approach to the scapula [31].

Pathophysiology

The suprascapular nerve arises from the upper trunk of the brachial plexus with contributions from the C5, C6, and occasionally the C4 nerve root and is responsible for motor innervation to the supraspinatus and infraspinatus (Fig. 39.1). The nerve travels posterior to the clavicle in an oblique fashion across the superior border of the scapula and enters into the suprascapular notch, traveling in an anterior to posterior direction. Generally, the nerve passes beneath the transverse scapular ligament in the suprascapular notch while its associated artery travels over the ligament. The suprascapular notch morphology and variants are well described including complete absence of a notch [32]. The suprascapular ligament forms the roof of the suprascapular notch and may even be ossified [33] (Fig. 39.2). Individual fascicles branch off to the supraspinatus, and the nerve continues to the spinoglenoid notch of the scapula on the undersurface of the supraspinatus muscle belly. After supplying the supraspinatus, the nerve passes beneath the spinoglenoid ligament (inferior transverse ligament) and takes an abrupt turn around the base of the scapular spine into the infraspinatus fossa where it provides branches to the infraspinatus muscle. The spinoglenoid ligament has been observed in 3–100 % of cadavers [34–37].

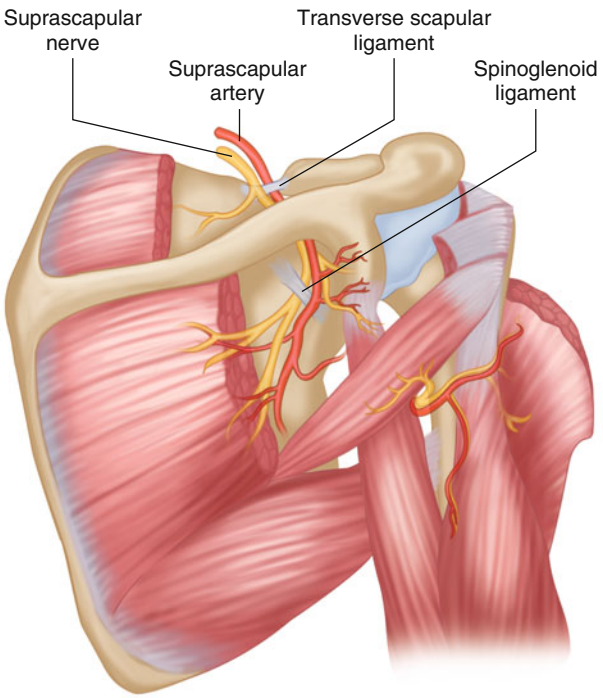

Fig. 39.1 Origin of the suprascapular nerve from the upper trunk of the brachial plexus (Modified from Boykin et al. [68])

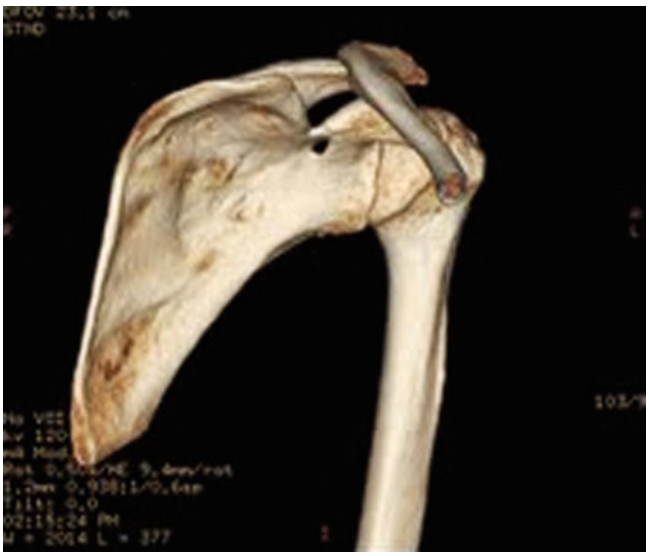

Fig. 39.2 Completely ossified suprascapular notch

Though the suprascapular nerve has historically been considered a pure motor nerve, cadaveric studies demonstrated sensory branches to the glenohumeral joint, acromioclavicular joint, coracoacromial ligament, and the skin [38, 39]. One anatomic study demonstrated a glenohumeral sensory branch present in 87 % of 31 cadavers and an acromial sensory branch in 74 % [40]. An improved and evolving understanding of the sensory contributions of the suprascapular nerve explains the associated pain resulting from compression, injury, or traction to this structure. Additional studies indicate the suprascapular nerve may supply up to 70 % of the sensation of the shoulder [41], and this concept is further confirmed by clinical investigations demonstrating improved postoperative pain after suprascapular nerve block in patients undergoing shoulder surgery [42, 43].

A number of mechanisms have been proposed to be a potential cause of injury to the suprascapular nerve. The types of injury to the nerve can be loosely classified into three categories: transection, traction, or compression. Transection injuries occur as a result of penetrating trauma or iatrogenic surgical causes. Traction injuries occur as a result of stretch phenomenon injuries. Described traction etiologies include overhead athletes who perform repeated shoulder abduction and external rotation motion and retracted rotator cuff tears. Repetitive overhead athletes such as professional volleyball players clearly are at risk for this type of injury [9, 20, 34]. A proposed mechanism is tightening of the spinoglenoid ligament when the shoulder is in a position of overhead throwing, resulting in increased pressure on the nerve [35].

Retracted supraspinatus and infraspinatus tears are also implicated in traction neuropathy. This has been confirmed clinically by several series including Mallon et al. and

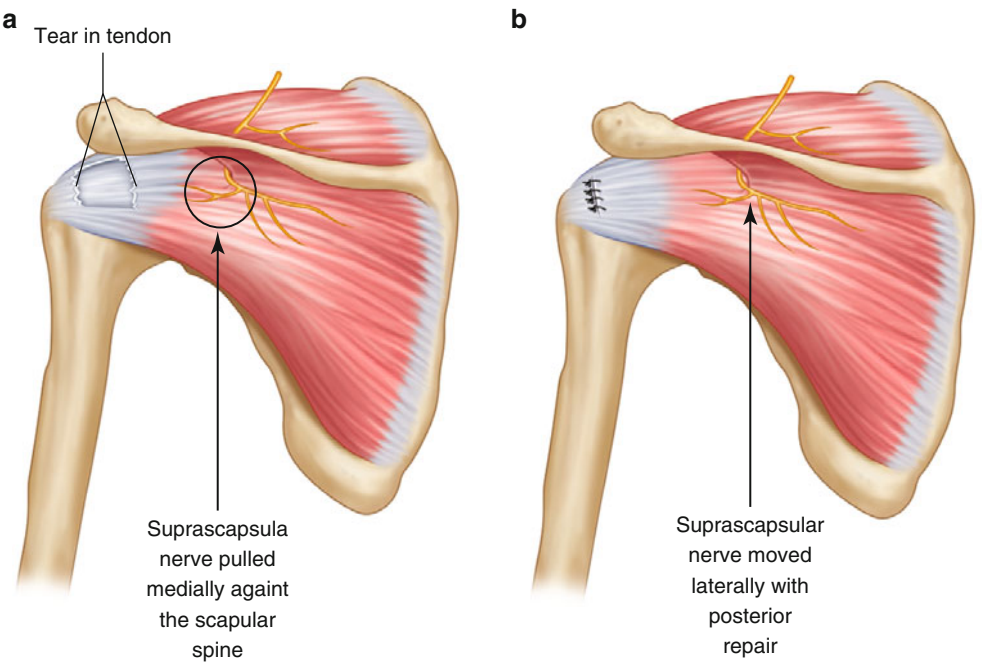

Fig. 39.3 (**a**) Representation of suprascapular nerve traction around the base of the spine of the scapula following medial and inferior retraction of a massive tear involving the supraspinatus and infraspinatus tendons (Modified from Costouros et al. [10]). (**b**) Representation that postulates the mechanism of resolution of suprascapular neuropathy by relieving traction around the base of the scapular spine with infraspinatus tendon repair (Modified from Costouros et al. [10])

Costouros et al. in which reversal of the neuropathy occurred after repair [10, 12] (Fig. 39.3). Albritton et al. and most recently Massimini et al. performed anatomic studies validating the concept of increased medial displacement of the suprascapular nerve in the presence of a retracted supraspinatus tendon tear [44, 45]. Massimini et al. also showed in this dynamic fluoroscopic study that repair of the rotator cuff tear restores the position of the nerve and that release of the transverse scapular ligament allows for 2.5 mm of medial and superior displacement of the suprascapular nerve [45].

Conversely, increased lateral advancement of the rotator cuff during repair may also result in a traction phenomenon of the nerve. Cadaveric studies have shown the maximum lateral advancement of a retracted rotator cuff tear is between 1 and 3 cm and with increased advancement the neurovascular pedicle is placed under increased tension [39, 46]. Although there is dispute about whether or not rotator cuff advancement results in clinical suprascapular neuropathy, Hoellrich et al. did not find any electrodiagnostic findings of suprascapular neuropathy after repairs of massive rotator cuff tears in nine patients with an average advancement of 2.5 cm (range 2.0–3.5 cm), they concluded the tendon could be mobilized and advanced up to 3.5 cm without risk to the nerve [47].

Finally, compressive neuropathy can result from tumors, fractures, prominent screws or implants, and labral cysts. Paralabral cysts probably represent the most common type of compression etiology at the spinoglenoid notch and these are typically treated with repair of the associated labral injury with or without decompression of the cyst. While direct decompression of the cyst is often advocated, simply repairing the labral tear appears to result in resolution of the cyst and neuropathy alone [48]. Removal of prominent screws compressing the suprascapular nerve following a Latarjet procedure has also been described as a successful method of treatment for suprascapular neuropathy [49].

History

A thorough history includes age, hand dominance, occupation, and determination of any previous trauma, repetitive overhead activity, or high-endurance overhead training. A high level of suspicion must be maintained as many of these patients will present with vague or mild symptoms. Patients typically describe an insidious onset of dull, aching pain in the superior, posterior, or posterolateral aspect of the shoulder. Weakness and fatigability with overhead activities is a common complaint, whereas the presence of night pain is variable. Also requisite is a detailed account of previous treatments and their results. For example, suspicion should be elevated when a patient presents with continued pain and external rotation weakness many months after a successful rotator cuff repair. It is difficult to diagnose a suprascapular nerve lesion on history alone, however, as it is often associated with other shoulder pathology [50].

Clinical Examination

All garments should be removed to allow full visualization of the back and shoulder region. Inspection of the shoulder may demonstrate atrophy of the infraspinatus and/or supraspinatus

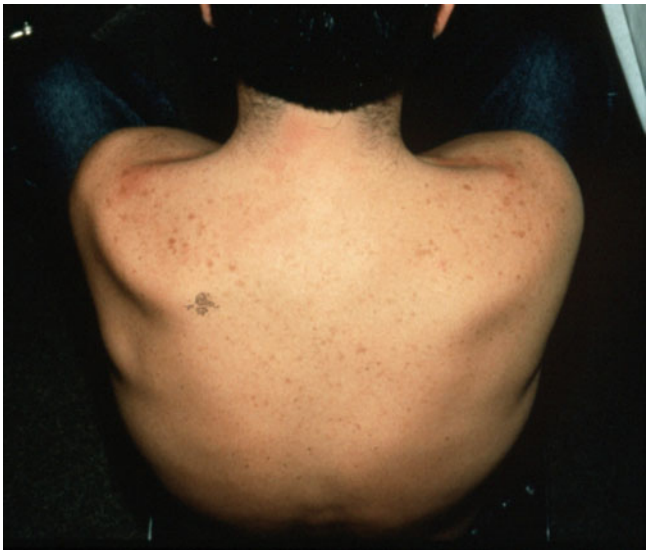

Fig. 39.4 Atrophy of the left supraspinatus and infraspinatus

fossa (Fig. 39.4). Isolated atrophy of the infraspinatus suggests the pathology is present at the spinoglenoid notch, whereas atrophy of both suggests compression proximal to or at the suprascapular notch. Severity of the atrophy may not always correlate with symptoms due to the compensatory stabilization of surrounding musculature. For example, in long-standing cases, the teres minor muscle may compensate for the loss of the infraspinatus muscle and maintain nearly normal strength in external rotation [20]. All regions of the shoulder should be evaluated for tenderness to palpation. Nerve injury at the level of the suprascapular notch may elicit tenderness posterior to the clavicle in the region between the clavicle and the scapular spine. In comparison, spinoglenoid notch compression may elicit tenderness deep and posterior to the acromioclavicular joint that can be accentuated with cross-body shoulder adduction, which causes tightening of the spinoglenoid ligament [35]. A thorough assessment of both passive and active range of motion and individual rotator cuff muscle strength should be completed. Subjectively grading rotator cuff muscle weakness can be difficult, and utilization of a handheld digital measuring device to assess strength can improve accuracy. Injury to the suprascapular nerve may demonstrate weakness with resisted abduction and external rotation of the shoulder, and the sensitivity of this test may be increased by comparing measured values to the normal, contralateral shoulder.

A complete physical examination of the cervical spine should also be performed. Differentiating between lesions proximal and distal to Erb's point is difficult yet essential. Lesions involving the C5, C6, and occasionally C4 nerve roots could demonstrate signs of suprascapular neuropathy as a combination of their fibers constitute the suprascapular nerve. However, if the injury were at the level of these nerve roots, one would expect to see decreased deltoid and biceps strength as well as sensory changes involving the lateral arm, forearm, or thumb. Detection of weakness in muscles other than the supraspinatus and/or infraspinatus and sensory or radicular symptoms in the arm or forearm suggests a nerve root lesion and can help clarify the location of the compressive lesion.

Imaging/Testing

AP and axillary radiographs are indicated for a history of trauma and the new onset of nerve symptoms. These are obtained to assess for fracture, exuberant callus formation, bone tumor, osseous dysplasia, and osseous variants in and around the suprascapular notch. Two additional films can be considered in addition to standard shoulder radiographs; a Stryker notch view (beam is directed 15 to 30 degrees cephalad) may allow for better evaluation of osseous notch variants. Computed tomography scans are excellent for definition of fractures and osseous anatomy and also provide information about the condition/level of fatty infiltration of the rotator cuff musculature. Magnetic resonance imaging provides excellent information about the condition of the rotator cuff as well as additional information regarding other soft tissue structures in the shoulder implicated in suprascapular neuropathy including labral pathology, cystic structures, and other soft tissue masses. Magnetic resonance imaging is particularly helpful in the clinical scenario of complete fatty infiltration of a rotator cuff muscle without evidence of a tendon tear (Fig. 39.5).

The most commonly used modality for diagnosis and confirmation of suprascapular nerve injury is electrodiagnostic studies, including electromyography (EMG) and nerve conduction velocity (NCV) measurement. These studies are currently considered the gold standard in diagnosis of suprascapular neuropathy. Indications for ordering EMG and NCV include atrophy and weakness of the shoulder without evidence of a rotator cuff tear or compressive etiology such as a paralabral cyst. Additional indications include fatty infiltration and inflammation of rotator cuff musculature as visualized on MRI. When denervation of the supraspinatus and infraspinatus occurs, electromyography of these muscles may demonstrate fibrillations and sharp waves. Nerve conduction velocity studies may reveal latency in motor conduction velocity of the suprascapular nerve. Sensory latency measurements are not established as a reliable method of testing. While normative standards have been established for evaluating electrodiagnostic testing, variability does remain and results in inconsistent interpretation between centers and practices [51–54]. The sensitivity and specificity of electrodiagnostic testing is variable and can result in both false-positive and false-negative results, further casting

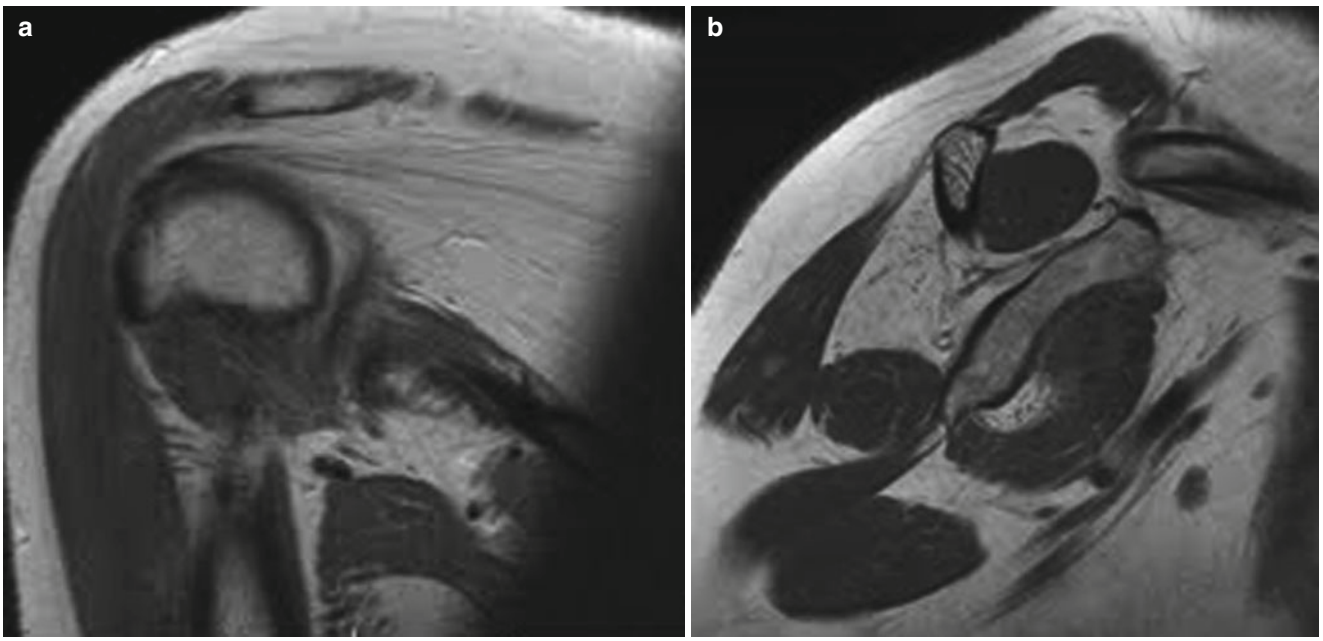

Fig. 39.5 (**a**) Coronal oblique T1 MRI showing fatty infiltrated supraspinatus with intact cuff. (**b**) Sagittal oblique T1 MRI showing same patient

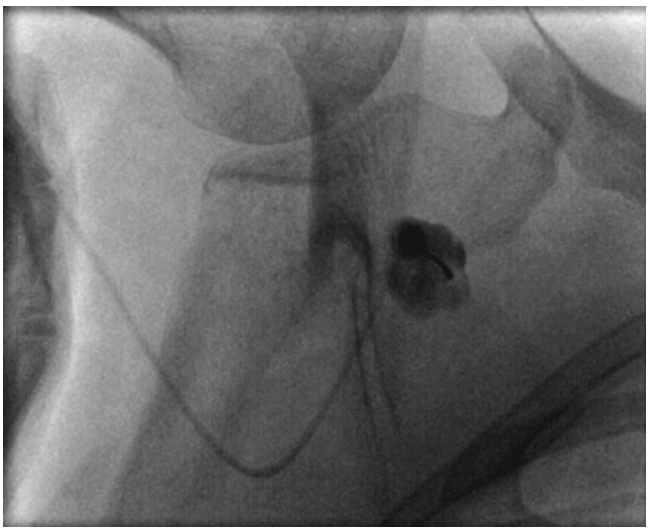

Fig. 39.6 Fluoroscopic-guided SSN injection

doubt on the accuracy of test results [22, 55]. Operator error and dependability remain a problem with these studies, and interpretation can be difficult.

A fluoroscopic-guided injection of local anesthetic into the region of the suprascapular nerve may be useful to evaluate for pain relief in patients for whom the findings of these diagnostic studies are negative or equivocal and continue to have unexplained symptoms. In currently unpublished data, the senior author found that 26/46 patients suspected of having suprascapular neuropathy had a negative EMG study but obtained relief from an injection. Of these 26 patients, 19/20 who underwent arthroscopic suprascapular nerve decompression with release of the transverse scapular ligament reported a good or excellent result. In this series, a fluoroscopic-guided injection of the suprascapular notch was found to be more sensitive than EMG and more predictive of outcome from suprascapular nerve decompression (Fig. 39.6).

Treatment: Indications and Contraindications

Nonoperative Treatment

Historically, the initial treatment for most isolated suprascapular nerve lesions was activity modification, nonsteroidal anti-inflammatory drugs, and physical therapy [55, 56]. Treatment included physical therapy focused on shoulder range of motion as well as strengthening and scapular-stabilizing exercises. Aggravating overhead exercises or activities are avoided in this period. As understanding of the sources of neuropathy increases and reliable imaging diagnosing the presence of cyst or other compressive lesion becomes available, earlier surgical treatment is more frequently advocated.

The results of conservative management are somewhat limited by a small number of studies. Black and Lombardo reported on four cases of neuropathy affecting only the infraspinatus, and all patients had improvement with use of nonoperative modalities over 6 months to 1 year [57]. In another study involving five patients, 3 had improvement with therapy, one failed conservative therapy and required

surgery, and another was lost to follow-up [58]. Drez reported on four patients with isolated suprascapular neuropathy that improved with therapy alone and recommended 6–8 months of nonoperative treatment [59]. Martin et al. reported on 15 patients with isolated suprascapular neuropathy managed nonoperatively with average follow-up of almost 4 years. Five patients had an excellent result, seven had a good result, and three required surgical intervention [55].

Some authors suggest surgical intervention should be the initial step once suprascapular neuropathy is diagnosed in order to prevent further muscle atrophy and degeneration [60, 61]. The majority of authors, however, believe conservative management is justified except in the setting of a space-occupying lesion or persistent pain [55]. Piatt et al. demonstrated 53 % of 19 patients treated conservatively with pain secondary to a spinoglenoid cyst were satisfied as compared to 96 % of 27 patients who were satisfied after surgical treatment [62].

Operative Treatment

The decision to proceed with surgical treatment is individualized. Surgery is generally recommended based on the individual clinical and diagnostic findings, the anatomic location of the lesion, and the cause of the injury. There are no prospective studies with an evaluation of operative versus nonoperative treatment. The decision when to perform a suprascapular nerve decompression is evolving. Isolated suprascapular neuropathy without an obvious anatomic reason (retracted rotator cuff tear, spinoglenoid cyst) is less common [5, 8, 63]. If an isolated suprascapular neuropathy has been diagnosed and has failed a course of conservative management, surgical intervention with suprascapular nerve decompression is indicated.

Controversy exists as to the role of suprascapular nerve release performed at the time of rotator cuff repair and cyst decompression with labral repair. There are no prospective studies evaluating the outcomes of rotator cuff repairs with and without suprascapular nerve release. Lafosse et al. presented their indications for suprascapular nerve release recently, and this included patients who presented with weakness of the infraspinatus, with or without wasting of the supraspinatus, pain, or positive electromyographic findings. They also recommended release if a thickened or ossified ligament was encountered during arthroscopic rotator cuff repair [61].

Decision-Making Algorithm

See Fig. 39.7

Clinical Case/Example

HPI: 45-year-old RHD male plumber presented for initial evaluation for continuing complaints of right shoulder pain. He sustained a full-thickness supraspinatus tear after a skiing accident and had subsequent arthroscopic repair. His initial recovery was excellent both in ROM and strength but experienced mild continued pain. The deep posterior/superior shoulder pain persisted, and 6 months later despite physical therapy, the weakness and loss of range of motion recurred. Clinical examination revealed significant atrophy, decreased range of motion, and marked weakness. Repeat MRI revealed a healed rotator cuff tendon and the absence of any other identifiable pathology. Electrodiagnostic testing was normal. However, given his isolated weakness with abduction and external rotation, and subjective pain over the superior/posterior aspect of his shoulder, suspicion for suprascapular neuropathy remained high. A fluoroscopically guided suprascapular notch injection consisting of 40 mg of triamcinolone and 5 cc of 1 % bupivacaine was performed and provided immediate, significant relief of pain. Given his positive response to the injection, arthroscopic suprascapular nerve decompression was performed. The rotator cuff was found to be well healed at the time of surgery. The patient subsequently had complete pain relief, full range of motion, and improved strength at 2 months follow-up. The atrophy persisted but the patient was able to return to work.

Arthroscopic Treatment: Surgical Technique (Video 39.1) (Box 39.1)

Box 39.1: Tips and Tricks

- When performing the initial diagnostic arthroscopy, place the anterior portal in a more superior position to allow for combined use in both the glenohumeral joint and suprascapular nerve release.
- If the portal is too low, it can be difficult to place the electrocautery device “over the top” of the coracoid base and into the supraspinatus fossa.
- It may be better to perform the nerve exploration and decompression before the glenohumeral joint surgery because soft tissue swelling makes the suprascapular nerve decompression portion of the operation more difficult [64].
- When uncertain about your anatomic location, return laterally to a known anatomic structure, such as the coracoacromial ligament or the anterolateral acromion, and then proceed again medially.

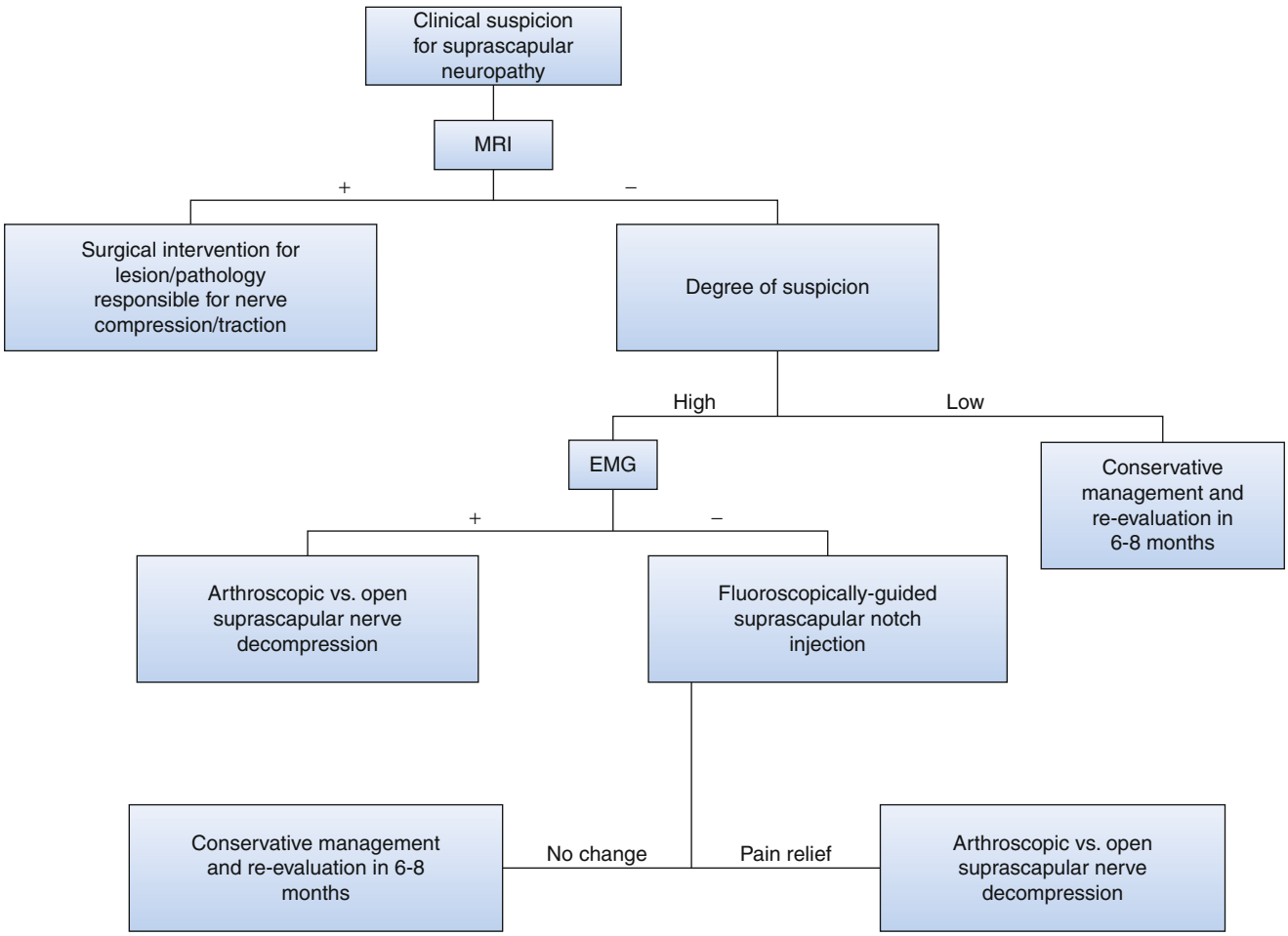

Fig. 39.7 Decision-making algorithm

Patient Positioning/Portals

The patient is typically placed in the beach-chair position. Care must be taken while draping to provide enough room over the superior aspect of the shoulder for portal placement. The arm can be placed in a mechanical arm holder. Depending on the clinical scenario, standard portals for diagnosis and treatment of related shoulder pathology are performed.

Diagnostic Arthroscopy: Understand and Recognizing the Pathology

The decision to decompress the nerve is determined preoperatively. Any pathological anomalies, such as an ossified transverse scapular ligament, should be assessed and recognized prior to the procedure. At the time of surgery, the suprascapular nerve may have a normal appearance or may show obvious signs of compression. Regardless of the appearance, the nerve should be decompressed if clinically indicated.

Step-by-Step Procedure: Decompression at the Suprascapular Notch

- Create standard posterior and anterior portals, perform diagnostic arthroscopy, and repair intra-articular pathology as indicated.
- Create lateral portal and perform bursectomy/acromioplasty as indicated.
- While in the subacromial space, create an accessory anterolateral portal. Place the arthroscope in the lateral portal and the electrocautery device/shaver in the accessory anterolateral portal (Fig. 39.8).
- Complete the subacromial bursectomy and release soft tissue adhesions medially.
- Follow the coracoacromial ligament inferomedially toward its attachment on the coracoid.
- Identify the tip of the coracoid and follow the coracoid medially while keeping the supraspinatus muscle visible and adjacent.
- Debridement should stop once the coracoclavicular ligaments are identified. The suprascapular nerve is protected

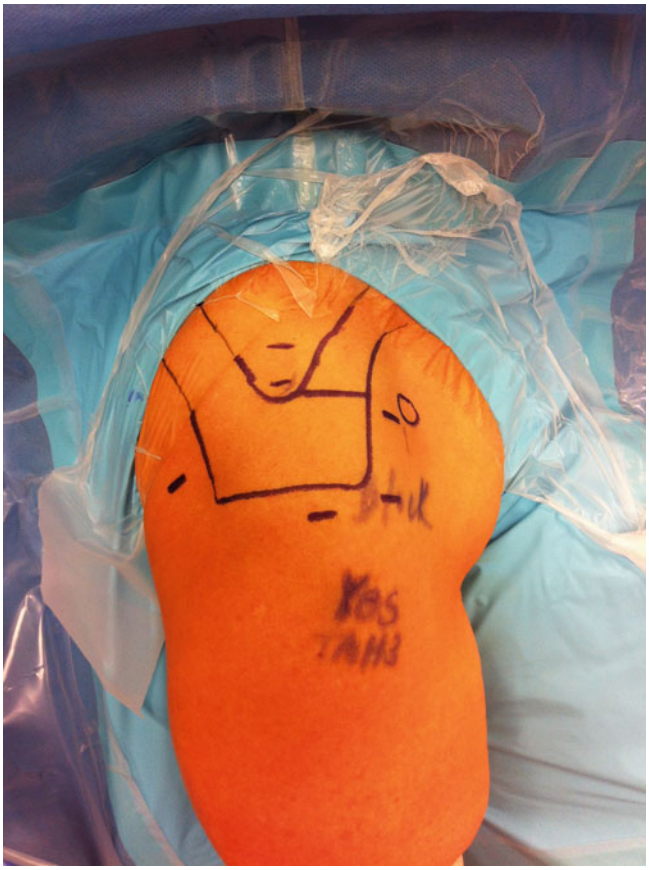

Fig. 39.8 Required arthroscopy portals

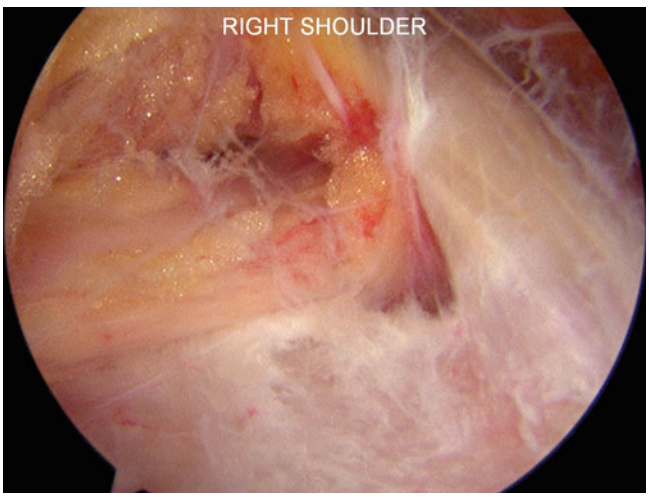

Fig. 39.9 Identification of transverse scapular ligament

by the transverse scapular ligament but the artery which overlies the ligament is not (Fig. 39.9).

- A portal approximately 2.5 cm medial to the triangular soft region formed by the confluence of the clavicle and scapular spine is created. Needle localization can be performed but care must be taken not to perforate the suprascapular artery and create bleeding.

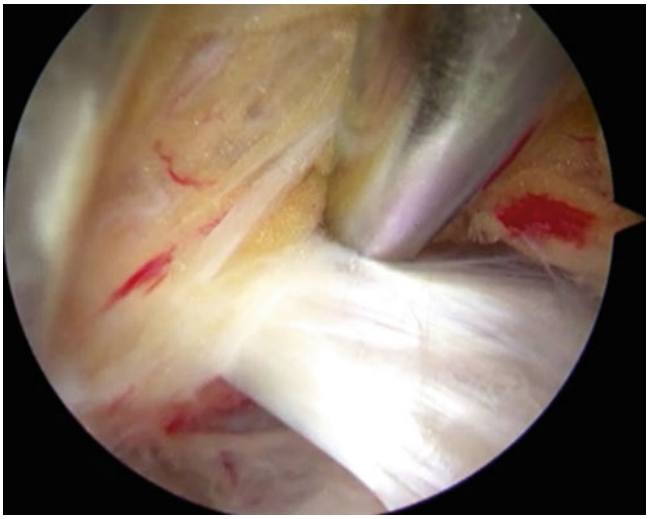

Fig. 39.10 Sweeping suprascapular nerve and artery medially

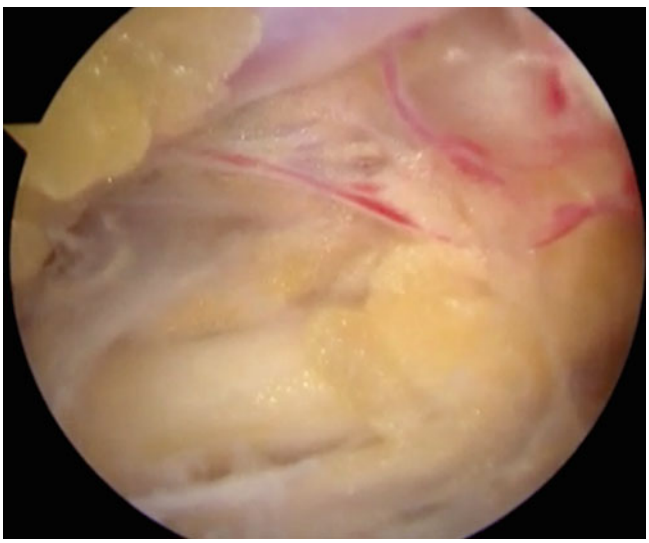

Fig. 39.11 Following release of the transverse scapular ligament

- A blunt trocar is then used to sweep medially to dissect the structures of the suprascapular notch. The lateral aspect of the transverse scapular ligament is usually the first identifiable structure to come into view and is a medial continuity of the conoid ligament. The nerve and artery may or may not be seen well at this point.
- The artery is not discretely visualized but its pulsations are typically seen. The artery is swept medially, and the full extent of the transverse scapular ligament is visualized. The nerve is visualized below the ligament and is swept medially with the artery using the blunt trocar (Fig. 39.10).
- A second portal is made 1 cm lateral to the previous portal, and while protecting the artery and nerve with the blunt trochar, the ligament is divided near its lateral insertion with arthroscopic scissors (Fig. 39.11).

- A specialized transverse scapular ligament cutter can also be used to perform the transverse scapular ligament release through a single incision without the need for placement of a separate trocar for retracting.
- In cases of an ossified ligament, a Kerrison rongeur and possibly a small burr may be needed to perform the decompression.
- Finally probe the nerve and verify that the entire ligament has been released.

Step-by-Step Procedure: Decompression at the Spinoglenoid Notch

- Standard anterior and posterior portals are created as well as a trans-rotator cuff portal through the muscular portion of the supraspinatus.
- The arthroscope is placed laterally through the trans-cuff portal.
- Through the posterior portal, perform a capsulotomy by releasing the posterosuperior capsule at the periphery of the labrum until the fibers of the supraspinatus muscle are identified.
- A retractor is then placed through the anterior portal and the supraspinatus muscle is elevated superiorly.
- The suprascapular nerve is identified 2.5–3 cm medial to the superior aspect of the glenoid at the base of the supraspinatus fossa [39].
- Trace the nerve distally as it passes deep to the supraspinatus muscle into the spinoglenoid notch.
- Identify the spinoglenoid ligament and release with arthroscopic scissors.
- Perform neurolysis with the use of handheld basket punches and arthroscopic probes.

Postoperative Care

Excluding any other surgical intervention other than suprascapular nerve decompression, the patient should be placed in a sling for comfort. After 2–3 days, the patient is encouraged to wean the sling and begin early active motion. Physical therapy without restrictions can be initiated as soon as the patient is comfortable.

Literature Review

Early results of the arthroscopic approach in ten patients with suprascapular neuropathy secondary to compression at the suprascapular notch have been reported [63]. At a mean of 6 months postoperatively, 90 % graded their outcome as excellent with complete relief of pain, 7 of 10 had complete normalization of electromyographic findings, and two others showed partial recovery of the nerve. The senior author recently reported on 27 patients who underwent arthroscopic suprascapular nerve decompression at the suprascapular and/or spinoglenoid notch. Preoperative positive electromyography and nerve conduction EMG/NCV studies were positive in 89 %, documenting suprascapular nerve pathology. All patients had either a computed tomography arthrogram or magnetic resonance imaging study to document rotator cuff integrity and rule out associated pathology. Average follow-up was 22.5 months (range, 3–44).

Statistically significant improvements were seen in Visual Analogue Scale, Subjective Shoulder Value, and American Shoulder and Elbow Society self-assessment scores [65].

Addressing the suprascapular neuropathy as a result of a massive retracted rotator cuff tear continues to be controversial. Mallon et al. reported that two of four patients with suprascapular neuropathy and a massive retracted rotator cuff tear showed reinnervation potentials after partial arthroscopic rotator cuff repair [12]. The senior author reported that six of six patients with electrodiagnostically confirmed suprascapular neuropathy demonstrated partial or full resolution of the suprascapular neuropathy on postoperative electromyography and nerve conduction velocity studies after partial or complete rotator cuff repair [10]. These two reports suggest suprascapular neuropathy secondary to traction associated with a rotator cuff tear may partially or completely resolve with repair of the rotator cuff alone.

Surgical intervention of a spinoglenoid notch cyst may encompass either decompression of the cyst, a labral repair, or both. An open decompression requires a posterior approach, but doing so limits access to the labrum in order to address other intra-articular pathological conditions [5]. The arthroscopic approach allows for cyst decompression while providing better visualization of labral lesions and repair. Some authors have reported lower recurrence rates using arthroscopic methods [62]. Fehrman et al. reported that five of six individuals with a spinoglenoid notch cyst had complete pain relief following a combined open and arthroscopic approach [24]. Chen et al. reported on three patients who had undergone arthroscopic decompression and superior labral anterior-posterior (SLAP) tear repair [66]. Magnetic resonance imaging confirmed removal of the cyst in all three patients and recovery of the suprascapular nerve was proved using electromyography (EMG). Westerheide et al. noted that in 14 patients treated only with arthroscopic cyst decompression, all patients had improved function and decreased pain, with no instances of recurrence [27]. Antoniou et al. examined 53 patients with suprascapular neuropathy and found that those who underwent surgical intervention fared better than those treated conservatively; however, these are a combination of open and arthroscopic cases [5]. Lichtenberg et al. reported 100 % pain relief and improvement in strength for eight patients with suprascapular neuropathy treated with arthroscopic decompression of the cyst and labral repair [67].

Summary

Although suprascapular neuropathy was historically considered a diagnosis of exclusion, the incidence of this condition will undoubtedly increase as our ability to recognize and accurately make the diagnosis improves. The condition may be more common than once believed as more recent reports are describing suprascapular neuropathy as a cause of substantial pain and weakness in patients with and without concomitant shoulder pathology. Insult to the suprascapular nerve can result from repetitive overhead activities, a space-occupying lesion, compression from the respective transverse ligament, or as more recent studies have demonstrated, a traction injury occurring with retraction of a large rotator cuff tear. Atrophy of the infraspinatus and/or supraspinatus rotator cuff muscles with resultant weakness in forward flexion and/or external rotation of the shoulder on physical examination may be demonstrated. Pain may be localized to the superior or posterolateral aspect of the shoulder. Magnetic resonance imaging is the preferred modality with which to assess atrophy of the rotator cuff muscles and potential lesions causing suprascapular nerve compression. Electromyography and nerve conduction velocity studies can be used for confirmation of the diagnosis of suprascapular neuropathy, but a fluoroscopic-guided suprascapular notch injection may become the diagnostic study of choice in the future. Initial management is usually conservative, consisting of activity modification, physical therapy, and nonsteroidal anti-inflammatory drugs. Surgical intervention is considered for patients with a known compressive lesion or for symptoms refractory to conservative measures. Decompression of the suprascapular nerve may be accomplished through an open or arthroscopic surgical approach. Arthroscopic release, however, requires advanced technical skill and detailed knowledge of the anatomy, but it is less invasive and potentially more effective in treating suprascapular neuropathy.

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Biologic Augmentation in Rotator Cuff Repair

40

Brian D. Dierckman, Randy R. Clark, Joseph P. Burns, and Stephen J. Snyder

Introduction

Full-thickness rotator cuff tears are common in the aging population, with an estimated incidence between 28 and 40 % in patients over age 60 [1, 2]. While some rotator cuff tears are asymptomatic, many will result in pain and dysfunction [3]. Surgery is often recommended for those tears causing disabling symptoms that fail reasonable conservative measures. A significant percentage of tears fail to heal following repair regardless of surgical technique [3–13]. Several factors that contribute to repair failure include patient age, tear chronicity, tear size, fatty infiltration, and muscle atrophy [14–26].

High failure rates have been reported specifically following treatment of large and massive cuff tears [14–22, 27, 28]. Revision repair of failed large and massive tears is often not effective. Alternative surgical options include tendon transfers and reverse total shoulder arthroplasty. These procedures are best suited for the lower demand and/or elderly patient with concomitant arthritis in addition to their massive rotator cuff tear. In the physiologically younger, active, and thus higher-demand patient, these procedures would require restrictions that would limit physically vigorous employment and many recreational activities. Additionally, a younger patient's life expectancy may exceed the wear characteristics of the implant materials. Anatomic biologic repair with expectant restoration of more normal joint biomechanics is the preferred option when possible.

The high failure rate seen in many patients following surgical cuff repair has impelled surgeons to develop supplemental techniques involving biologic scaffolds to enhance the repair constructs. These biologic grafts are intended to provide an immediate repair scaffold that adds initial strength to the repair while facilitating cellular repopulation and tissue regeneration through cell recruitment and adherence [29, 30].

Another important consideration is patients undergoing revision rotator cuff repair. These patients are more likely to have complex tears with tissue loss and muscle atrophy. Efforts at revision repair of these challenging tears often fail. Biologic tissue graft for interposition is a useful option in this clinical setting, as it may provide a scaffold for bridging and healing the tissue defect.

Neviaser first reported the use of an interposition allograft for rotator cuff repair in 1978, using freeze-dried rotator cuff tendon [31]. Since then, a variety of materials have been developed and tested, including synthetic grafts, xenografts, and allografts. Factors to consider when choosing a rotator cuff augment include origin, processing, physical properties, results of animal testing, and clinical outcomes [32]. Many authors have studied and described the biomechanics, biochemistry, and cellular characteristics of the currently commercially available grafts (Table 40.1).

Rotator cuff grafts are ideally suited for patients who have two or three tendon tears greater than 3 cm prior to repair. Chronic, retracted tears are more likely to require either patch augmentation or interposition than acute tears, since they are often not easily reduced to the footprint with minimal tension. If a partial repair with a residual defect greater than 1 cm can be performed, we recommend an interposition reconstruction (inlay). Augmentation (onlay) is an option for

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Table 40.1 Summary of different commercially available scaffolds for rotator cuff augmentation

Product name	ECM type	ECM source	Marketed by
ArthroFlex	Dermis	Human	Arthrex
BioBlanket	Dermis	Bovine	Kensey Nash
Conexa	Dermis	Porcine	Tornier
GraftJacket	Dermis	Human	Wright Medical
TissueMend	Dermis	Bovine	Stryker Orthopaedics
Zimmer Collagen Repair	Dermis	Porcine	Zimmer
CuffPatch	SIS	Porcine	Biomet
Restore	SIS	Porcine	DePuy Orthopaedics
OrthoADAPT	Equine pericardium	Equine	Synovis Surgical Innovations
SportMesh Soft Tissue Reinforcement	Poly(urethane urea)	Synthetic	Biomet Sports Medicine
X-Repair	Poly-L-lactide	Synthetic	Synthasome
Biomerix RCT Patch	Polycarbonate poly(urethane urea)	Synthetic	Biomerix

complete repairs as well as for repairs with a residual defect less than 1 cm.

Some authors have reported on their early experience combining biologic factors, such as platelet-rich plasma and mesenchymal stem cells, with rotator cuff grafts in an attempt to maximize tissue regeneration [33]. Further research is needed on this topic, but it is certainly an appealing option, as these collagen-based scaffolds can serve as carriers for a multitude of biologic factors, especially when the native biologic environment is compromised in chronic, dysvascular cuff tears.

Synthetic Scaffolds

Synthetic scaffolds are comprised of nonbiologic materials such as polyurethane urea and polylactic acid [34, 35] (Table 40.1). These materials are constructed from biologically inert materials that are machined into a rotator cuff patch. There is a paucity of clinical data and outcomes with regard to the application of synthetic scaffolds, although several series report promising data [34, 35]. Recently, Encalada-Diaz and colleagues reported on their experience with a synthetic scaffold in 10 patients. Nine patients had an intact repair and one patient had a re-tear on MRI at 1 year. Their patients had significant overall improvements in UCLA, ASES, and SST scores, and there were no adverse events [36].

In the authors' opinion, the use of synthetic scaffolds holds much promise. However, each material is unique and must be independently studied. The fears with all current synthetic materials include possible limited biologic ingrowth, the potential for acute and chronic inflammatory reactions, and foreign body rejection or encapsulation [37, 38]. Further investigations are needed to better define the role of synthetic scaffolds and their clinical application, but we believe that this is an attractive opportunity for future development especially if and when they can be combined with biologically active materials.

Xenografts

Numerous xenograft materials, including porcine and bovine dermis, porcine small intestinal submucosa (SIS), and equine pericardium, are commercially available for soft tissue augmentation. A review of the literature indicates mixed results with xenograft patches, with some early success [39], while other studies detail significant concerns that have discouraged widespread clinical acceptance [40, 41].

Iannotti et al. published a randomized trial comparing open repair of two-tendon cuff tears with and without a porcine SIS graft [42]. Nine of the 15 repairs healed in the control group, while only 4 of the 15 healed in the augment group; functional scores were significantly better in the control group as well. The authors recommended against the use of porcine SIS in this setting.

Walton et al. also recommended against using porcine SIS after four of their ten patients experienced failure [43]. They also noted a mass of collagenous debris in one of the cases that underwent a further surgical procedure. This was presumed to be the liquefying collagen implant as there were no other residual signs of the implant. Histological examination revealed necroinflammatory exudate admixed with fibrinous material and chronic synovitis. There has been another recent and similar report of porcine SIS implants in rotator cuff repairs causing nonspecific inflammatory reactions with early failure of the repair [44]. Traditional xenografts from porcine sources are known to contain the Gal epitope, which has been implicated in non-human graft rejection [45].

Based upon the currently available evidence, we do not recommend use of xenografts for rotator cuff repair.

Human Freeze-Dried Allograft Rotator Cuff Tendon

The first published reports using human allograft material for rotator cuff reconstruction evaluated freeze-dried cuff tendon. Neviaser reported early success with the use of human rotator cuff allograft but long-term follow-up was not reported [31]. Other authors ultimately recommended against this application [46]. We have no experience with this graft material, and since there is little reverent published data on freeze-dried rotator cuff allografts, we cannot currently recommend its use.

Acellular Human Dermal Allografts (AHDAs)

There is now significant interest in AHDAs, with numerous studies documenting the enhanced biocompatibility and capacity for stimulating tissue regeneration, especially when compared to synthetic and xenograft scaffolds [37, 38, 40, 41]. The proprietary graft processing that removes cellular genetic material renders the AHDA tissue immunogenically quiet. The collagen fibers, microvascular tubules, and proteoglycan extracellular matrix are not damaged during processing, thus providing a strong, dense nonreactive scaffold into which new host tissue can regenerate.

Several studies have evaluated the biomechanical characteristics of AHDAs. Barber et al. evaluated the load to failure and failure pattern of several commercially available rotator cuff scaffold products. The authors found that AHDA materials have superior load to failure and suture pullout strengths compared to other porcine/bovine skin and porcine SIS products [47]. Barber et al. also evaluated biomechanical properties of soft tissue augmentation devices, and they determined AHDAs were stronger and stiffer than synthetic and equine pericardium patches and AHDAs also demonstrated superior suture retention [48].

Barber et al. compared cadaveric rotator cuff repairs augmented with AHDAs to traditional repairs. They found that AHDAs significantly increased the strength of the repair construct in a cadaveric biomechanical model [49]. Other biomechanical studies support the concept that patch augmentation improves the time-zero strength of the repair construct [50, 51].

The results of patch augmentations performed in animal models have been promising. Ide et al. showed that in a rat model, reconstruction of a large rotator cuff tear with an acellular dermal matrix graft had a higher modified tendon maturation score and required greater ultimate force to failure as compared to a control specimen. At 12 weeks, the graft had fully incorporated and demonstrated histology resembling the control specimens [52]. Adams et al. reported on their experience with AHDA in a canine model [53]. Within 6 weeks, there was evidence of native cellular infiltration and neotendon development, and by 12 weeks, the strength of the graft repair was equivalent to the autogenous repair (control). The results of several other animal models support the use of patch augmentations of cuff repairs [54–57].

Despite the favorable literature in support of scaffolds in animals, some authors remain skeptical. Derwin et al. compared scaffolds for rotator cuff tendon repair in a canine model. The AHDAs had superior in vitro biomechanical properties compared to the other patches tested, but they were still found to be inferior to canine infraspinatus tendon. Their ultimate recommendation was for limited use of these patches for augmentation of tendon repair [58]. Chaudhury et al. evaluated the key mechanical properties of rotator cuff patches and found significant variability and inferiority compared to the human rotator cuff tendon [59].

Surgical Technique

Both open and arthroscopic techniques have been reported for rotator cuff augmentation and interposition. Because of the potential patient-related advantages of minimally invasive techniques along with improved visualization, we prefer to perform rotator cuff augmentation/interposition using an arthroscopic technique somewhat similar to that described by Leuzinger et al. [60], Seldes and Abramchayev [61], but Labbe [62] and modified by the senior authors (SJS and JPB). It is important to note that no clear differences in outcomes between open and arthroscopic techniques have been reported to date.

Disclaimer – *AHDAs are FDA-approved for augmentation of rotator cuff repairs with a residual defect of less than 1 cm. AHDAs are currently not FDA-approved for use as an interposition graft.*

Surgical Technique: Augmentation

The following technique summarizes our experience with patch augmentation for large and massive rotator cuff tears. The basic equipment necessary is listed in Table 40.2.

Positioning and Preparation

We perform arthroscopic rotator cuff surgery in the lateral decubitus position. Ten pounds of balanced suspension in 70° of shoulder abduction is used for glenohumeral work, 15 lb in 15° of abduction is used for standard bursal work, and 10 lb in approximately 45° of abduction is used as a "mid-position" for accessing the lateral aspect of the greater tuberosity from the bursal space.

Table 40.2 List of essential equipment for allograft patch AHDA augmentation/interposition of rotator cuff repair

Materials and preparation
Acellular human dermal allograft (>1.8 mm thick)
3 cannulae: posterior, anterior, anterolateral (8.5 mm diameter), plus a posterolateral viewing portal
Knotted measuring suture: knots at 1 cm increments
6–7 No.2 sutures
2–3 push-in style suture anchors (lateral fixation)
Suture hook system for shuttling

A complete arthroscopic glenohumeral evaluation is performed, viewing from both the posterior and anterior portals. Releases of the rotator cuff are initially performed from within the glenohumeral joint space.

In the bursal space, the arm is adducted and a full evaluation of the anatomy is performed. An anterolateral working cannula and a posterolateral viewing portal are established along with standard anterior and posterior portals. Bursal tissue is debrided to maximize visualization. Subacromial decompression is performed as needed. Further bursal-sided release of the rotator cuff tendon is performed at this time.

Rotator Cuff Repair

An arthroscopic rotator cuff repair is performed using customary arthroscopic techniques, including tissue releases and margin convergence sutures when indicated. Most tears can be completely repaired back to bone. Augmentation can be performed in the setting of a small residual gap (<1 cm) after rotator cuff repair, in the setting of a previously failed repair or in a situation where the rotator cuff tendon quality is poor and attritional. We prefer to use the "SCOI Row" technique: a *medially based*, *single-row* of *triple-loaded* suture anchors, combined with *bone marrow venting* of the lateral tuberosity, allowing egress of healing elements from the bone marrow. We prefer this technique for its reproducibility, strength [63], high healing rate, minimal tissue tension, and surgical time/cost benefits.

Graft Measurement and Preparation

Prior to repair, the tendon is debrided and tear size is measured in both anterior-to-posterior and medial-to-lateral dimensions. We use a knotted suture as a measuring device, which is made by placing single knots in a No. 1 suture in 1 cm increments (Fig. 40.1).

On the back table, the allograft patch is hydrated and cut to size. The graft is intentionally oversized by 3 mm per side to allow for placement of the short-tailed interference knot (STIK) suture. A STIK suture consists of a mulberry-type knot tied on the end of a No. 2 suture, which is subsequently passed through the graft. The STIK permits graft manipulation without suture pullout. Depending on the size of the patch augment, the authors recommend three to five STIK sutures,

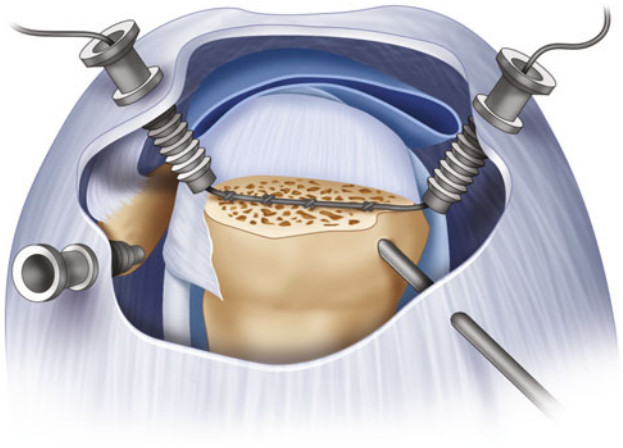

Fig. 40.1 Illustration depicting proper portal placement as well as a medially based, triple-loaded anchor rotator cuff repair with bone marrow vents in the lateral tuberosity

which are placed circumferentially around the anterior, medial, and posterior graft 3 mm from the graft edge. A midline mark is placed on the lateral aspect of the graft. Two No. 2 sutures are placed laterally, equidistant from midline. In our experience, the medial-to-lateral graft width is usually 3–3.5 cm to provide adequate tuberosity and tendon coverage.

Suture Passage

We recommend use of an 8.5 mm anterolateral cannula for ease of graft passage. The arthroscope is maintained in a posterolateral viewing portal throughout the procedure.

Initially, a suture hook is inserted through the posterior cannula and used to penetrate the most posterior and lateral cuff tissue. A shuttle suture is retrieved out the anterolateral cannula with a grasper (Fig. 40.2). The free end of the corresponding STIK suture is then shuttled through the cuff tendon and back out the posterior cannula.

The shuttling technique is repeated, progressing medially and then anteriorly from the posterolateral edge of the graft/cuff tissue, shuttling all the STIK sutures. *It is imperative that the sutures are not tangled during these steps*. This is accomplished by passing each suture *anterior* to the prior one. *The paths of each subsequent suture hook*, *shuttling suture*, *grasper*, *and STIK suture must all be kept anterior to the previously passed sutures*. In a careful and organized fashion, we can thereby place any number of sutures without entanglement. The anterior STIK sutures are passed with the suture hook in the anterior cannula.

Graft Insertion

After passing the STIK sutures through the tissue, the graft is ready for insertion through the anterolateral cannula (Fig. 40.3). The slack is pulled out of all the sutures, thereby

docking the graft at the aperture of the anterolateral cannula. The graft is rolled onto itself to facilitate graft passage through the cannula. Next, a "push-pull" technique is used; as the graft is pushed down the cannula with a grasper, the STIK suture ends are pulled from their respective posterior and anterior cannulae (Figs. 40.4 and 40.5). Once the graft enters the subacromial space, each suture end is sequentially pulled to unfold the graft and cover the repair site. Each STIK can be retrieved and sequentially tied, stabilizing the graft anteriorly, medially, and posteriorly. The authors prefer to tie the most anterior sutures first and progress posteriorly.

Lateral Fixation

Two push-in suture anchors are used to stabilize the lateral edge of the graft over the lateral greater tuberosity. To access the lateral tuberosity, the arm is abducted. The midline mark on the graft is a helpful reference to maintain orientation during anchor placement. A pilot hole is created and the anterolateral suture limbs are loaded into the push-in anchor outside the anterolateral cannula. The loaded push-in suture anchor is placed and the anterolateral graft is tensioned and secured to the tuberosity. Using the same technique and with internal rotation of the arm, the posterolateral anchor is placed, thereby securing the lateral edge of the graft (Fig. 40.6).

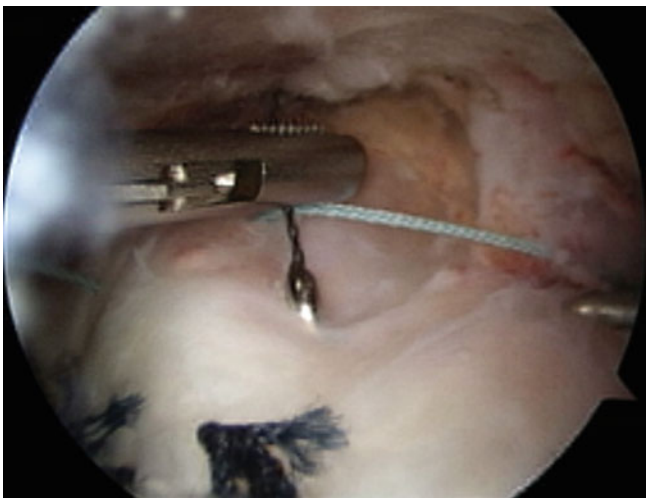

Fig. 40.2 The suture hook and arthroscopic grasper are used to shuttle a STIK suture through the repaired rotator cuff tendon, medial to the bone-tendon interface. The arthroscope is viewing from the posterolateral portal

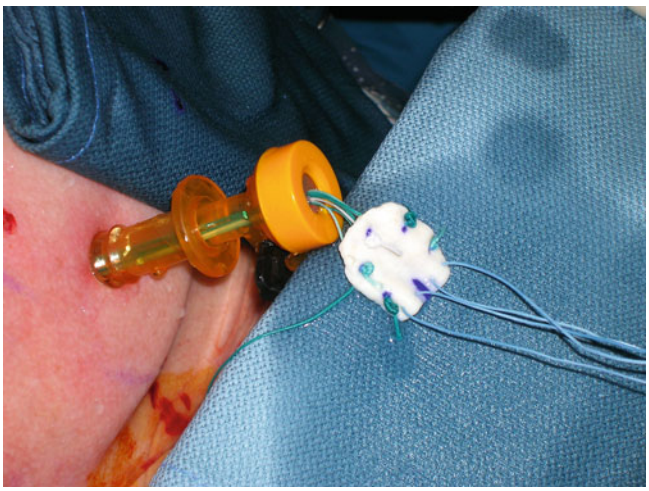

Fig. 40.4 Appearance of the graft as sutures are passed out the anterolateral cannula and prior to delivery of the graft into the cannula and joint

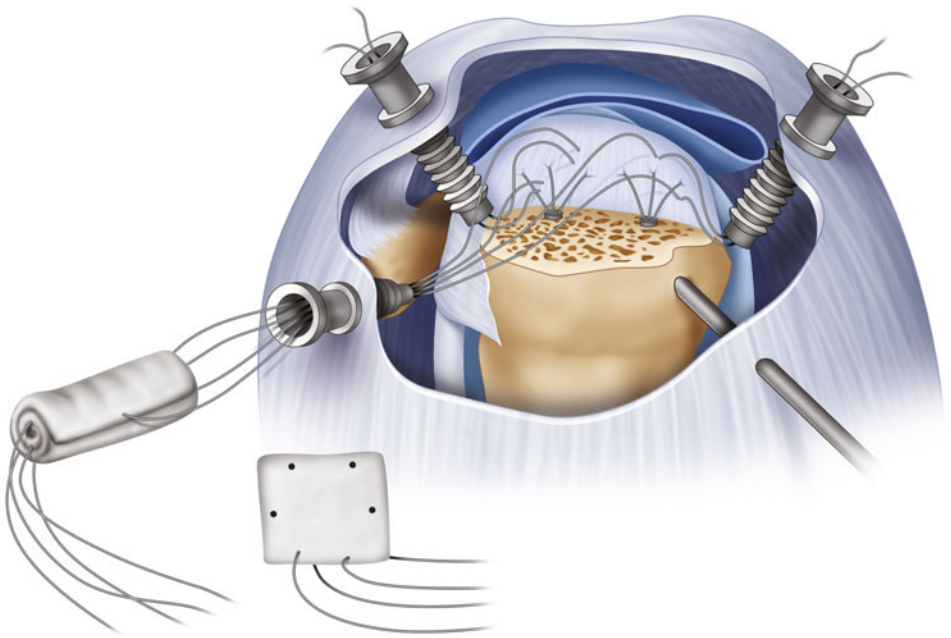

Fig. 40.3 Illustration demonstrating completed passage of all STIK sutures through the rotator cuff. The graft has been rolled and is docked at the aperture of the anterolateral cannula for introduction into the joint. Notice the laterally based sutures remain outside of the anterolateral cannula after passing the graft for later push-in anchor fixation

Fig. 40.5 Appearance of the graft as it leaves the cannula and enters the joint as viewed from the posterolateral portal

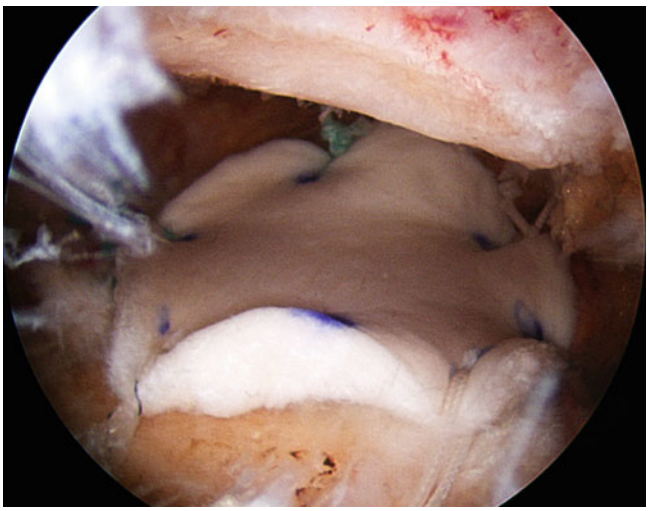

Fig. 40.6 Final appearance of the patch graft after the STIKs have been retrieved and tied as viewed from the anterolateral portal

Rehabilitation

We have found that patients respond well to a postoperative rehabilitation protocol similar to that used for massive rotator cuff repairs. In our experience the therapy program does not differ between patients who undergo AHDA augmentation or interposition. A sling with an abduction pillow is worn for 6 weeks. Initial rehabilitation includes gentle pendulums, as well as elbow, wrist, and hand motion exercises, which are performed three times daily. Formal physical therapy is begun after the second postoperative evaluation, around 6 weeks after surgery. Formal therapy focuses on passive motion with progression to active motion as tolerated. Strengthening is allowed once the patient has obtained full painless active elevation. This usually occurs between the third and fourth postoperative months.

Surgical Technique: Interposition

The positioning, preparation, and equipment are the same for cuff augmentation and interposition.

Debridement and Initial Repair

For most large and massive tears, we begin by performing a thorough superior capsule and cuff release and bursectomy. We also attempt to repair as much of the native posterior cuff and subscapularis as possible to create stable margins for later graft attachment. We prefer to use a graft large enough to fill the residual defect as opposed to over-tensioning of the partial repair. The rotator cuff footprint is debrided of soft tissue, and bone marrow vents are created in the tuberosity lateral to the anchor insertion sites.

A triple-loaded suture anchor is inserted into the posterior aspect of the footprint, just lateral to the articular margin and just anterior to the posterior edge of the remaining native cuff tendon. The most posterior and medial suture from the anchor is passed through the posterior cuff at the level of the anchor using the shuttle technique. This suture is tied using a sliding, locking knot; this serves as a partial repair and establishes the posterior edge of the frame for graft attachment.

A second triple-loaded anchor is then inserted into the anterior aspect of the footprint, just lateral to the articular margin and posterior to the biceps tendon (or within the bicipital groove if tendon is absent). As most chronic, massive tears have very poor quality anterior cuff/interval tissue, we prefer to perform a biceps tenodesis when possible. The biceps tendon enhances the quality of anterior tissue for graft attachment and support. Using the suture shuttle technique, the most anterior and medial suture from this anchor is passed through the interval tissue and biceps tendon multiple times and tied with a non-sliding knot. This establishes the anterior frame for graft attachment.

Graft Measurement and Preparation

Using the knotted suture technique as described earlier (Fig. 40.1), the anterior-posterior and medial-lateral cuff tear dimensions are measured on all four sides.

On the back table, after the allograft has been properly prepared, it is cut to size. The graft is intentionally oversized by 3 mm per side to allow for placement of the STIK suture and provide overlap between the graft and native tissue. The center of the medial and lateral edges of the graft is marked with vertical lines, and smaller dots are made for the location of each STIK suture (Fig. 40.7). STIK sutures are placed 3 mm from the graft edge at 5–7 mm intervals circumferentially around the anterior, medial, and posterior graft. We prefer to use alternating dark and light green STIK suture along the anterior and posterior edges of the graft and alternating plain white and white sutures with

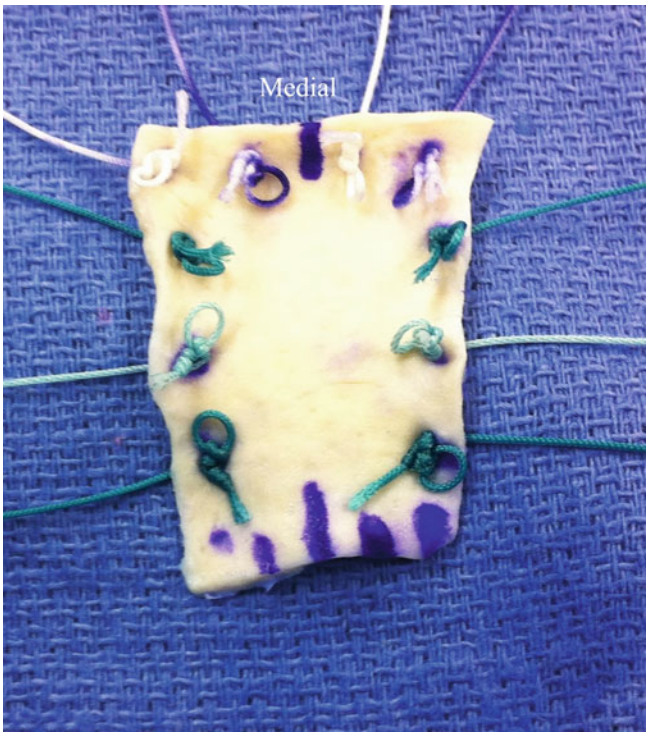

Fig. 40.7 Appearance of the graft after STIK sutures have been passed through graft. Note the *white sutures* have been passed through the medial aspect of the graft

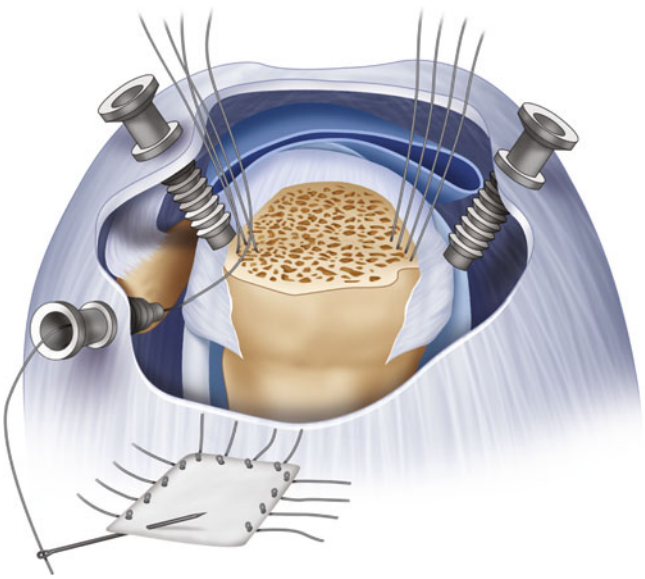

Fig. 40.8 Illustration demonstrating passage of a suture retrieved from posterior anchor through graft outside shoulder

purple STIK knots along the medial aspect. These variable color sutures facilitate retrieving the correct suture partners when tying knots.

Suture Passage

We recommend use of an 8.5 mm mid-lateral cannula and 7 mm operating cannulae in both the anterior and posterior portals. The arthroscope is maintained in the anterior cannula for the first part of the procedure. The graft with all the STIK knots in place is clipped on a moistened towel that is fixed around the upper arm just lateral to the lateral cannula. The graft is oriented such that the lateral edge is positioned away from the lateral cannula so that it represents the position that it will eventually sit after it is pulled into the shoulder.

We begin suture passing by retrieving the most posterior/medial suture from the posterior anchor out the anterolateral cannula with a crochet hook. This suture is passed through the graft from its under surface (dermal or fuzzy side) to its upper surface (shiny side) at the posterolateral corner of the graft (Fig. 40.8). A STIK knot is tied. This will eventually aid in seating the posterolateral corner of the graft to the anchor.

Next, a crescent-shaped suture passer is inserted through the posterior cannula to puncture the posterior edge of the native cuff 5 mm medial from the posterior anchor. A shuttle suture is passed through the suture passer and retrieved into the lateral cannula, taking great care to always keep each suture *anterior* to the previous one. If this step is not carefully observed, the sutures may be crossed, causing the graft to twist when it is pulled into the joint. The corresponding STIK suture is then shuttled through the native cuff tissue and pulled out the posterior cannula. These sutures are left inside the posterior cannula.

The shuttling technique is repeated, progressing along the posterior edge, the medial margin, and finally anteriorly along the rotator interval and biceps tendon (Fig. 40.9). The scope is moved to the posterior portal while the anterior stitching proceeds via the anterior cannula. A combination of straight and curved suture hooks are needed depending on the position of the tissue being stitched and the portal used. *It is imperative that the sutures are not tangled during these steps*. This is accomplished by passing each suture *anterior* to the prior one. The shuttle suture must always be retrieved between the two suture anchors (never anterior or posterior to them). It is critical to visualize the grasper every time as it enters into the joint through the lateral cannula, thus ensuring that it is passed anterior to all the other sutures within the lateral cannula (Fig. 40.10). In this careful and organized fashion, we can thereby place any number of sutures without entanglement.

A suprascapular notch portal is created and all of the white STIK sutures passing through the medial portion of the cuff stump are retrieved out this portal. This will assist in later graft passage.

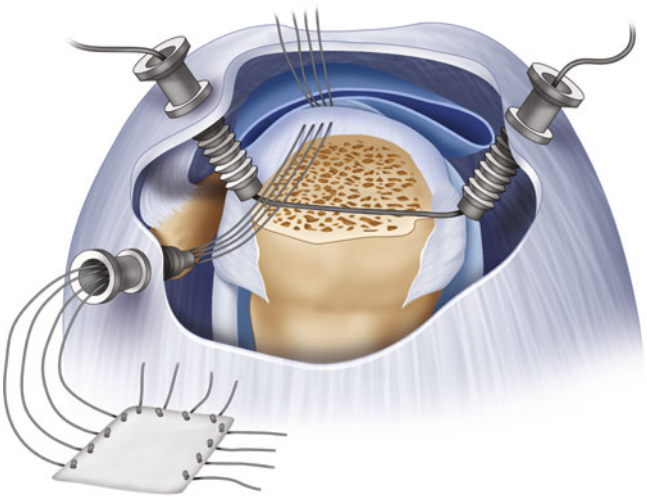

Fig. 40.9 Illustration depicting the passage of the four posterior STIK sutures through the posterior edge of the cuff tendon

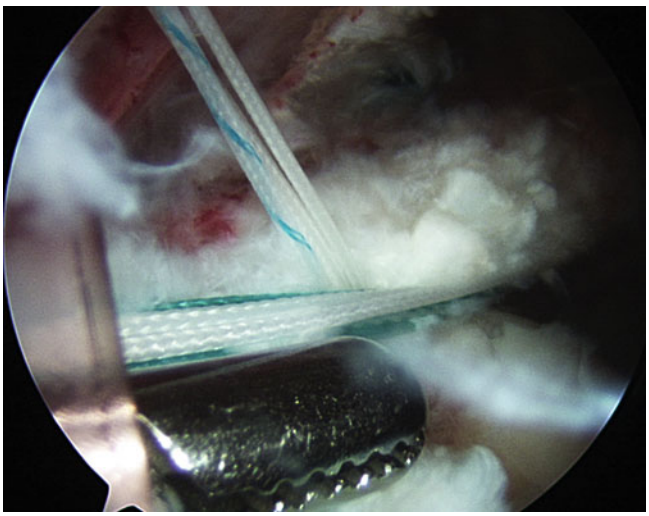

Fig. 40.10 A grasper is inserted through the lateral cannula to retrieve a shuttle suture. With the arthroscope in the anterior cannula, note that the grasper is passing anterior to the previously passed sutures exiting the lateral cannula

Finally, the most anterior/medial suture from the anterior anchor is retrieved out the anterolateral cannula with a crochet hook. Using a straight needle with an eyelet, this suture is passed through the anterolateral corner of the graft from its under surface to its upper surface. A STIK knot is tied; this will eventually aid in seating of the anterolateral corner of the graft.

Graft Insertion

After passing the STIK sutures through the tissue, the graft is ready for insertion through the lateral cannula (Fig. 40.11). The slack is pulled out of all the sutures, thereby docking the graft at the aperture of the lateral cannula. The graft is rolled onto itself to facilitate graft passage through the cannula. Next, a "push-pull" technique is used; as the graft is pushed down the cannula with a grasper, the STIK suture ends exiting through the suprascapular notch portal are pulled first, and those exiting the posterior and anterior cannulae are then pulled. Once the graft enters the subacromial space, each suture end is sequentially pulled to unfold the graft and cover the repair site. It is very helpful to always keep all slack out of the sutures obviating the possibility of having a loop of suture catch on a STIK and cause a twist of the graft.

Suture Tying

The scope is maintained in the lateral portal for all knot tying. All sutures are tied through the posterior cannula. All the free ends of the STIK sutures exiting the posterior portal are retrieved out the anterior cannula. In a systematic fashion, each suture is then tied; starting posterior/lateral, a sliding locking knot is tied with each STIK suture. The free end is first retrieved out the posterior cannula, and after loading the free end into a knot pusher, the knotted end of the STIK suture is retrieved and the knot is tied.

Lateral Fixation

Once all the STIK sutures have been tied, the lateral aspect of the graft needs fixated to the tuberosity. The final suture from the posterior anchor is then passed through the posterior portion of the lateral edge of the graft using a curved suture passer and shuttle technique. The remaining suture from the anterior anchor is then passed through the anterior portion of the lateral edge of the graft in a similar manner. These sutures can be stored in suture savers or tied at this point.

Next, either one or two double-loaded suture anchors are placed just lateral to the graft. We recommend using one double-loaded anchor for each cm of remaining lateral graft tissue. These sutures are passed through the lateral edge of the graft from posterior to anterior using a shuttle technique. The lateral sutures are then sequentially retrieved from anterior to posterior out the lateral cannula and tied down using a locking sliding knot (Fig. 40.12).

Rehabilitation

We follow the same protocol for cuff augmentation and interposition; please reference earlier section for details.

Literature Review

Several authors have reported their clinical experiences with AHDAs for human rotator cuff interposition/augmentation [64–68]. These studies have shown beneficial outcomes with no adverse effects related to the graft. Although limited, early clinical results appear promising. Of note, Snyder et al.

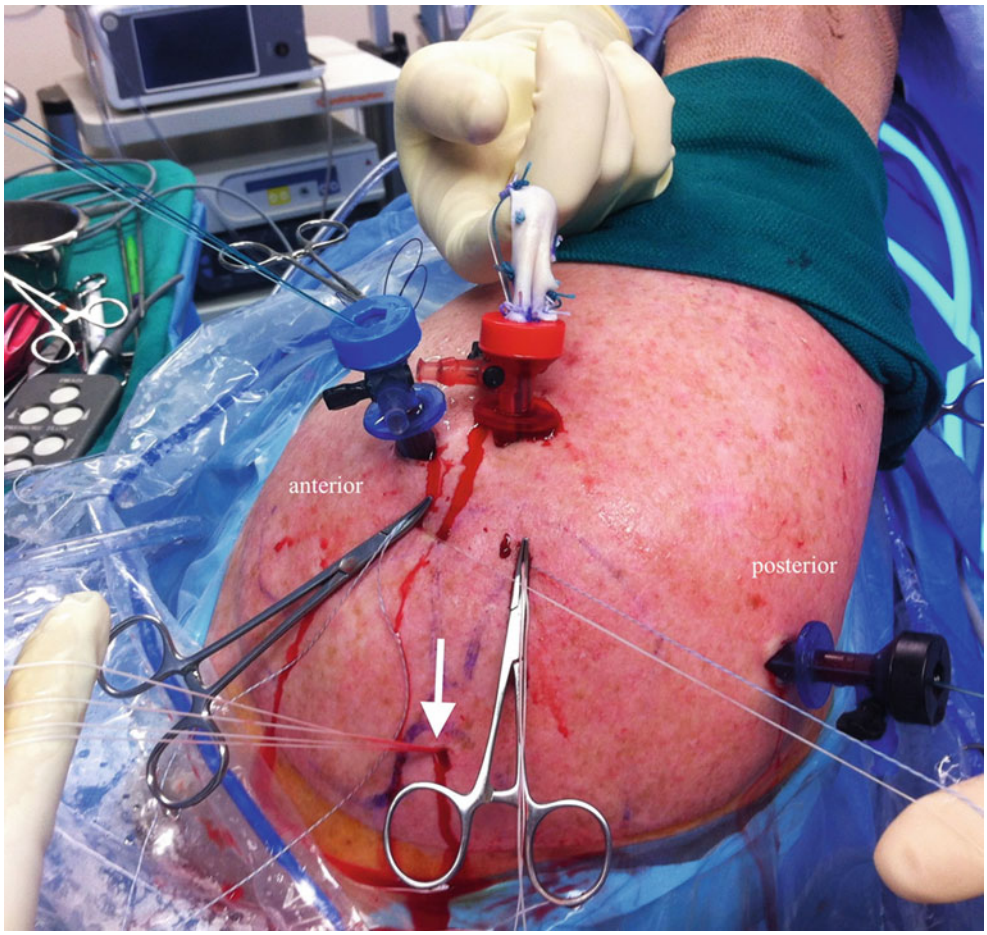

Fig. 40.11 View of a right shoulder with the graft docked at the aperture of the lateral cannula, ready for insertion into the joint. Note the sutures exiting the posterior cannula, the suprascapular notch portal (*arrow*), and the anterior cannula

reported a biopsy 3 months after rotator cuff interposition demonstrating intact graft material with infiltration of host blood vessels and other cells. There was evidence of early regeneration with no signs of inflammation [29].

Augmentation

Barber et al. recently published the only prospective randomized study on rotator cuff augmentation. They conducted a multicenter study of patients undergoing arthroscopic repair of two-tendon rotator cuff tears measuring greater than 3 cm. Twenty-two patients were randomized to arthroscopic single-row rotator cuff repair with AHDA augmentation (group 1) and 20 patients were included in the group without augmentation (group 2). At 24 months' follow-up, Constant scores and ASES scores showed significant differences between the groups in favor of augmentation. Gadolinium-enhanced MRI at 14.5 months' mean follow-up showed intact cuffs in 85 % of group 1 repairs and 40 % in group 2 repairs. No adverse events were reported [67].

Rotini et al. reported on their preliminary experience using AHDA augmentation in 5 patients. They had three intact repairs, one partial tear, and one complete re-tear at 1 year with no adverse events [69].

Recently, Agrawal reported on his experience with AHDA augmentation in a group of 14 patients [70]. At a mean follow-up of 16.8 months, 12 of 14 patients had an intact repair on MRI (85.7 %); significant improvements in strength, pain, and functional scores were reported as well.

Interposition

Bond et al. published the results of their initial experience with AHDA as an interposition graft in 2008 [65]. Sixteen patients underwent cuff reconstruction with AHDA and were followed up at a mean of 26.8 months with MRI and clinical evaluation. Mean UCLA scores improved from 18.4 to 30.4 and Constant scores improved from 53.8 to 84.0; significant improvements in pain and strength scores were reported, and 15 of 16 patients were satisfied with the results of their procedure. Thirteen of 16 patients had full graft incorporation on MRI evaluation. No complications were reported in this cohort of patients.

In 2010, Wong et al. followed up on their initial experience with AHDA interposition grafts for massive irreparable cuff tears with a larger cohort of patients [66]. Forty-five patients were followed for a minimum of 2 years (range 24–68 months), and similar outcome scores were reported in this larger group of patients; however, MRI evaluation was not reported.

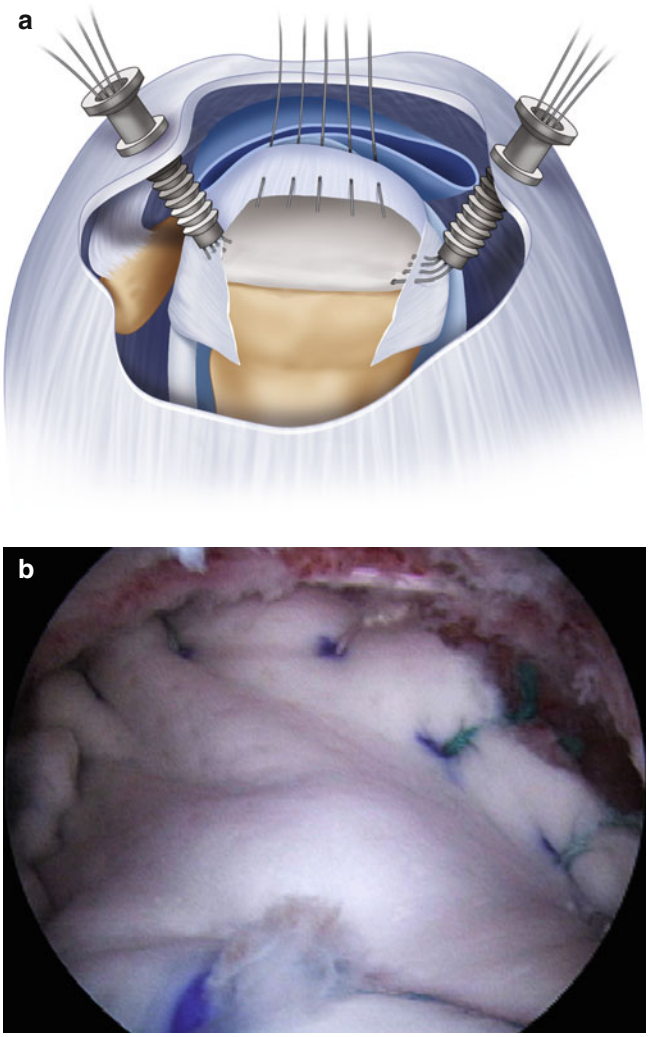

Fig. 40.12 (**a**) Illustration showing graft in place, with all STIK sutures passed through native cuff tissue. (**b**) Final arthroscopic view with interposition graft tied in place

Recently, Gupta et al. prospectively reported on their experience of AHDA graft interposition for treatment of irreparable massive cuff tears in 24 patients [71]. Using an open technique, the authors reported complete healing by ultrasonography in 76 % of patients and partial healing in the remaining patients. ASES scores improved from 66.6 to 88.7, and significant improvements in pain, strength, and functional scores were reported.

Summary

Surgical management of large and massive rotator cuff tears remains a challenging problem. AHDAs appear to be a potentially beneficial biologic and biomechanical augmentation/interposition option. Biomechanical studies support the use of AHDAs over other xenografts and synthetic scaffolds as augments for rotator cuff repairs at this time. Early clinical studies show promise for improved outcomes and healing rates for large/massive rotator cuff tears treated with graft augmentation or interposition. While these scaffolds provide a unique three-dimensional structure and serve as a conduit for regeneration of rotator cuff tissue, the unique biology of the tendon-bone interface has yet to be reproduced and remains an ongoing goal for researchers [72, 73]. Until then, graft augmentation/interposition appears to be a promising step towards improving outcomes of rotator cuff repairs, especially for chronic, large/massive tears that remain challenging even for the most experienced of surgeons.

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The Role of Platelet-Rich Plasma in Rotator Cuff Repair

41

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Introduction

Rotator cuff tendon tears are a common source of shoulder pain and combine both traumatic and degenerative elements. The incidence of this condition is increasing along with an aging population [1]. The management of rotator cuff tears is complex and multifactorial. Partial-thickness tears may heal with conservative management and avoidance of predisposing factors. Operative treatment allows primary repair to be performed as either an open or an arthroscopic procedure. Despite satisfactory results for primary rotator cuff repair, the incidence of persistent tendon defects or retears is still significant [2–5].

Several studies have demonstrated that native tendon-bone insertions are not restored after tendon-to-bone repair [6]. Healing of repaired tendons occurs via fibrous scar tissue formation rather than via the regeneration of a histologically normal insertion, and thus repaired tendons have inferior mechanical properties and are more susceptible to retearing [7–9]. Considering the relatively high percentage of repair failure, reported as 11–94 % [5, 10–12], it is important to explore techniques of biological augmentation to reduce the postsurgical recurrence rate and improve long-term shoulder function after rotator cuff repair.

The use of platelet-rich plasma (PRP) as a biological solution to improve rotator cuff tendon healing has gained popularity over the last several years. There are a variety of PRP formulations and techniques used to augment rotator cuff. This chapter discusses and explores the available evidence to determine the efficacy of the PRP in arthroscopic rotator cuff repair. Emphasis will be placed on published peer review data investigating the role of this biological tool in rotator cuff tendon healing.

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Platelet-Rich Plasma

Platelet-rich plasma (PRP) is a whole blood fraction containing high platelet concentrations that, once activated, provides a release of various growth factors (GFs) that participate in tissue repair processes. PRP includes many of the GFs identified as crucial in normal bone-to-tendon healing, such as transforming growth factor-beta (TGF-β), fibroblast growth factor (FGF), platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF), connective tissue growth factors, and epidermal growth factor (EGF) [13, 14].

Platelets also have dense granules that house a variety of important bioactive molecules such as serotonin, calcium, and adenosine [15]. Over 300 proteins have been identified in platelet releasate using a proteomics approach. These proteins function in an autocrine or paracrine fashion to modulate cell signaling and chemotaxis promoting tendon healing.

There are clear differences in PRP formulations in terms of GF concentration and catabolic enzyme content [16]. A PRP classification system exists which is based on the presence or absence of white blood cells and whether the PRP is used in an activated or inactivated form. Platelets can be activated ex vivo with thrombin and/or calcium. This technique can result in immediate release of GFs. Use of PRP in an inactivated manner, without thrombin or calcium, relies on in vivo activation via endogenous collagen. PRP may be prepared via centrifugation as a pure platelet concentrate suspended in plasma or as a mixture with white blood cells. These versions have been known as leukocyte-poor and leukocyte-rich PRP, respectively [17].

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Literature Review

The rotator cuff literature has shown that PRP can be applied by either direct injection or local application of a PRP matrix scaffold to repaired tissues. The main characteristics of clinical studies analyzed in this paper are reported in Table 41.1.

Randelli et al. [18] published the first paper investigating the use of PRP in rotator cuff repair. Fourteen patients received an intraoperative injection of activated PRP at the end of the surgical procedure. The PRP was obtained by centrifugation of 54 ml of whole blood drawn preoperatively from the patients. The PRP was mixed to concentrated plasma and activated with an autologous thrombin component (Fig. 41.1). A single-row suture anchor technique was used to repair the rotator cuff tears. The PRP was delivered between the bone and the repaired rotator cuff in a dried subacromial space (Figs. 41.2 and 41.3). Patients started passive assisted exercises at 10 days after surgery and were prospectively followed for 24 months. A significant improvement in terms of VAS, UCLA, and Constant score was observed during the observed period compared to the preoperative values. The authors concluded that their technique for PRP application in arthroscopic shoulder surgery was effective and safe with no reported complications. However, this investigation did not include a control group and no evaluation of the repair integrity was performed at last follow-up.

Using a similar PRP preparation technique, Randelli et al. [20] recently published the results of a randomized controlled study on the efficacy of intraoperative use of PRP in arthroscopic rotator cuff repair. The subjects were randomly divided into two groups, and those in the treatment group received a local injection of PRP at the end of surgery. The control group did not receive any additional treatment outside of the standard arthroscopic repair. All patients had the same accelerated rehabilitation protocol. The pain score in the treatment group was lower than the control group at 3, 7, 14, and 30 days after surgery ($p<0.05$). Clinical outcomes were significantly higher in the treatment group than the control group at 3 months after surgery ($p<0.05$). There was no difference between the two groups after 6, 12, and 24 months. Magnetic resonance imaging (MRI) studies at a minimum 1-year follow-up showed no significant difference in the healing rate of the rotator cuff (PRP group, 40 %; control group, 52 %; $p>0.05$). In the subgroup of patients with smaller tears, strength in external rotation in the PRP group was significantly higher at 3, 6, 12, and 24 months postoperative ($p<0.05$). The number of identified retears was 2 (14 %) in the PRP subgroup and 6 (37 %) in the control subgroup ($p>0.05$).

Jo et al. [21] augmented surgical treatment of full-thickness rotator cuff tears in 19 patients by using PRP activated with 10 % calcium gluconate. They compared this treatment group to a control group of 23 patients who underwent standard repair. PRP was prepared 1 day before surgery via plateletpheresis. A minimal amount of white blood cells was found in the PRP gel. An attempt was made to standardize the PRP at a target concentration of 3.5-fold increase over baseline. It was applied in the form of a gel threaded to a suture and placed at the interface between the tendon and bone. The rotator cuff repair was performed using a transosseous-equivalent technique in all cases. Passive range of motion and active-assisted exercises were allowed at 4–6 weeks after surgery depending on the size of the tear. Higher functional scores were observed in the control group at 3 months postoperatively; however, bias existed in this data set. The PRP group contained a larger proportion of patients with massive tears that began rehabilitation 6 weeks after surgery while the

Table 41.1 Studies investigating the use of PRP in arthroscopic rotator cuff repair

Authors	Evidence	PRP formulation	No. of patients	Surgical technique
Randelli et al. [18]	Level 4 Prospective case series	Injectable PRP (GPS system Biomet Biologics)	14	Single-row technique
Castricini et al. [19]	Level 1 Randomized controlled study	Suturable PRP (CASCADE system)	88	Double-row technique
Randelli et al. [20]	Level 1 Randomized controlled study	Injectable PRP (GPS system Biomet Biologics)	53	Single-row technique
Jo et al. [21]	Level 2 Prospective cohort study	Suturable PRP (Cell-separator system, COBE Spectra LRS Turbo)	42	Transosseous-equivalent technique
Barber et al. [22]	Level 3 Case control study	Suturable PRP (CASCADE system)	40	Single-row technique
Gumina et al. [23]	Level 1 Randomized controlled study	Suturable PRP (RegenKit; Regen Lab)	76	Single-row technique
Rodeo et al. [24]	Level 2 Randomized controlled study	Suturable PRP (CASCADE system)	67	Single- or double-row/ Transosseous-equivalent technique
Bergeson et al. [25]	Level 3 Cohort study	Suturable PRP (CASCADE system)	37	Single- or double-row technique

Fig. 41.1 PRP and autologous thrombin transferred to the sterile field at the end of the surgical repair

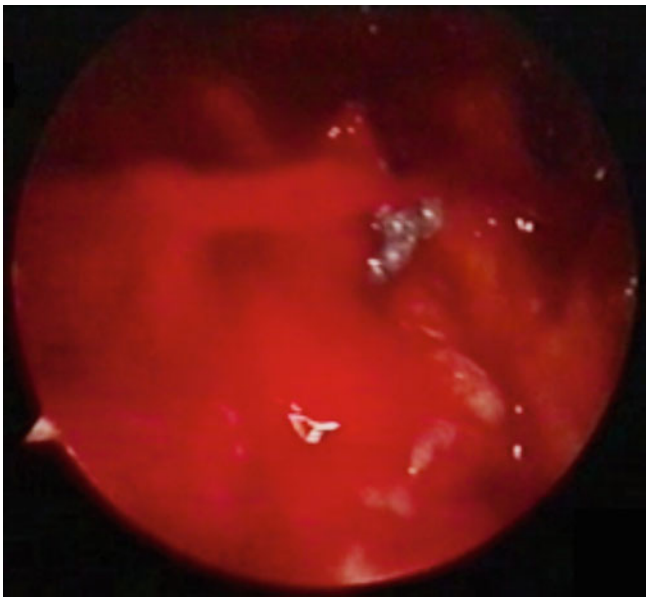

Fig. 41.3 Rotator cuff repair after PRP injection

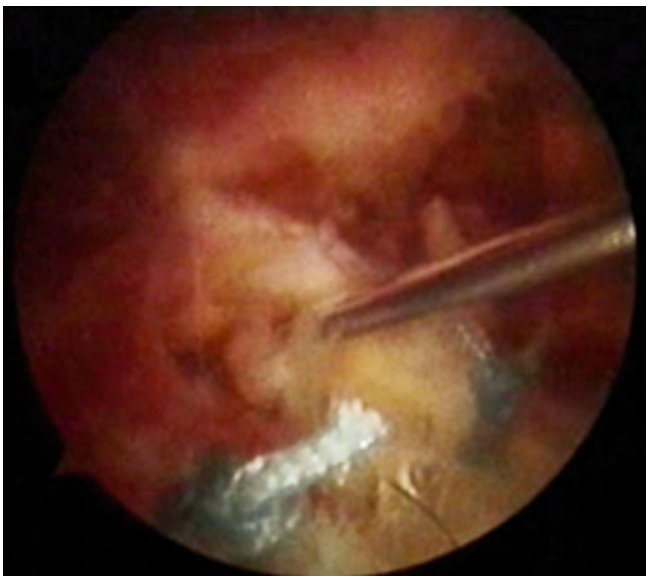

Fig. 41.2 Arthroscopic rotator cuff repair

control group favored those with small-to-large tears that began rehabilitation at 4 weeks. No significant difference was seen at 6 and 12 months and at the final follow-up. Despite the greater proportion of large-to-massive tears in the PRP group, MRI showed a retear rate of 26.7 % in the PRP group compared to 41.2 % in the control group at a minimum of 9 months after surgery ($p > 0.05$). In tears less than 3 cm the retear rate was 12.5 % (1 of 8) for patients in the PRP group and 35.7 % (5 of 14) for patients in the control group ($p > 0.05$).

Gumina et al. [23] most recently evaluated the clinical and magnetic resonance imaging (MRI) results of arthroscopic rotator cuff repair with and without the use of platelet-leukocyte membrane in patients with a large posterosuperior rotator cuff tear. Eighty consecutive patients were randomized to treatment either with or without a platelet-leukocyte membrane inserted between the rotator cuff tendon and its footprint. Platelet-rich plasma was obtained from 10 ml of peripheral blood drawn preoperatively from the patients (RegenKit; Regen Lab, Le Mont-sur-Lausanne, Switzerland). A second spin of the PRP was done in the presence of calcium gluconate and batroxobin for 20–30 min to obtain a round membrane with a diameter of 13 mm and a thickness of 3–4 mm which was incorporated into the suture construct repair of the PRP patients group (40 patients). All tears were repaired using an arthroscopic single-row technique. One membrane was utilized for each anchor in the PRP group. Passive shoulder motion was initiated under supervision during the first postoperative week. At a mean of 13 months of follow-up, rotator cuff retears were observed only in the control group. The use of the membrane was associated with significantly better repair integrity ($p = 0.04$).

Several authors used the CASCADE Autologous Platelet System (Cascade Medical Enterprises, Wayne, NJ) to create a platelet-rich plasma from autologous blood.

Castricini et al. [19] firstly performed a prospective randomized controlled double-blind study of 88 patients undergoing arthroscopic rotator cuff repair with (43 patients) and without (45 patients) augmentation with autologous platelet-rich fibrin matrix (PRFM). The membrane of autologous suturable fibrin was obtained by processing 9 ml of venous blood drawn from patients before surgery. A second spin of the PRP was done in the presence of calcium chloride for 25 min. This resulting membrane was incorporated into the suture construct and placed at the interface between the tendon and the greater tuberosity under continuous saline lavage. A double-row suture anchor technique was used in

all cases. Passive and assisted active exercises were initiated after 3 weeks of immobilization. At the 16-month follow-up evaluation, there was no statistically significant difference in functional outcomes as measured by Constant score or by MRI appearance. However, the retear rate was 10.5 % in the control group compared to 2.5 % in the treatment group with a statistical trend approaching significance ($p=0.07$). Arnoczky reevaluated the MRI data from this paper using the binomial chi-square test. He concluded that the PRP augmented rotator cuff repairs result in a statistically significant return to normal footprint ($p=0.02$) and signal intensity ($p<0.001$) [26].

Using the same PRP preparation technique, Rodeo et al. [24] most recently published results of a prospective, randomized, double-blinded study on the efficacy of use of PRFM (CASCADE Autologous Platelet System) in arthroscopic rotator cuff repair. Forty patients were randomized to receive the experimental treatment and 39 were randomized to the control group. PRP group was treated with a single membrane of autologous suturable fibrin that was placed at the interface between the tendon and the greater tuberosity. A single-row repair was performed in 26 patients. In the remaining patients the rotator cuff was repaired using a double-row technique. Tendon healing was evaluated by ultrasound at 6 and 12 weeks. No difference in tendon healing was found between the two groups. At 12 weeks, 80.6 % rotator cuff tendons were intact in the control group compared with 66.7 % in the PRP group ($p=0.2$). The vascularity in the peribursal, peritendinous, and musculotendinous areas also was comparable in the two groups ($p>0.05$).

Barber et al. [22] prospectively compared 40 patients undergoing arthroscopic rotator cuff repair with and without PRP augmentation. The PRP group protocol consisted of drawing 18 ml of whole blood from patients preoperatively to form two membranes of autologous suturable fibrin that were then inserted between the tendon and bone during the surgical repair. An arthroscopic single-row rotator cuff repair was performed in all cases and passive rehabilitation was allowed at 6 weeks. MRI studies obtained at 4 months after surgery showed persistent full-thickness tendon defects in 60 % of the control group compared to 30 % of the PRP augmented group ($p=0.03$). In tears of less than 3 cm, the PRP group had an 86 % healing rate compared to a 50 % healing rate in the control group ($p<0.05$). No difference was observed in clinical outcome scores at the unique final follow-up (average: 31 months).

Similarly, Bergeson et al. [25] used PRFM clot (CASCADE Autologous Platelet System) to augment arthroscopic cuff repair of 16 patients with rotator cuff tears at risk for retears. The PRP group was compared to a historical control group of 21 patients with similar at risk tears who underwent standard repair without PRP augmentation. Single- or double-row techniques were performed at the discretion of the surgeon in both the PRP and control group. Passive range of motion was started at 4–6 weeks postoperatively. MRI studies at a minimum of 1-year follow-up showed a significant difference in the retear rate in favor of the control group (PRP group, 56 %; control group, 38 %; $p=0.024$). The significant difference remained when double-row repairs were removed from the analysis (PRP group, 62 %; control group, 40 %; $p=0.022$).

Data Analysis

Clinical studies published so far had different experimental designs and the strength of evidence has ranged from 1 to 4. Furthermore, 4 different PRP preparation systems were used among the studies (Table 41.1). The volume of autologous blood, centrifugation rate and time, delivery method, activating agent, leukocyte concentration, final PRP volume, and final platelet and GF concentrations vary among these systems.

The surgical technique (transosseous-equivalent, single- or double-row technique) and rehabilitation protocol (standard or accelerated) also were not the same across the trials. In 4 of them a single-row technique was used for arthroscopic cuff repair. A double-row technique was used in 2 trials, whereas in the remaining 2 studies, the rotator cuff repair was performed at the discretion of the surgeon with a single- or double-row technique. There were also differences in rotator cuff tear sizes and tendon involved among the studies.

Although there are differences in the surgical techniques, PRP preparation, and tear size, the retear rate was recalculated by pooling the available data from trials in order to determine the PRP potentiality for rotator cuff healing. The difference in terms of retear rate between the PRP and control group was assessed by a chi-square test or Fisher's exact test.

The analysis of all seven studies showed there was no significant difference in the pooled retear rate between the PRP and control group. The retear rate was 22 % (41 of 188) and 28 % (52 of 187), respectively ($p>0.05$). Data from the pilot study of Randelli et al. [18] were not available, and they were not included in this analysis.

Interestingly, a trend towards a lower rotator cuff retear rate in the PRP group was found when the results from Level 1 studies were pooled alone (9.9 % and 19.4 % in the PRP and control group, respectively; $p=0.05$; chi-square test) (Table 41.2) [19, 20, 23].

Three studies identified small and medium cuff tears as those measuring less than 3 cm [19, 21, 22]. Randelli et al. [20] graded rotator cuff tears according to the amount of retraction. If the tear exposed the humeral head but did not retract all the way to the glenoid surface, it was defined as a

Table 41.2 Retear rate by pooling data from the available trials

Analysis for Level 1 studies			Analysis for small and medium tears		
Authors	PRP	Control	Authors	PRP	Control
Castricini et al. [19]	2.5 % (1/40)	10.5 % (4/38)	Castricini et al. [19]	2.5 % (1/40)	10.5 % (4/38)
Randelli et al. [20]	40.9 % (9/22)	52 % (12/23)	Randelli et al. [20]	14.2 % (2/14)	37.5 % (6/16)
Gumina et al. [23]	0 % (0/39)	8.2 % (3/37)	Jo et al. [21]	12.5 % (1/8)	35.7 % (5/14)
			Barber et al. [22]	14.2 % (2/14)	50 % (7/14)
Total*	**9.9 % (10/101)**	**19.4 % (19/98)**	**Total****	**7.9 % (6/76)**	**26.8 % (22/82)**

* p-value = 0.05; ** p-value = 0.002

small or medium tear. A significant difference was found when a stratified analysis was made to analyze results of small and medium cuff tears. The rate of retear was 7.9 % among patients treated with PRP compared to 26.8 % of those treated without PRP ($p=0.002$; chi-square test) (Table 41.2). Data about retear rates for small and medium cuff tears were not available in 3 clinical studies, and they were not included in this analysis [23–25].

No difference was found when a subgroup analysis was performed for studies in which patients underwent a double-row or transosseous-equivalent repair [PRP group, 19.3 % (16 of 83); control group, 20.8 % (16 of 77); $p>0.05$] [19, 21, 24, 25]. Similarly, analyzing patients who underwent single-row repair, no significant difference was found between the PRP and control group [23.8 % (25 of 105) and 32.7 % (36 of 110), respectively; $p>0.05$] [20, 22–25].

No difference was seen when we conducted the analysis for clinical studies that used the same PRP preparation systems (CASCADE Autologous Platelet System) [19, 22, 24, 25]. The retear rate was 25 % (28 of 112) and 27 % (30 of 110) for patients treated with PRP and those treated without PRP, respectively ($p>0.05$). Notably, except for 2 cases of infections, no complications were reported from its use. Bergeson et al. [25] showed infection rate of 12 % among patients treated with PRFM compared to 0 % in the control group. However, this difference did not reach the statistical significance and no differences in infection rates or complication rates were found in the remainder of the seven studies.

Discussion

Although clinical studies have produced conflicting results, literature data suggest a beneficial effect on the healing process when PRP is applied during rotator cuff repair. Particularly, a borderline significance level ($p=0.05$) was found when we pooled data from studies with higher level of evidence (Level 1). The stratified analysis of small or medium cuff tears has showed a significant lower overall retear rate in the PRP group. Therefore, PRP can improve healing of small and medium rotator cuff tears that may be more prone to the biological enhancement by GFs.

Namazi recently has highlighted the major mechanisms by which platelet-rich plasma could reduce the rotator cuff retear rate [27]. The interleukin-1β beta (IL-1β beta) level is correlated with degeneration of the rotator cuff tendon. In contrast, TGF-β (beta) can enhance cuff tendon repair strength. Recent studies have shown that PRP not only can inhibit inflammatory effects of IL-1β but also can potentiate TGF-β (beta) production [28, 29].

Furthermore, recently, in vitro studies evaluating the PRP effect on human tenocytes isolated from human rotator cuff tendons with degenerative tears have shown that platelet-released GFs can enhance cell tenocyte proliferation and promote synthesis of tendon extracellular matrix [30, 31].

Summary

Clinical studies have produced conflicting results, and definitive conclusions on the efficacy of the use of PRP in rotator cuff repair are difficult to draw. More prospective randomized controlled studies (Level 1) are needed to determine the role of PRP in improving healing when compared with standard treatment, especially in small-to-medium sized tears.

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Arthroscopically Assisted Latissimus Dorsi Transfer

42

Enrico Gervasi and Alessandro Spicuzza

Introduction

Surgery of rotator cuff tears was initially focused on anatomic repair of the tendon tear. However, imaging studies demonstrated high failure or recurrence rates after tendon-to-bone repair of massive rotator cuff tears, even for complete and "apparently" firm repairs [1, 2]. Therefore, search for effective alternatives to tendon-to-bone repair led to consider several solutions to treat massive, irreparable rotator cuff deficiency, such as debridement [3], partial rotator cuff repair [4], subscapularis tendon transfer [5], transfer of the subscapularis and teres minor [6], transfer of the long head of triceps [7], teres major transfer [8, 9], interposition of a biceps tendon autograft [10], freeze-dried rotator cuff allograft [11], and synthetic grafts [12].

Muscle transfers techniques were used in the past to treat patients with neurological injuries, such as obstetric palsies. In these patients, external rotation was impaired due to injury of the cervical nerve roots (the fifth cervical root being the most involved), and internal rotators become predominant, resulting in the inability of the patients to bring their hand to the mouth or neck.

The L'Episcopo technique [13] was used in these conditions to correct the external rotation deficit and can be considered the precursor of muscle transfer techniques currently used to address irreparable tears of the posterior-superior cuff. It consists of detachment of latissimus dorsi(LD) and teres major (TM) tendons off from the medial edge of the bicipital groove and their reinsertion to the humeral diaphysis, rotated in the opposite direction: from medial and posterior to lateral and anterior. Gerber et al. [14] first modified and adapted this open technique for the treatment of irreparable rotator cuff tears. More recently, an arthroscopically assisted latissimus dorsi transfer (LDT) technique was developed for the treatment of massive, irreparable posterior-superior rotator cuff tears [15].

Why the arthroscopic choice? Because the scope allows a thorough management of intra-articular and extra-articular steps of the procedure, is less invasive, and associated with lesser morbidity than the scalpel. Moreover, a deltoid-sparing approach also guarantees a way out in case of failure, when the disease progress toward a cuff-tear arthropathy and joint replacement with a reverse shoulder arthroplasty (RSA) becomes necessary.

Pathophysiology

Massive, Irreparable Rotator Cuff Tears

Different classifications define as "massive" a tear in relation to particular aspects: number of tendons involved, tear retraction, tear extension in width, and acromiohumeral distance. Indeed, each classification has limitations, and there are no classifications of massive tears specifically focused on indication to tendon transfer.

Among massive cuff tears, some have "dignity" to be repaired; the others, even when the repair is technically feasible, are doomed to fail due to changes in the tendons (tendon degeneration and poor tissue vitality) and/or muscles (atrophy and fatty infiltration).

Striated voluntary muscles differ for some characteristics depending on their function, such as length, cross-sectional area, direction of the fibers with respect to force vectors (pennation), number of muscle bellies, type of contraction, return speed, and movement control (fine-tuned or not). The total tension in a muscle depends on the amount of the active fraction, which is the sum of active (contractile) and passive forces. The atrophic muscle, besides not producing active contraction, is anelastic; therefore, it stretches rapidly and does not work passively as a spring. We consider that the concept of reparability should be replaced by that of "opportunity of repair."

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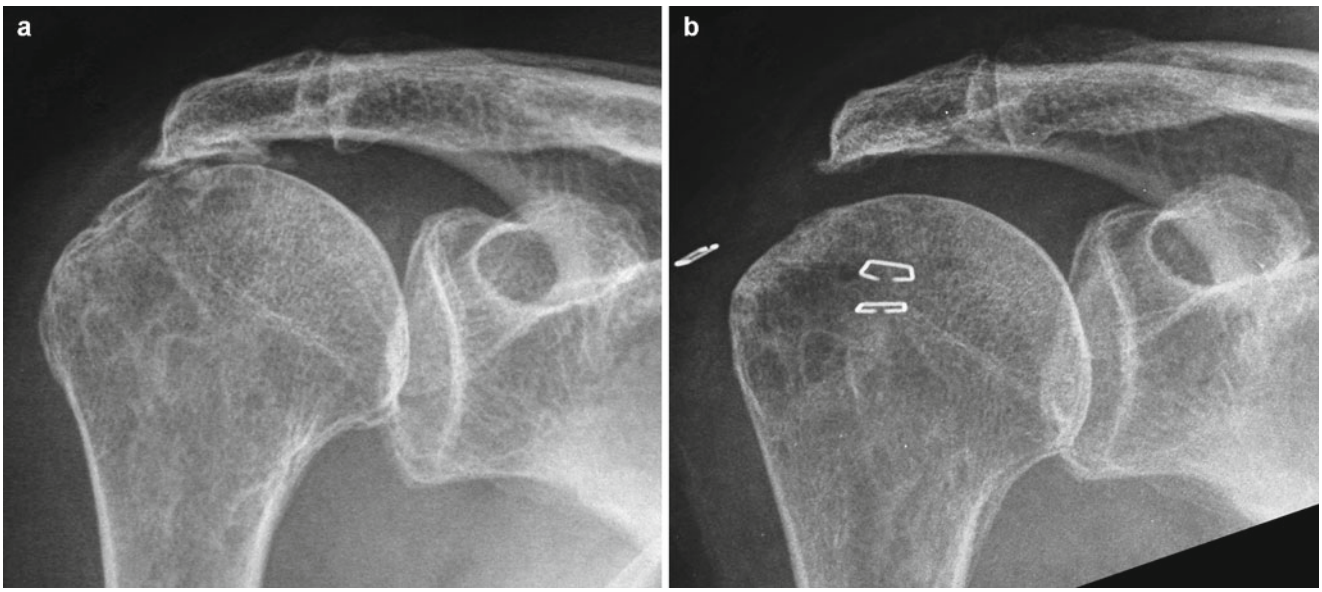

Fig. 42.1 Humeral head re-centered. (**a**) Preoperative X-ray. (**b**) Postoperative X-ray (© Enrico Gervasi MD reproduced with permission)

Bone Components

Radiographic examination of a shoulder with a massive rotator cuff tear can show the following different patterns:

- Cranial migration of the humeral head with minimal signs of bone adaptation
- Contact between the humeral head and the undersurface of the acromion and anterior acromial spur, which limits upper humeral migration

In the latter situation, the relationship between the rotation center of the humeral head and the center of the glenoid is maintained, as the intact acromial arc opposes to the cranial migration of the humeral head. Therefore, excessive shortening of the deltoid muscle fibers is prevented during muscle activation, and the linear force vector of the deltoid produces the rotation of the humeral head. The more congruent are the surfaces of the acromiohumeral neo-articulation, modified by taking an acetabular pattern, the greater is the described effect. Even the remaining rotator cuff tendons take advantage of it, as they are not required to supply humeral head centering and can solely act as rotators. This explains why the acromioplasty sometimes leads to a functionally catastrophic result: a weak elevation becomes pseudoparalysis after surgery.

"Cotyloid" appearance of the glenoid depends on wearing of the bone surfaces, due to friction during movement. Therefore, patients with valid movement and massive cuff tear can be expected to have "compensatory" aspect of concentric rather than eccentric, glenoid wear. On the contrary, patients with acute onset of severe deficit are often affected by traumatic extension of an existing lesion. As it happens in the absence of gradual adaptation, the bone surfaces are regular.

According to some authors, cranial migration of the humeral head and reduction of the acromiohumeral distance are contraindications to tendon transfer, as it cannot contribute to re-centering of the humeral head. In our experience, routine postoperative radiographs brought us to consider that the transferred tendon would act as a passive restraint. Hence, we consider as favorable prognostic factor the postoperative widening of the acromiohumeral space (Fig. 42.1).

Dynamic Equilibrium

Established the influence of bone components, we have to focus on the dynamic equilibrium of the scapular-humeral articulation. Balancing of muscle forces directed in opposite directions favors the stability of the center of rotation of the humeral head, which is subjected to the deltoid action. The cocontraction of the anterior (subscapularis) and posterior (infraspinatus and teres minor) muscles determines the elevation in the scapular plane. Loss of balancing forces in the sagittal plane causes rotation first, then translation of the humeral head with loss of its centering. This is similar to what happens in the voluntary shoulder instability: inhibition of agonist muscles and simultaneous sudden contraction of the antagonists. Humeral instability due to force imbalance in the massive, decompensated cuff tears is the cause of weakness and consequent inability to abduct the arm. For these reasons tendon transfers, when possible, should be associated with repair of the rotator cuff tear, especially the proximal part of the subscapularis. This might improve the outcome by balancing of the force couples and humeral head centering.

Author's Classification of Massive Rotator Cuff Tears Related to Tendon Transfer

Let us consider full-thickness retracted rotator cuff tears on the sagittal plane and assimilate the tendon tears to the atrophy of their muscle bellies [16], without further distinctions between the two conditions (they are functionally comparable). The supraspinatus is not considered in the following classification as it is always torn and has no relevance in making a treatment choice. According to tear location, we classify the tears into posterior (P) and anterior (A). The letter "t," which means "total," can be associated to P and A if the tear involves the teres minor at the posterior side (Pt) or the distal part of the subscapularis at the anterior side (At). This extension is clinically relevant because it changes the prognosis. Therefore, tear pattern can be classified as follows (Fig. 42.2):

- P: infraspinatus
- Pt: infraspinatus + teres minor
- PtA: infraspinatus + teres minor + proximal part of subscapularis
- A: proximal part of subscapularis
- At: subscapularis (almost entire)
- PA: infraspinatus + proximal part of subscapularis
- PtAt: the whole rotator cuff (no tendons are intact)

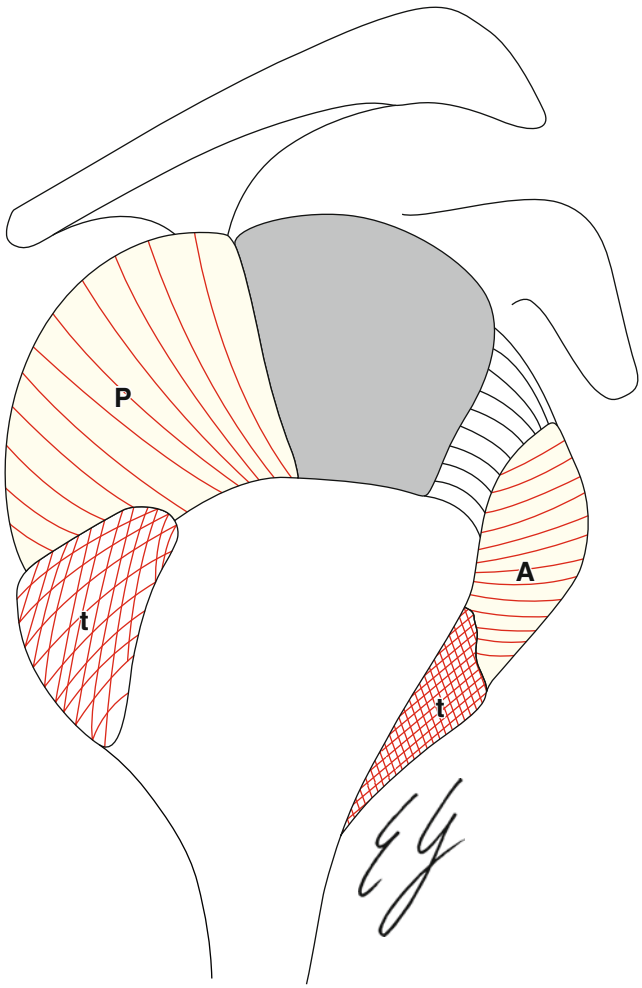

Fig. 42.2 Cuff tears classification set for transfers. *P* posterior cuff, *t* total (teres minor included). *A* anterior cuff, *t* total (inferior subscapularis included) (© Enrico Gervasi MD reproduced with permission)

Rationale of Tendon Transfer

Patients eligible for tendon transfer are those with a cuff tear classified with "P" according to the specific classification for transfers (see above). When "P" comes before other letters, this means that the main deficiency is at the posterior cuff. When "P" is associated with "A," then the tear extends to the subscapularis, which has to be repaired before LDT.

Potential advantages of the tendon transfer are related to the following issues:

- Brain sets motor tasks rather than activity of individual muscles.
- The transposed tendon is not affected by regressive phenomena (degeneration), as conversely happens to the cuff tendons working in the subacromial space.
- The success of the "tendon-transfer surgery" is less influenced by the typical "lazy biology" of the torn cuff.

Graft Choice

Graft suitability varies according to some factors, such as strength (size and force vectors), excursion, and synergistic effect (if the graft is natural agonist for the function to be restored). Graft accessibility is another relevant issue in making a choice, as harvesting and transfer should be performed with the lowest risk of injury to the surrounding soft tissues and neurovascular structures.

Herzberg et al. [17] reported on some anatomic features of shoulder girdle muscles which are potential graft sources for tendon transfer. These muscles were classified into three categories: thoraco-scapular, thoraco-humeral, and scapular-humeral. The following characteristics were analyzed: length at rest, potential range of elongation, and relative basal tension (ratio between the cross-sectional area of each muscle and that of the whole group of muscles acting on the shoulder). The relative basal tension and potential elongation of the grafts were compared with those of the muscle-tendon units to be replaced, to estimate their suitability. The results of this study showed that potential grafts vary more according to strength than potential elongation. However, analysis was exclusively performed on the sagittal plane, for the measurement of the cross-sectional area, and the coronal plane, to assess length at rest and potential elongation. Therefore, muscle features were not investigated along the real direction of the force vectors.

LDT to supply the posterior cuff can be performed as isolated or combined with the TM transfer. Indeed, relative basal tension of LD is small and can be increased by simultaneous transfer of TM [17]. However, the LD tendon is considerably longer than that of TM, and less tension is required on LT muscle to achieve isolated transfer [18]. Furthermore, potential elongation of TM is very limited, and combined LD and TM transfer is unsuitable in some patients because the TM is too short and bulky [19]. Therefore, if the TM-LD tendon complex is transferred as a single unit, the TM wraps and restrains the LD, thus hindering it to reach the most anterior part of the greater tuberosity. Finally, the excessive volume of the two muscle bellies might cause compression injuries to the axillary nerve. For these reasons, the isolated LDT seems to be the most consistent transfer procedure to address massive, irreparable tears of the posterior-superior rotator cuff.

Graft Fixation

Ling et al. [20] performed a biomechanical study by a three-dimensional upper-extremity computational model and showed that graft fixation at the insertion site of the infraspinatus tendon was preferable to those of both the supraspinatus and subscapularis tendons, while insertion site of the teres minor tendon was not recommended.

Conversely, we fix the tendon to the most anterior part of the greater tuberosity, in continuity with the footprint of the subscapularis. According to our hypothesis, the transposed LD acts as an active external rotator and also by a passive stabilizing effect on the humeral head. This favors the action of the deltoid, in a similar way to the constraint of a RSA. Furthermore, we abandoned the idea of using part of the transferred tendon to "cover the hole" by fixing it medially to the torn cuff, as proposed by other authors.

Decision-Making Algorithm

Tendon transfer should be considered when direct tendon-to-bone repair cannot predict function recover, which depends on the topography of cuff lesion.

Clinical Examination

The physical examination is important to assess deltoid integrity and joint stiffness, which is relatively frequent in patients who undergo tendon transfer after trauma or failed previous surgery. Indeed, expected results in revisions are less favorable, and stiffness, even inconspicuous, prejudices the possibility of humeral head re-centering, thus impairing functional effectiveness of the transfer.

Some patients have a functionally compensated large or massive cuff tear, and their main concern is pain. If function is partially regained after an injection of local anesthetic into the subacromial space due to pain reduction, then tendon transfer is not indicated as the prerequisite of essential loss of function is lacking. In these cases, palliative and less invasive procedures, such as biceps tenotomy and smooth contouring of the greater tuberosity, can be considered. In fact, these shoulders have a relatively stable humeral head, as contact between the humerus and acromion acts as a fulcrum for the deltoid to elevate the arm.

Imaging

Preoperative imaging studies include a radiographic series (true anterior-posterior, outlet and axillary views). Radiographic exams can show signs of arthropathy (glenohumeral space reduction) and acetabular transformation of the acromion, with alteration of the profile of the upper glenoid. The presence of these features represents a contraindication to the procedure.

Magnetic resonance imaging (MRI) is routinely performed to assess the tendon defect on the coronal oblique and axial images; muscle atrophy and fatty infiltration can be quantified on the sagittal images. MRI is crucial to the preoperative planning, especially to evaluate the subscapularis tendon. If a concomitant subscapularis tear has to be addressed before tendon transfer, the operating room should be set by placing the patient in the modified beach-chair position rather than in lateral decubitus, as a rule.

Indications and Contraindications

The ideal candidate for tendon transfer is a patient with massive, irreparable rotator cuff tear with loss of function of the upper limb incompatible with activities of daily living.

The goals of surgery, limitations, and possible complications as well should be discussed with the patient during the visit. Transfer is not suitable for return to repetitive manual work, as partially restored force is exhaustible, and has no resistance to fatigue or cyclic loading. The weight of the limb itself, when significant, adversely affects the prognosis. Therefore, it is important to point out that recovery, even when good, is never complete and considerable efforts must be forever avoided.

Patient's compliance to understand objectives and limits of the long-lasting postoperative rehabilitation is crucial to the success of the procedure. Although mechanical improvement by tenodesis effect of the transferred tendon is used during early postoperative phase, a long rehabilitative program is required to reprogram the motor tasks by selective stimulation of the transposed unit. Therefore, older patients

must be carefully drafted to a so complex treatment, since RSA is a viable alternative, which requires simpler and shorter postoperative rehabilitation.

Although shoulder replacement is rarely indicated in patients younger than 70 years, treatment choice should be based on factors other than age, such as general health status ("biologic age"), patient activity (involvement in recreational sports) and expectations, cartilage integrity, and subscapularis integrity or reparability.

When a concomitant subscapularis tendon tear cannot be repaired, the LDT is ineffective. This rule has anecdotal exceptions, whose biomechanical justification (why sometimes the transposed tendon is still effective?) is not yet clear. The combined anterior and posterior transfer is definitively rare in the literature, and reported results are unsatisfactory, probably because it is impossible to reprogram either the external and internal rotation motor tasks.

Surgery is contraindicated when the acromiohumeral contact caused morphologic changes to the humeral head, glenoid ("cotyloid" aspect), or acromion. Rounding of the greater tuberosity is not a contraindication.

Absolute contraindications to transfer are severe rotator cuff tear arthropathy; chronic, irreparable deltoid rupture as result of previous surgery; and axillary nerve palsy.

Latissimus Dorsi Transfer: Surgical Technique

Patient Positioning

The patient is maintained with a vacuum mattress in lateral decubitus position, without the classic dorsal tilt of 30°. This prevents the posterior deltoid to be too close to the humerus and remaining cuff, thus reducing the space where to isolate the axillary nerve and create the tunnel for the graft passage. A long-sized vacuum mattress allows to firmly fix the patient's head and neck. Gel pads are placed under the bone prominences in order to prevent skin ulcers or nerve palsies. The upper limb and shoulder girdle are draped, thus delimiting the operating field that extends to the lateral and posterior chest wall. A limb positioner (Spider; Smith & Nephew, Andover, MA, USA) is placed and secured to the opposite edge of the operating table and covered with sterile drapes. If a specific arm-holder is unavailable, a long-arm thigh holder can be used. This holder is used to support the forearm during tendon harvesting, with the arm abducted and elbow flexed at 90°, while standard longitudinal arm traction device is used during arthroscopy. When associated subscapularis repair is performed or if surgeon is not familiar with the lateral decubitus position, a modified beach-chair can be used. The pelvis and trunk are rotated by 30° toward the unaffected side. A protective gel pad supports under the gluteal region to prevent compression of the sciatic nerve.

Step-by-Step Procedure

Step #1: Diagnostic Arthroscopy and Subacromial Space Preparation

Preparation of the subacromial space is the first step. Tendon is harvested successively to avoid loss of irrigation fluid through the surgical incision, which can cause turbulence and impair quality of the arthroscopic view. As the rotator cuff has a massive tear, the scope can be directly introduced into the subacromial space through a posterior-lateral portal, and there is no need for a standard posterior portal.

Diagnostic arthroscopy is started by assessing the extent of the posterior-superior rotator cuff tear and the status of the subscapularis tendon; a large tear of this tendon should be repaired first. Remaining posterior cuff can be also re-tensioned by partial repair. The greater tuberosity is superficially debrided with a shaver, so as not to violate the cortical bone, to promote bone healing of the transferred tendon. We do not perform acromioplasty because the coracoacromial arch is an important restrain against the cranial migration of the humeral head, and its violation might worsen shoulder function, also compared to the initial condition.

If the long head of the biceps (LHB) is still intact but unstable or partially torn, tenotomy or tenodesis (less frequently) of this tendon should be performed as it might be a source of postoperative pain. Cortical bone of the bicipital groove is denser than that of the greater tuberosity and can be an optimal site for anchors placement during graft fixation. To this purpose, LHB biceps tenotomy or tenodesis should be performed at the distal part of the groove, so leaving the proximal part available for anchor placement.

Step #2: Capsular Release

Capsular release is essential to re-centering of the humeral head (Fig. 42.3). The goal of capsular release is to reproduce the so-called Gothic arch (the inferior profile of the scapular neck and the inferior profile of humeral neck appear in continuity on A-P radiographic view). The first step of the capsular release consists of detachment of the coracohumeral ligament (CHL). The ligament is ablated at its insertion on the base of the coracoid process with a radiofrequency device from an anterior portal. Next step is the release of the middle and inferior glenohumeral ligaments right off from their attachment at the glenoid rim, thus exposing the fibers of the subscapularis muscle. The axillary nerve is at risk only at the most distal part of the capsule, below the subscapularis muscle. At this site we have to be extremely cautious with the radiofrequency device; suction is connected to the device and tissue resection is carried out intermittently, so the fluids do not reach such high temperatures that might damage the nerve. Moreover, the tip of the device must be directed to the glenoid in order to avoid direct contact with the nerve. Progression of release from the anterior portal toward the

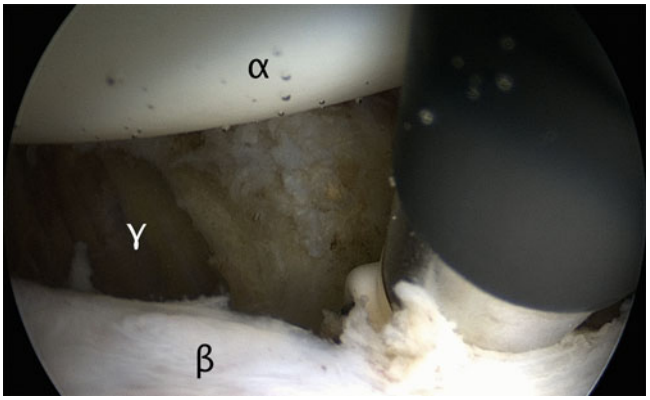

Fig. 42.3 Capsular release (α humeral head, β glenoid labrum, γ axillary pouch) (© Enrico Gervasi MD reproduced with permission)

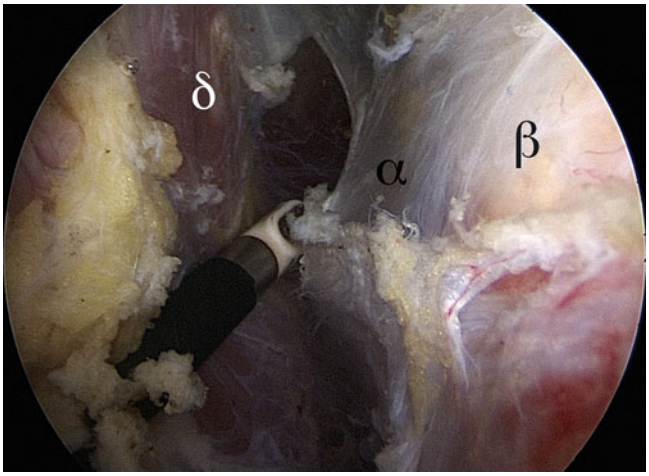

Fig. 42.4 Release of the reflection of the deltoid fascia (α [alpha], fascia; β [beta], teres minor; δ [delta], deltoid) (© Enrico Gervasi MD reproduced with permission)

axillary pouch is limited by the convexity and gliding backward of the humeral head, because of tightness of the posterior capsule. For this reason, the scope is shifted to the anterior portal, and release is completed from the posterior-lateral portal by sectioning the posterior capsule right off from its insertion at the glenoid rim. The suprascapular nerve runs1 cm medial to the posterior glenoid and is at risk during this step. Connecting the anterior and posterior releases completes the procedure.

Step #3: Deltoid Fascia Release

At this point, adequate space between the deltoid and the humeral head has to be created for graft passage. The deltoid is restrained to the humerus by its deep fascia. The release of the continuity of the deep deltoid fascia, either proximally from the acromion or distally by opening it at the level of its distal reflection, allows the muscle belly moving away from the humerus in the manner of a parachute. The axillary nerve runs just below the reflection of the deltoid fascia. It comes from the anterior aspect of the shoulder, emerges posteriorly through the quadrilateral space giving a branch to the teres minor, and moves laterally to reach the deltoid with several branches. Release of the fascia reflection allows the direct vision of the axillary nerve and its branches and facilitates the creation of a wide tunnel for the graft passage, thus preventing the muscle belly of the LD hinders the axillary nerve, as occurs in canalicular syndromes (Fig. 42.4). This is the last preliminary arthroscopic step, since extravasation of fluids, no longer retained by the fascia and bursa, quickly infiltrates soft tissues at the axilla.

Step #4: Surgical Exposure of Latissimus Dorsi

LD harvesting can be performed, according to the authors' preferred techniques, with an "all-endoscopic" or mini-open approach.

The operating table is placed in a Trendelenburg position to facilitate access to the surgical field and its lighting. The incision at the axilla is centered along the Langer's lines in order to avoid retracting skin scars. The incision crosses the posterior pillar of the axilla and the cutaneous projection of the lower edge of the posterior deltoid then runs distally along the contour of LD. This incision allows an adequate exposure of the thoracodorsal nerve and its pedicle anteriorly, the LD tendon and its insertion on the humerus anterior-superiorly, the muscle belly of LD inferiorly, and the posterior-inferior edge of the deltoid posterior-superiorly, where the tunnel for the graft passage to the subacromial space has to be created (Fig. 42.5). This approach, at the beginning intended for the "all-endoscopic" technique, differs from the extended incisions proposed by other authors in the past [6, 21, 22].

The "all-endoscopic" technique starts with a 6 cm incision, enough for the passage of endoscopic dedicated instruments: a large diameter scope fitted into lighting retractors, long scissors, and specific dissectors. These instruments are typically used in reconstructive plastic surgery procedures, such as breast reconstructions. During those procedures, retraction is obtained with devices holding the endoscope and expanders. In addition, short additional incisions were occasionally used to lift the teguments of the very peripheral areas.

The peculiarity of the "all-endoscopic" technique of LD harvesting we have developed and applied since 2003 is that the procedure is carried out without distension, neither with fluids nor gas, but only by means of mechanical retraction. We then moved on to a tissue-sparing open procedure since the difficulties of endoscopic dissection are not balanced by the small advantage, limited to better cosmesis. Moreover, scar is located in a hidden area, and patients undergoing this surgery are not usually too attentive to aesthetics.

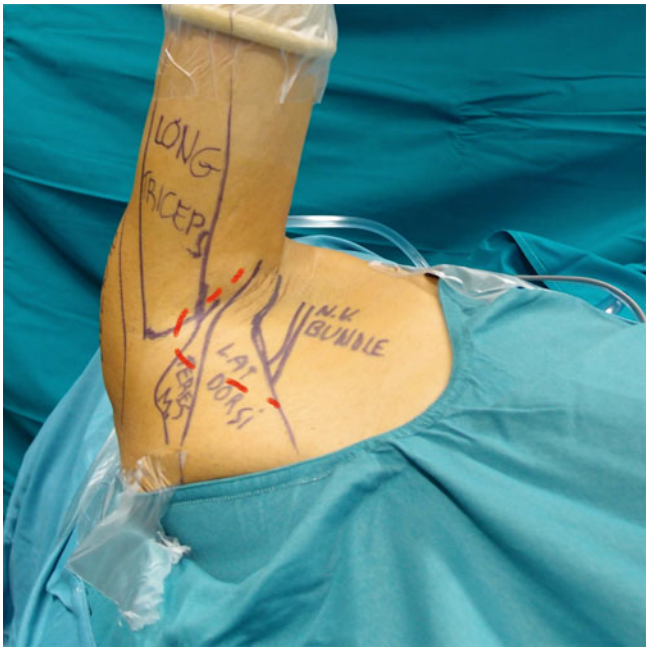

Fig. 42.5 Skin incision at the axilla. The red line shows the path of the skin incision, going close to the "pivot" anatomic structures. Landmarks for the humeral insertion of latissimus dorsi, neurovascular pedicle, long head of the triceps, and the quadrangular lateral axillary foramen are also shown (© Enrico Gervasi MD reproduced with permission)

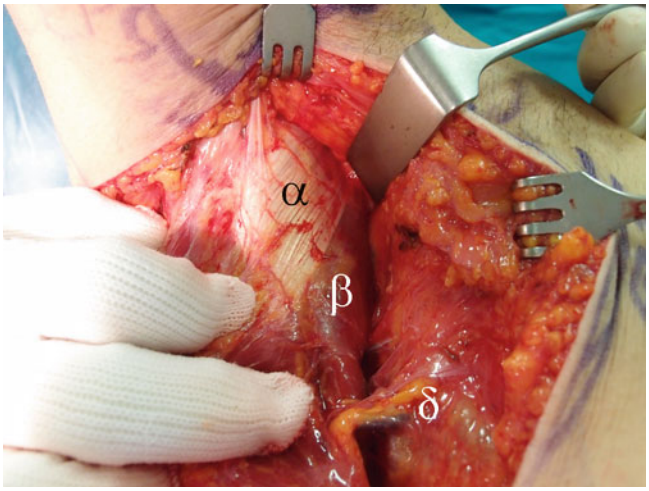

Fig. 42.6 Anatomic relationship between latissimus dorsi tendon and teres major muscle (α [alpha], latissimus dorsi tendon; β [beta], muscle fibers of the teres major; δ [delta], thoracodorsal neurovascular pedicle) (© Enrico Gervasi MD reproduced with permission)

After the incision, the skin and subcutaneous layer are lifted away from the underlying muscular fascia with retractors or traction sutures. LD is the most anterior musculotendinous structure of the posterior pillar of the axilla, so it cannot be confused with other muscles. The incision falls just distal to the LD tendon. The tendon has a ribbonlike appearance and ranges from 2 to 5 cm in width and 3–7 cm in length. Once reached, it can be released from surrounding adipose tissue by rubbing on it with gauze pads. Muscular fascia should not be violated to make easier the cleavage between LD and TM. Identification of these two musculotendinous units can be made at two levels: the tendinous and muscular part. The axillary nerve is superior to the TM tendon. The brachial plexus is deep and anterior, and the radial nerve is distal to the LD tendon. The proximal edge of the LD tendon must be freed with sharp dissection by a dense fibrous band, which belongs to the extensor fascia of the arm and runs perpendicular to the tendon [23]. The distal edge of the tendon is free from muscle fibers, and those going beyond the distal part of the LD tendon belong to TM (Fig. 42.6). The TM muscle can reach the humerus through a short tendon (equal or less than 2 cm), independent or confluent with that of the LD with a variable amount of fibers. In the latter case, the isolation of the LD tendon might be more demanding. An elevator can help remove muscle fibers of TM off from the LD tendon.

The separation of LD and TM muscle bellies takes place a few centimeters distal to the LD tendon. At this level there is an adipose raphe. Removal of this tissue allows to identify a clear cleavage between the two muscles.

We recommend detaching the LD tendon only after complete tendon and muscle isolation, so as to keep adequate tension during tendon isolation and release.

Step #5: Identification of the Thoracodorsal Pedicle

Operating table is now placed parallel to the floor. At the anterior part of the operating field, we look for the neurovascular thoracodorsal pedicle. It comes from the front and reaches the muscle belly at about 10 cm from its insertion to the humerus (8.5–14 cm), hidden into the adipose tissue for the whole path (Fig. 42.7). The pedicle more frequently consists of a double trunk, sometimes single and rarely triple. Its isolation increases mobility of the graft. Care must be taken during this surgical step to avoid damage to the innervation of TM, coming from the lower subscapular nerve.

Step #6: Latissimus Dorsi Distal Release

The operating table is now placed in anti-Trendelenburg position to facilitate the access to the surgical field and its lighting. Long valve retractors are used. The LD is intimately connected to the lower corner of the scapula and often with the serratus anterior by dense connective fibrous tissue. Both these muscles act on the scapular blade as a paddle, getting it attached to the chest. The more the posterior side of LD is approaching the lower corner of the scapula, the greater is the thickness of the fascia, which must be cut to gain extensibility of the graft. Anterior dissection is carried out by taking care of the subcutaneous adipose layer on the superficial aspect and the serratus anterior and the chest wall on the deep surface. Some vessels run between the LD and

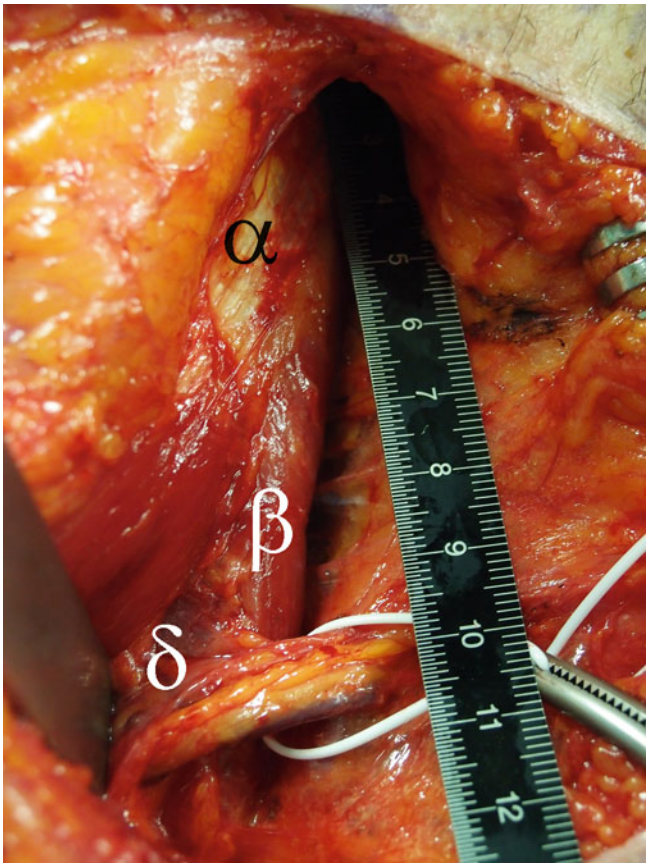

Fig. 42.7 Thoracodorsal neurovascular pedicle (α [alpha], latissimus dorsi tendon; β [beta], teres major; δ [delta], thoracodorsal neurovascular pedicle) (© Enrico Gervasi MD reproduced with permission)

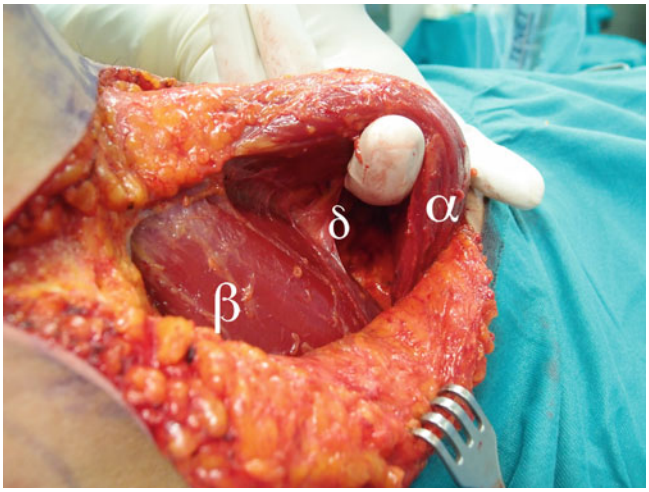

Fig. 42.8 Release of latissimus dorsi distal to the neurovascular pedicle (posterior view) (α [alpha], latissimus dorsi; β [beta], teres major; δ [delta], thoracodorsal neurovascular pedicle) (© Enrico Gervasi MD reproduced with permission)

subcutaneous tissue which should be coagulated in order to prevent a postoperative hematoma. A venous plexus is visible at the deep surface, close to the chest and serratus anterior. Release is complete if the surgeon's hand can pass from back to front under the deep surface of LD distally to the thoracodorsal pedicle (Fig. 42.8).

Step #7: Proximal Tendon Detachment

The humerus is placed in abduction and full internal rotation to expose the insertion of the LD tendon. Abduction should not exceed 90°; otherwise the humeral head, slipping distally, might engage the tendon, thus limiting the procedure.

A blunt curve Hohmann retractor is placed on the anterior aspect of the humeral shaft, between the pectoralis major and LD tendons. A valve retractor placed laterally protects the radial nerve, which runs 2 cm distal to the inferior margin of the LD tendon and is not visible. The Hohmann or a valve retractor protects the brachial plexus. The tendon is sharply detached, proceeding from its distal to proximal end (Fig. 42.9). Care must be taken to the contiguity of the axillary nerve and posterior circumflex vessels.

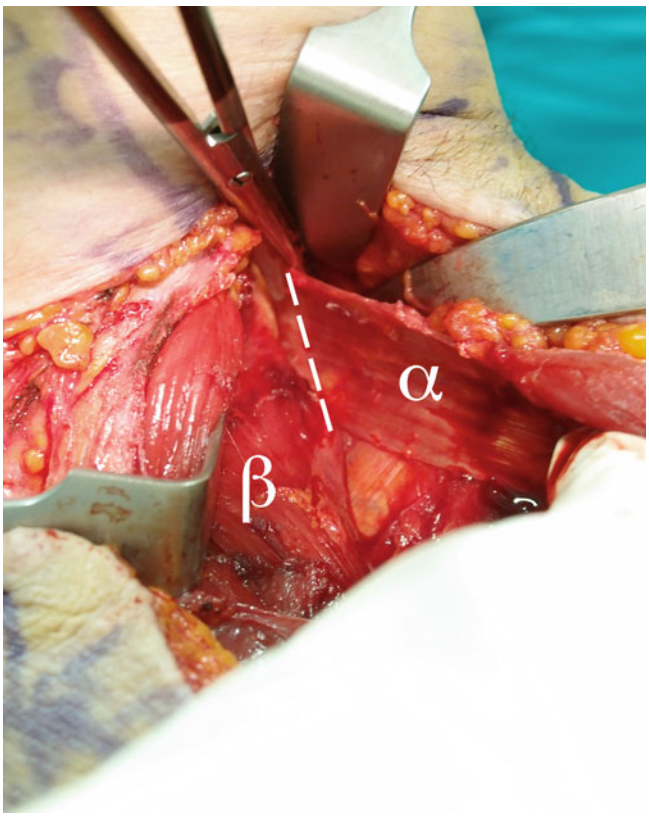

Fig. 42.9 Detachment of latissimus dorsi tendon (α [alpha], latissimus dorsi tendon retracted anteriorly; the dashed line shows the tendon humeral insertion, where it is has to be divided; β [beta], teres major retracted posteriorly) (© Enrico Gervasi MD reproduced with permission)

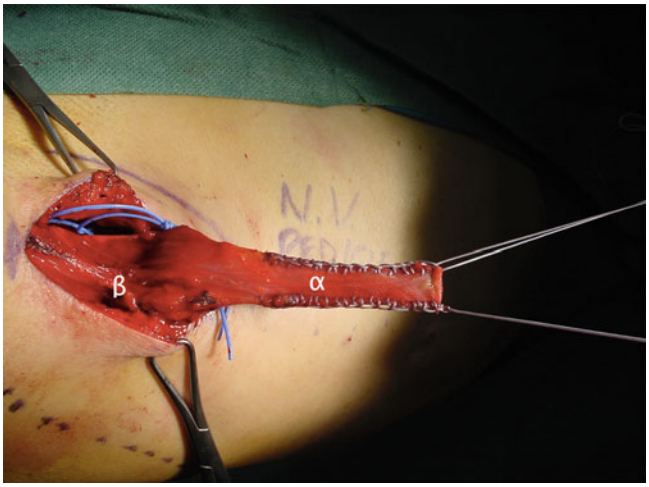

Fig. 42.10 Latissimus dorsi harvested (α [alpha], latissimus dorsi tendon with anterior sutures; β [beta], thoracodorsal pedicle entering the muscle belly of latissimus dorsi) (© Enrico Gervasi MD reproduced with permission)

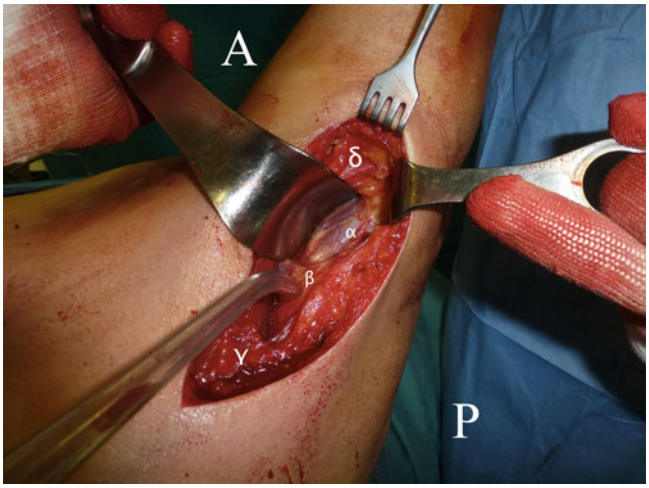

Fig. 42.11 Tunnel to the subacromial space (α [alpha], reflection of the deltoid fascia; β [beta], circumflex bundle; γ [gamma], teres major; δ [delta], triceps; *A* anterior, *P* posterior) (© Enrico Gervasi MD reproduced with permission)

Step #8: Tendon Preparation

As tendon fibers are longitudinally oriented, traction sutures must be placed along the same direction to avoid tendon splitting. Two #2 high-strength braided sutures of different colors are placed in a Krakow fashion along either side of the tendon (Fig. 42.10). The two free ends of two sutures are at the free edge of the tendon. The other two sutures, placed about 3 cm from the tendon free ending, can be used to increase tendon fixation or drive its rear part over the greater tuberosity. Care must be taken during graft passage to pull simultaneously sutures on both sides of the tendon. At the beginning of our experience, we usually placed a transverse suture across the top of the tendon to prevent the risk of longitudinal splitting. Keeping the tendon wet reduces the risk of weakness due to dehydration.

Step #9: Tunnel Preparation for Graft Passage

The operating table is again placed in Trendelenburg position. The arm should not be abducted more than 90° to prevent the humeral head stretching the structures going to be identified. The key structure of this step is the long head of the triceps (LHT) muscle and tendon. The LHT tendon inserts at the infraglenoid tubercle and can be easily identified when tensioned by elbow flexion. Muscle fibers of LHT are visible at both sides of the tendon. The lower edge of the posterior deltoid rises with arm abduction, thus not being visible on the operative field. The structure at risk during this step is the axillary nerve, which passes from anterior to posterior, behind the very base of the LHT (Fig. 42.11). The space to pass the graft into the subacromial space is just lateral to the nerve, between the deltoid and teres minor. The tendon graft passes deep to the course of the posterior branch of the axillary nerve and the superior-lateral brachial cutaneous nerve [24].

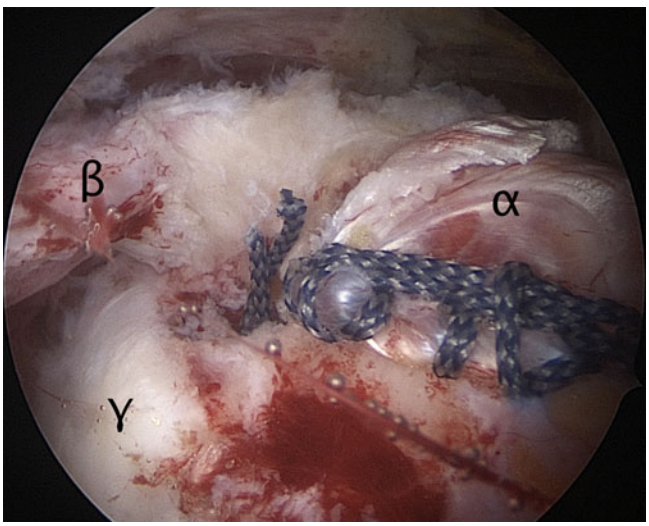

Fig. 42.12 Latissimus dorsi and subscapularis relationship (α [alpha], latissimus dorsi tendon; β [beta], subscapularis tendon; γ [gamma], humeral head) (© Enrico Gervasi MD reproduced with permission)

Step #10: Graft Passage

Graft passage is performed by endoscopically assisted technique. The goal of this step is to pass the graft into the subacromial space and fix it into the bicipital groove (Fig. 42.12). This is facilitated by introducing a shuttling device through an anterior portal. This portal should be as high as possible, close to the anterior aspect of the acromion; this allows the shuttling device to overcome the convexity of the humeral head, and the graft to slide along a straight line, thus offering

less resistance to its passage. The scope is introduced thorough the posterior-lateral portal.

In order to ease a surgical procedure otherwise demanding, the shuttling device should have some specific features, as follows:

- Its tip is smooth to avoid damage to the axillary nerve and catching into the muscle belly of the deltoid.
- It is rigid to drive the graft where the surgeon wants.
- It is cannulated, to shuttle the sutures coming from the graft.
- It has a system for locking the sutures.
- It has two parallel channels to avoid inadvertent graft rotation during passage.

The inflow is closed until the LD muscle engages the tunnel between the deltoid and teres minor. This prevents loss of irrigation fluid through surgical incision, thus creating turbulence and impairing arthroscopic view.

Step #11: Graft Fixation

The graft is driven in the subacromial space up to the bicipital groove. The surgeon chooses the sequence of sutures fixation. We prefer to fix the lateral pair of sutures first because medial fixation can crimp the lateral edge of the tendon, thus making its fixation more difficult (Fig. 42.13).

When the graft slides medially or laterally to the greater tuberosity, rear sutures can be used to achieve correct placement. In this case suture anchors are introduced through a lateral portal, close to the lateral edge of the acromion. Internal and external arm rotations confirm the stability of the graft on the greater tuberosity.

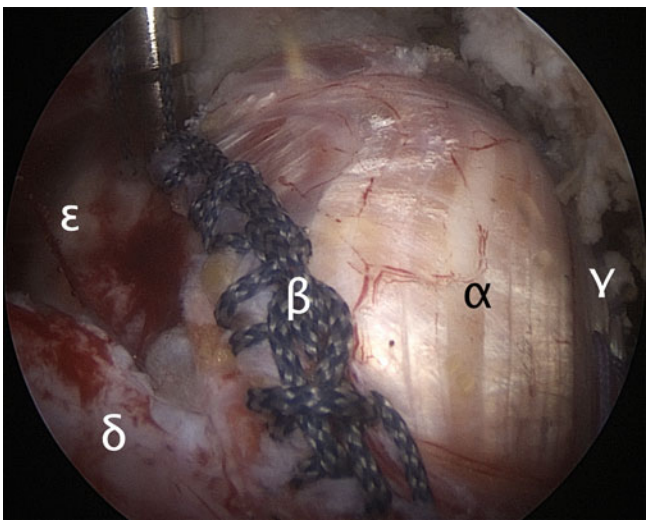

Fig. 42.13 Latissimus dorsi fixation: medial anchor (second one) (α [alpha], latissimus dorsi tendon; β [beta], medial-row sutures; γ [gamma], lateral-row sutures; δ [delta], teres minor tendon; ε [epsilon], humeral head) (© Enrico Gervasi MD reproduced with permission)

Step #12: Wound Closure

Two large drains are placed in the lower part of the surgical site. After skin closure, a compressive dressing is applied on the chest, with upholstered pads and elastic knit.

Postoperative Care

Postoperative protection of LDT follows the general rules used for repair of massive rotator cuff tears. As postoperative pain and apprehension to use anatomic structures involved in the surgical procedure make postoperative training very difficult, patients should be educated to perform analytic exercises for shoulder girdle muscles before surgery.

During the first postoperative period, active motion is allowed under strict supervision. Sling with 15° of external rotation or an abduction pillow is continued for 6 weeks. Patients are allowed to perform few pendulum exercises during this period to prevent scarring between teres minor and deltoid. Elbow flexion and hand exercises are encouraged, as well as rehabilitation program for the scapulothoracic articulation, trunk, and lower limbs. Core rehabilitation not involving the shoulder girdle can facilitate early functional recovery. Water rehabilitation (hot pool) is helpful during this phase as well as during the period of functional recovery.

Active shoulder motion is allowed gradually. Shoulder exercises should be performed with both limbs to use the "mirror" effect, thus favoring muscle trophism and recovery of motor engram. The patient starts the exercises lying on the uninjured side, the back tilted of 30° backward, in order to place the glenoid parallel to the ground and thereby reducing shear forces acting on the humeral head. The elbow is maintained flexion at first, so reducing the lever arm. The gradual erection of the trunk gets ready to perform exercises in a full upright position. Rehabilitation takes many motor tasks from the activities of daily living to motivate the patient.

Functional recovery takes place ranging from a few months to 1 year after surgery. The transposed LD provides ability to work actively, as shown in the transposition procedures carried out for obstetric palsies, in which LD is transposed around the diaphyseal cylinder.

Literature Review

Namdari et al. [25] analyzed, in a systematic review, the outcomes of ten cohort studies on patients who underwent LDT for irreparable rotator cuff tears. Mean adjusted Constant score reported in six studies improved from 45.9 preoperatively to 73.2 postoperatively. Mean pain score reported in six studies improved from 4.8 preoperatively

to 12.1 postoperatively on a 15-point scale. Regarding the functional assessment reported in all studies, active forward elevation improved from 101.9° preoperatively to 137.4° postoperatively, and active external rotation improved from a frequency-weighted mean of 16.8–26.7°. Active abduction reported in five studies improved from 91.4° to 130.7° postoperatively.

Irlenbusch et al. [26] showed in an electromyography study that activity pattern of LD in the operated shoulder (the healthy side used as reference standard) correlated with the Constant score. Hence, functional improvement is due to not only an interposition or tenodesis effect but also to an active muscle effect. Activity of the transposed LD lowers the humeral head, thus improving its centering into the glenoid cavity and creating better preconditions for the extrinsic shoulder muscles.

Miniaci et al. [22] showed significant clinical improvement after rotator cuff revision with LDT. Warner and Parsons [27] argued that the LDT in revision surgery of rotator cuff provides poorer outcome than primary LDT. On the contrary, Costouros et al. [28] reported comparable improvement in pain relief and function following LDT in either primary or revision surgery.

Female patients with poor shoulder function and generalized muscle weakness prior to surgery and obese patients with heavy arms are more likely to have poor clinical outcome [29].

The role of integrity of the teres minor on clinical outcome of LDT is unclear [17]. Indeed, the presence of a teres minor tear is associated with loss of strength in external rotation. However, fatty infiltration of the muscle belly of teres minor rather than tendon tear seems to significantly influence the outcome of the LDT.

Integrity of the subscapularis muscle-tendon unit as prerequisite of LDT is debated. Codsi et al. [30] suggested that in presence of an irreparable subscapularis tear, LDT should be combined with a pectoralis muscle transfer. Although subscapularis repair is strongly recommended when possible, we experienced favorable, whenever unexpected, results in patients with associated subscapularis tears, even if large.

Comparative long-term radiographic studies showed the reduction of the acromiohumeral distance over time and progression of arthritic changes after LDT [21, 31–33]. Aoki et al. [21] observed progression of glenohumeral osteoarthritis in 41 % of their cases, and Gerber et al. [32] observed the same findings in 30 % of their cases. Moursy et al. [33] confirmed these observations, with a progression rate of osteoarthritis in 29 % of the cases, regardless of surgical technique that had been used. Gerhardt et al. [19] found that despite an initial increase in acromiohumeral distance, superior migration of the humeral head with concomitant cuff arthropathy was evident at 5-year follow-up.

Deltoid detachment is one of the most frequent complications in LDT [34]. Aoki et al. [21] showed that graft rupture was more frequent in revision surgery (44 % of the cases) than in primary surgery (17 % of the cases). Other complications reported in the literature are sensitive injuries of the ulnar nerve, brachial plexus, or axillary nerve, recovering spontaneously within 6 months [32], infections, and reflex sympathetic dystrophy [35].

Author's Experience

We evaluated our first cohort of 20 patients who underwent arthroscopically assisted LDT with at least 3 years of follow-up (unpublished data). Strength recovery was more evident in elevation than in external rotation. The postoperative centering of the humeral head into the glenoid quantified on postoperative radiographs was favorable to functional recovery, even if partial. In our opinion, humeral centering was greatly favored by the extensive capsulo-ligamentous release. Radiographs also showed in some cases a dimple on the tuberosity, as it would have been carved by the compressive effect of the transposed tendon.

Results were less favorable when LDT was performed as revision of failed rotator cuff repair, especially after large acromioplasty, as already reported in the literature on revision rotator cuff repair [22, 27].

Integrity of the subscapularis was crucial although some patients with an irreparable subscapularis tear equally benefited from the procedure. Arm weight was a negative prognostic factor.

We did not observe major complications. More recently, we had two neurologic complications involving the axillary nerve: one primary surgical injury and one nervous damage subsequent to infection and debridement occurred in a patient affected by acne. Few patients developed blood or serous effusion at the site of muscle harvesting. In those cases, superficial tissues did not adhere to the underlying muscle layer, thus leading to a "third space" formation. Treatment consisted of percutaneous drainage in the most distal part of the effusion. However, care to coagulate vessels crossing distally from muscle to subcutaneous makes negligible this complication.

Summary

In conclusion, we consider the arthroscopically assisted LDT as the procedure of choice for functionally disabling and irreparable posterior-superior rotator cuff tears. However, the basal tension of LD is insufficient to reproduce the whole strength of the posterior-superior cuff. Graft fixation to the most anterior part of the greater tuberosity improves elevation more than external rotation.

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Arthroscopic Treatment Options for Glenohumeral Osteoarthritis

43

Werner Anderl, Brenda Laky, and Philipp R. Heuberer

Introduction

Osteoarthritis (OA) of the shoulder is a chronic, progressive, and multifactorial disease characterized by degenerative and inflammatory processes affecting the glenohumeral joint. The incidence of primary OA has been reported as approximately 5 % of patients with shoulder complaints [1]. Although OA in the shoulder is less common than OA of the knee or hip, OA can cause severe pain and dysfunction of the shoulder. Pathologic changes in shoulder OA involve the progressive breakdown of the articular cartilage within the glenohumeral joint starting with narrowing of the joint space and fibrillation of the surface, followed by osteochondral lesions, osteophyte formations, labrum degradation, capsular tightness, and inflammation. As OA may also affect supporting structures such as muscles, tendons, and ligaments, shoulder joint degeneration can also be linked to secondary causes such as rotator cuff tendon tears [2, 3], shoulder instability especially in young patients [4, 5], and trauma [6].

Indications for Shoulder Arthroscopy

Clinically, OA is diagnosed using patient's history, physical examination, and plain radiographs to detect loss of glenohumeral joint space and osteophytes (Fig. 43.1). It has also been reported that no evidence of cartilage loss at preoperative plain radiographs or with magnetic resonance images (MRIs) was detected, whereas small cartilage lesions could be observed with arthroscopy [1]. Incidental findings of cartilage lesions during arthroscopic treatment for rotator cuff tear, impingement, and glenohumeral instability are common [1]. Arthroscopy is not only performed to evaluate the glenohumeral joint to stage disease but also to treat the arthritic glenohumeral joint.

Since there is no direct cure for OA, treatments mainly focus on pain relief and maintaining joint mobility. In general shoulder arthroplasty, following unsuccessful nonoperative treatment, is the therapy of choice. Nonetheless, arthroscopic surgery might be a beneficial treatment option in young active high demanding patients with special regard to longevity risks of joint replacements and in elderly patients unfit for an open procedure and especially for all patients with the desire to avoid major surgery. As the major advantages of arthroscopic procedures are low complication rates, maintaining the subscapularis tendon intact and therefore immediate full active range of motion after surgery, the development of new techniques, instruments, and implants is advancing to expand the indications for arthroscopic shoulder surgery.

Arthroscopic Treatment Options to Relieve Symptoms

Debridement with Capsular Release and Osteophytectomy

Arthroscopic debridement of the shoulder is usually performed with a shaver to remove loose or injured cartilage tissue from the articular surface. This procedure is commonly combined with arthroscopic lavage to flush out inflammatory mediators. Such techniques may improve pain relief or recovery of the shoulder function for a short period; however, they are not useful to stop progression of the degenerative changes in the joint in the long term.

Diagnostic arthroscopy, as the first step in the standard setup at our clinic, is usually performed under scalenus

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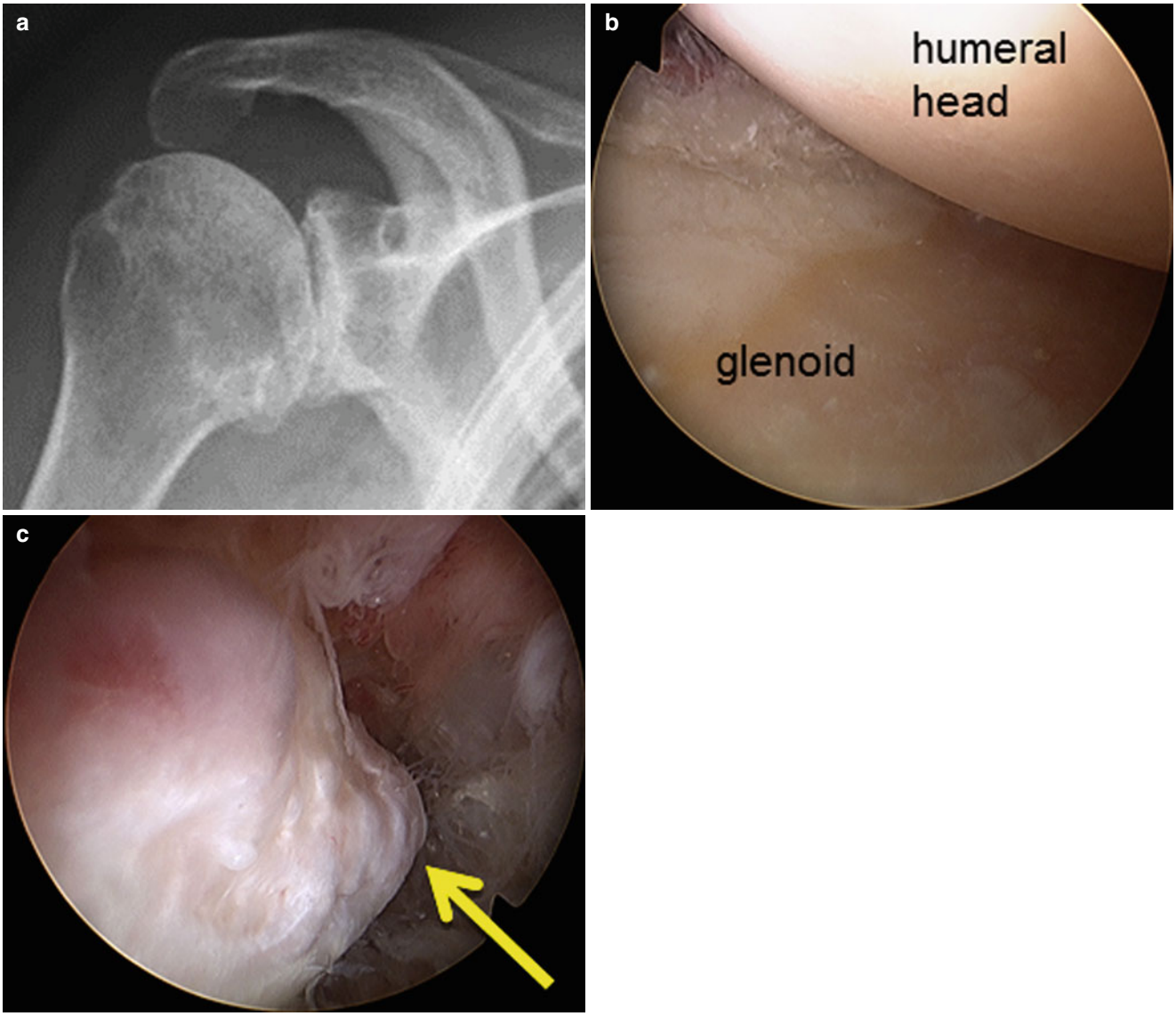

Fig. 43.1 Osteoarthritis of the right humeral head and glenoid of a 72-year-old female on a plain radiograph (**a**) and at arthroscopy (**b**, **c**). Arthroscopic view of an osteophyte (**c**, *arrow*)

anesthesia in the lateral decubitus position in double extension; a standard posterior portal is used to enter the glenohumeral joint. Firstly, diagnostic arthroscopy is performed to evaluate the cartilage, to detect and measure defects, to inspect rotator cuff tendons, and to prospect for osteophytes, capsular contractures, and thickened middle glenohumeral ligament.

At our institution, depending on patient's pathology, arthroscopic treatment includes the following procedures:

- Step #1: Tenodesis or tenotomy of the biceps tendon in case of a tendinopathy.
- Step #2: Capsular release starting at the rotator interval with an intra- and extra-articular release of the subscapularis tendon; thereafter the anterior capsule is divided to the 6 o'clock position. After changing the scope to the anterior portal, the capsule is released posteriorly.
- Step #3: If the inferior capsule is extensively ongrown to the osteophyte, the capsule is carefully dissected from the glenoid with a diathermy. During the partial detachment of the inferior capsule and exposure of the axillary nerve, a Wissinger rod is introduced through an extended posterior-inferior portal to protect the axillary nerve and accompanying vessels.
- Step #4: After capsular release, osteophytes are dissected and removed with a burr and/or a curved chisel, while the axillary nerve and accompany vessels remain protected by a Wissinger rod (Video 43.1).
- Step #5: Articular cartilage defects are debrided and loose bodies are removed.

To finalize the procedure, intraoperative radiographs are obtained to confirm proper removal of osteophytes.

First results after arthroscopic debridement were described by Ogilvie-Harris and Wiley, who reported satisfactory outcome after 3 years for two-thirds of patients with mild arthritis, but only for one-third of patients with moderate to severe arthritis [7]. A similar study by Weinstein et al. [8] showed that most patients (80 %) reported excellent or good results more than 2 years (average: 34 months) after arthroscopic debridement to treat mild OA. A retrospective study by Kerr et al. [9] reported worse outcome after arthroscopic debridement in patients with arthritis involving both humeral head and glenoid compared to unipolar glenohumeral cartilage lesions. Cameron et al. [10] investigated 2-year outcomes of patients with grade IV osteochondral lesions of the glenohumeral articular surface and found only sizes larger than 2 cm^2 related to pain and failure, but not location (glenoid, humeral head). They suggested for patients with a loss of motion of more than 15° to additionally perform arthroscopic capsular release. Weinstein et al. [8] have also shown that soft tissue pathologies are commonly detected in OA. The combined technique of debridement and capsular release has further been described by Richards and Burkhart [11]. Their preliminary data suggested good functional outcome and a symptom-free interval of 9 months. Results of arthroscopic debridement generally compare favorably to open techniques; however, for patients with large osteophytes, debridement may not be as successful as reported by a retrospective study of Van Thiel et al. [12]. Along with the presence of grade 4 bipolar joint disease and joint space of less than 2 mm, large osteophytes were detected as significant risk factors for failure after arthroscopic debridement and additional procedures such as capsular release, biceps tenodesis or tenotomy, microfracture, loose body removal, osteophyte resection, and subacromial decompression [12]. Our joint-preserving technique, also described by Millett and Gaskill [13], combines extensive glenohumeral debridement, capsular release with osteophyte removal from the humerus, and arthroscopic trans-capsular axillary nerve decompression. According to our clinical experiences (unpublished data), such a procedure may provide better outcome regarding pain relief and shoulder function than simple debridement alone. In summary, arthroscopic debridement with or without additional techniques such as capsule release and osteophyte removal may be an effective temporal treatment to delay progression of early disease; however, it is not recommendable to treat patients with severe OA and in the long term.

Arthroscopic Treatment Options for Articular Cartilage Defects

In the past, arthroscopic treatments were limited to short-term symptom relief procedures such as lavage, debridement, and abrasion of chondral lesions, whereas more recently new methods have been developed to regenerate, repair, or reconstruct cartilage defects and which may last longer.

Microfracture

Popularized in knee surgery [14], microfracture is a procedure creating small fractures into subchondral bone to generate a bone marrow-stimulated fibrocartilage response. Siebold et al. [15] first reported the microfracture technique for the shoulder joint. However, they introduced an open technique combining microfracture and periosteal flap. A few years later, Millett and coworkers [16] published 2-year outcomes of 30 patients treated with arthroscopic microfracture. They reported a 19 % failure rate for microfracture and a better clinical improvement in patients with smaller lesions of the humerus compared to patients with bipolar lesions. Another study investigating microfracture reported improvements regarding pain and shoulder function [17]. However, both studies have also indicated the need for further, especially long-term, studies. Our experiences with microfracture with younger patients (Fig. 43.2a) were similar to Millett et al. findings [16]. However, with patients older than 40 years (Fig. 43.2b), results were not as promising as the current literature (unpublished data).

Biologic Resurfacing

Biological approaches are other new treatment options to restore cartilage defects of the glenohumeral joint, whereas damages of the humeral head may be treated differently to those of the glenoid. Biologic treatment of cartilage injuries, as in the knee joint, comprises osteochondral autologous transfer (OATS), autologous chondrocyte implantation (ACI), or matrix-induced ACI (MACI). In open shoulder surgery, ACI has only been reported in a case report [18] and our clinical experiences with MACI (Fig. 43.3) comprise only three cases (unpublished data). Results of OATS have been reported by Scheibel et al. [19] in a small series of patients and showed a significant improvement of the Constant score, but progression of OA could not be delayed. Although the efficacy of ACI and MACI has been widely studied in the knee joint, and with new generations of scaffolds and matrices, an all-arthroscopic chondrocyte implantation is technically achievable [20, 21], yet an all-arthroscopic procedure in the glenohumeral joint has not been described.

While investigations regarding biologic arthroscopic cartilage restoration of the humeral head might be a long time coming, several all-arthroscopic glenoid resurfacing techniques have been published. Savoie et al. [22] published an arthroscopic glenoid resurfacing technique with a biologic

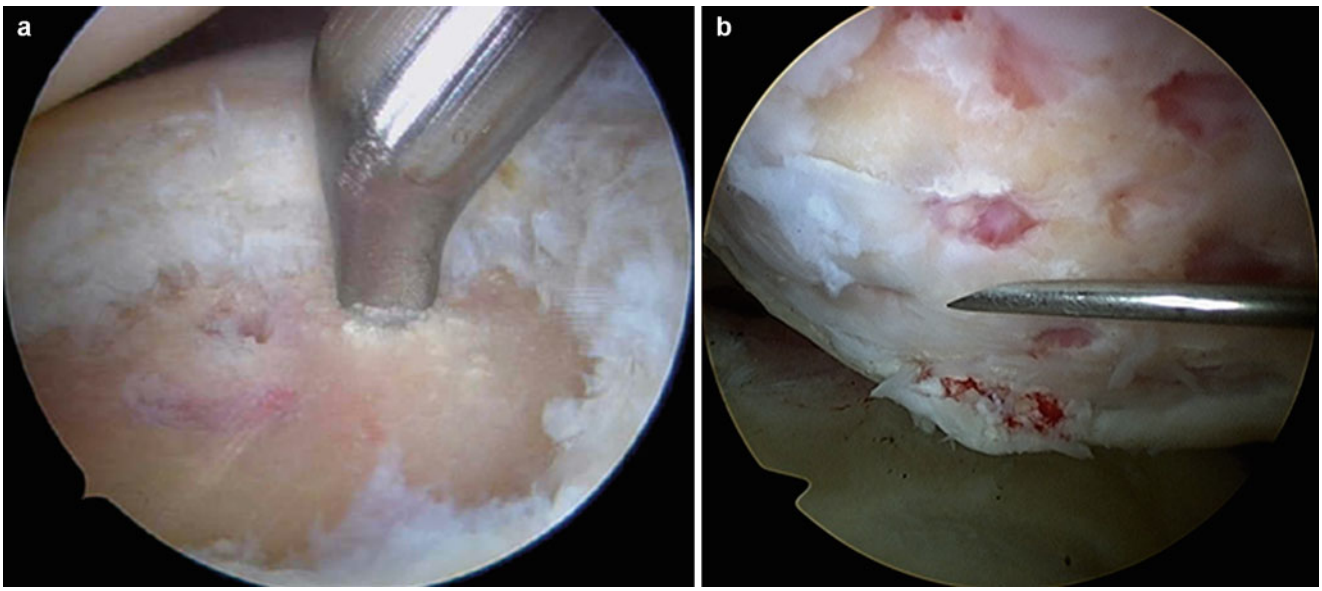

Fig. 43.2 Arthroscopic microfracture technique of a unipolar focal chondral defect of the glenoid of an 18-year-old female contact athlete (**a**). Arthroscopic view of the humeral head of a 55-year-old male patient showing a failed microfracture due to no fibrous cartilage response (**b**)

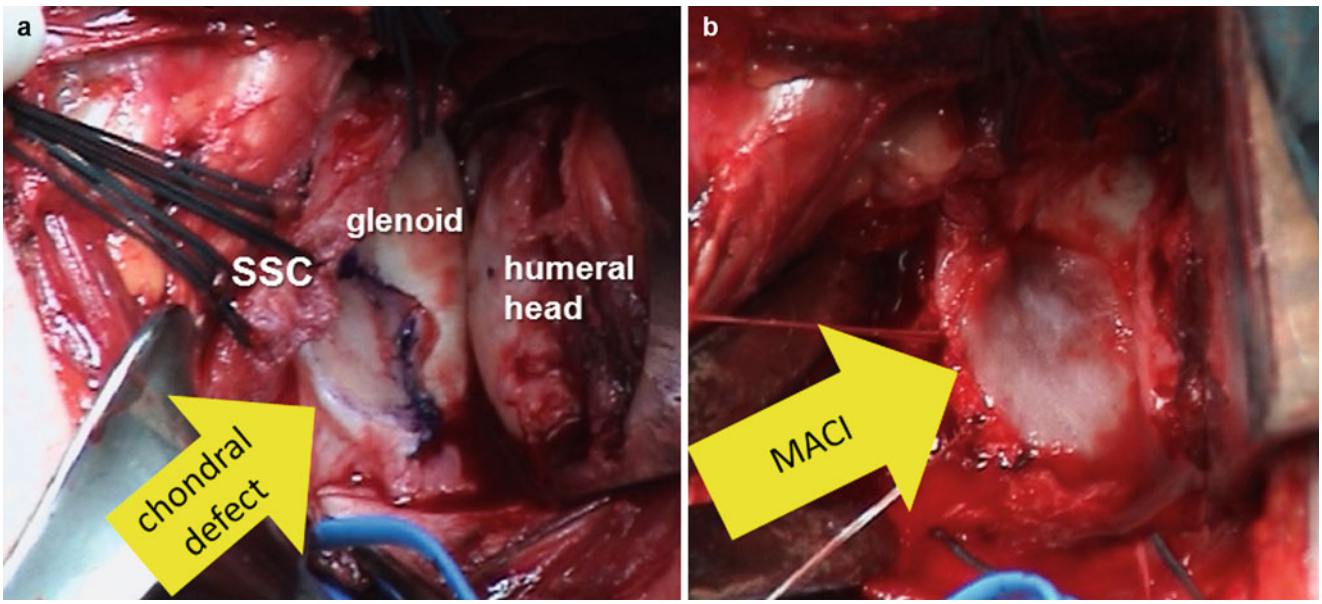

Fig. 43.3 Large chondral defect in the inferior part of the glenoid (**a**). Matrix-induced autologous chondrocyte implant (Hyalograft C, Fidia Advanced Biomaterials, Italy) (**b**)

patch (Restore; DePuy Orthopaedics, Warsaw, IN, USA) for young patients with severe glenohumeral arthritis showing an overall success rate of 75 % after 3–6 years. Their results were similar to a study by DeBeer et al. [23], who reported intermediate-term results after arthroscopic debridement in combination with an allograft human dermal matrix-based scaffold (GraftJacket; Wright Medical Technology, Inc. Arlington, TN, USA). Failure more than 2–4 years after surgery was detected in 9 of 32 patients (28 %) and included patients who were not satisfied with treatment or developed some kind of complications including transient axillary nerve palsy, foreign-body reaction to biological graft material, interlayer dissociation, mild chronic nonspecific synovitis, and post-traumatic contusion. Our experiences treating severe glenohumeral arthritis in young patients using a xenograft (porcine dermal collagen or "Zimmer Patch," formerly known as "Permacol"; Tissue Science Laboratories plc, Aldershot, Hampshire, UK) (Fig. 43.4) showed a failure rate

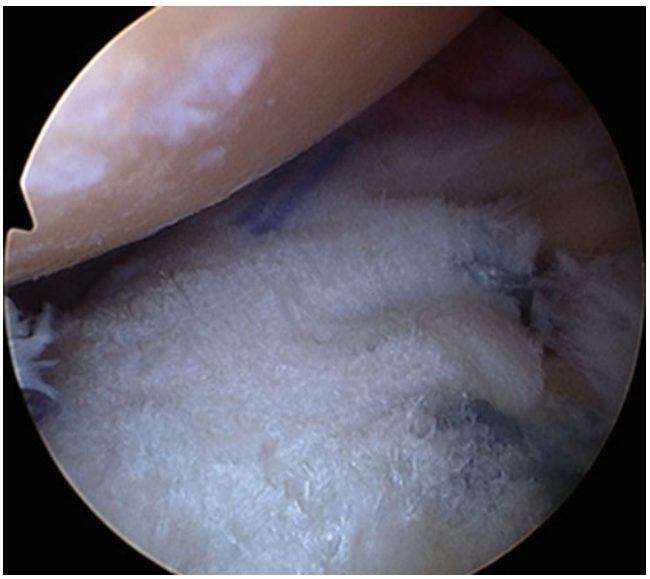

Fig. 43.4 Xenograft patch (porcine dermal collagen) in a severe osteoarthritic shoulder of a 38-year-old female patient

Fig. 43.5 Partial Eclipse prosthesis

of more than 80 % (unpublished data). However, such a high failure rate might be due to the fact that we used a nonhuman graft. Before such procedures can be recommended, further prospective investigations, especially with longer follow-up periods, are required.

Biologic Total Shoulder Resurfacing

Gobezie et al. [24] reported a novel all-arthroscopic biologic total shoulder resurfacing technique for patients with large focal defects and arthritis. Their idea was to replace the worn cartilage of the humeral head and of the glenoid by allografts taken from "healthy" humeral head and medial tibial condyle from a cadaver donor. Implantation was performed using instruments and techniques provided by Arthrex Inc. (Naples, FL, USA). All-arthroscopic biologic total shoulder resurfacing sounds very promising; however, there may be some limitations associated with allografts as some potential risks are known and due to legal issues in various countries. Furthermore, data from 1-month follow-up of only ten patients are not enough for a technique to be recommendable.

Partial Resurfacing of the Humeral Head

Another all-arthroscopic technique to resurface the humeral head to treat focal chondral or large osteochondral defects was developed at our institution. The Partial Eclipse™ prosthesis (Arthrex Inc., Naples, FL, USA) consists of a partial humeral head and a threaded stem component (Fig. 43.5) and was developed to maintain joint biomechanics and preserve the intact cartilage as well as the subscapularis tendon and therefore enables an early postoperative mobilization.

The insertion of the Partial Eclipse prosthesis follows an easy-to-implant technique (Fig. 43.6) in an all-arthroscopic fashion through the rotator interval. Following a standard shoulder arthroscopy with tenotomy or tenodesis of the long head of biceps, capsular release, and removal of osteophytes, if required, the cartilage defect is detected. With the shaver all soft tissue of the rotator interval is removed to create enough space for instrument and implant handling. After extension of the anterior skin incision up to 2.5 cm, the portal is widened using the forefinger. Then a rectangular drill guide is introduced, which is also used to measure the defect. The drill guide's center tip indicates the center of the implant. Where the other end of the drill guide laterally on the upper arm touches the skin, a 2.0-cm-long skin incision is done and soft tissue is dissected bluntly until bone can be reached. To protect the axillary nerve, all further instrumentation is performed only through tissue protectors. With the drill guide, a transhumeral canal is drilled from lateral through the humeral head for retrograde reaming. Therefore, a reamer is introduced through the rotator interval and connected to a threaded pin in the transhumeral canal. Having prepared the subchondral bone bed, through a shuttle system, the correct sized implant is then brought in. Under retrograde screwing through the transhumeral canal, the implant is fit into the humeral head. The ideal final implant position is slightly beneath the intact cartilage surface.

Results regarding focal chondral defects of our first case series (unpublished data) suggest comparable outcomes

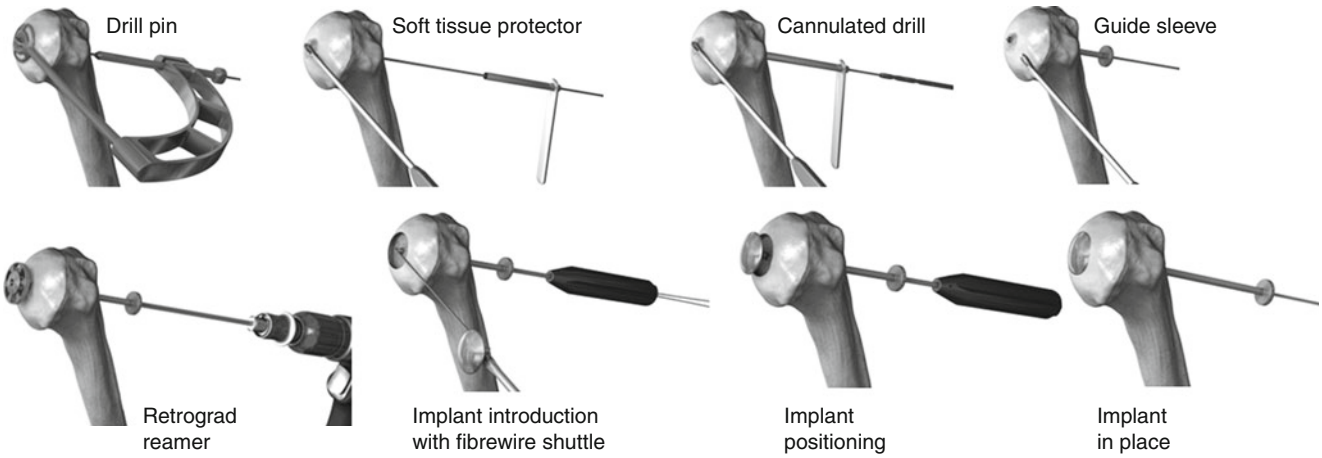

Fig. 43.6 Arthroscopic insertion technique of the Partial Eclipse prosthesis

(Fig. 43.7) to open techniques for partial humeral head resurfacing [25].

Furthermore, our first findings lead to the development of larger implant sizes to treat patients with even larger chondral defects and glenohumeral joint arthritis. With such large implants (Fig. 43.8), at least two-thirds of the humeral head can be covered. A trial to evaluate 35-mm Partial Eclipse prosthesis is already running. Preliminary results are promising and indicate better joint biomechanics compared to hemiarthroplasty. However, the major benefits of an all-arthroscopic partial humeral head resurfacing are immediate mobilization and the full range of motion. An algorithm for our current approach to treat glenohumeral cartilage defects is presented in Fig. 43.9.

Although early investigations present encouraging results, continued evaluations and further implant developments are needed. For the future a transhumeral reamer for a ream-and-run procedure [26] and an arthroscopic partial glenoid resurfacing with an inlay technique to restore osteoarthritic changes of the glenoid is currently under development.

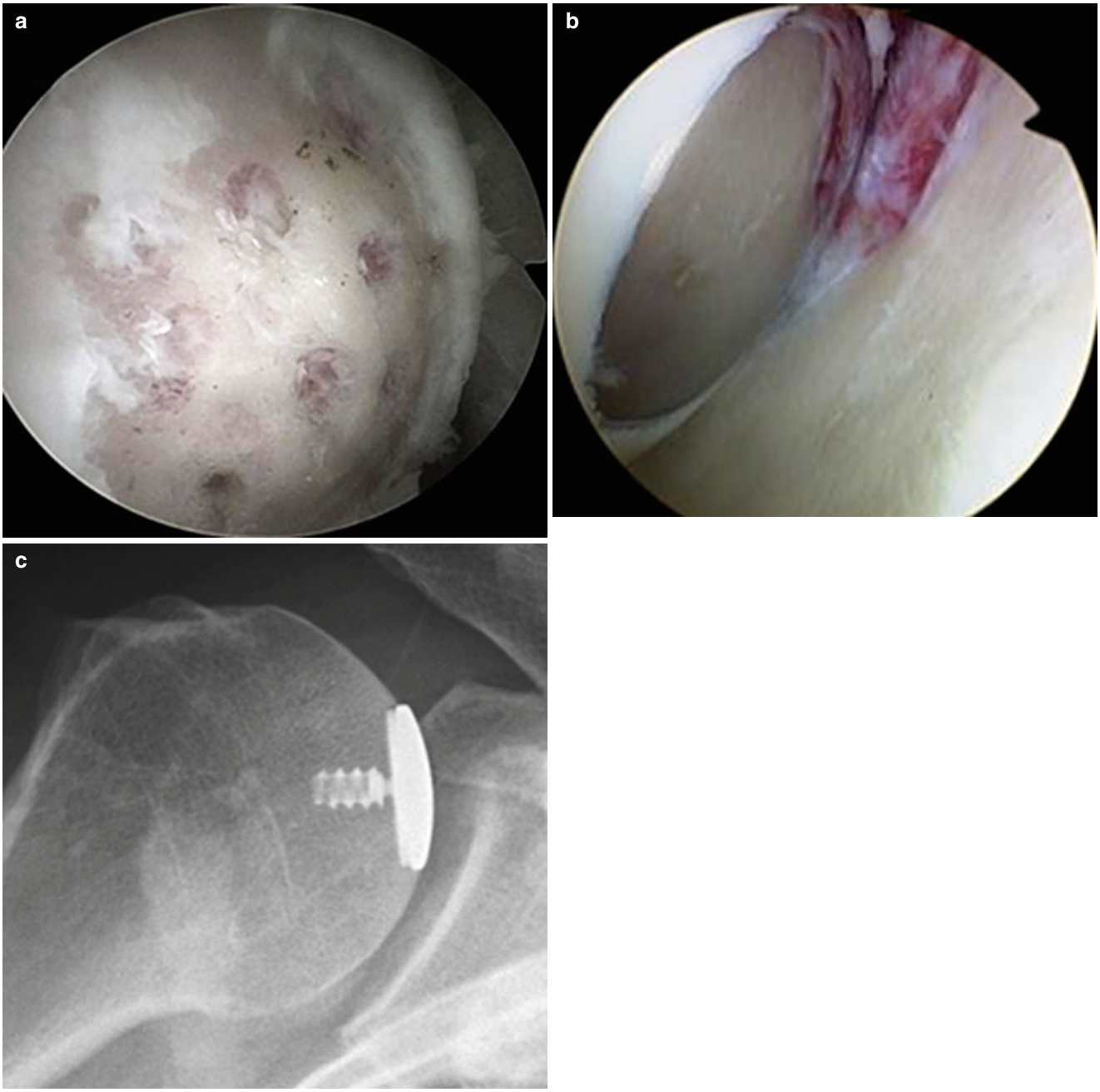

Fig. 43.7 Arthroscopic view of a failed microfracture 1 year postoperative of a 48-year-old male patient (**a**). Arthroscopic view of the implanted Partial Eclipse prosthesis (**b**). Postoperative plain, anteroposterior radiographs after 2 years demonstrating excellent osseointegration and no radiolucent lines at the implant-bone interface after all-arthroscopic partial shoulder resurfacing (**c**)

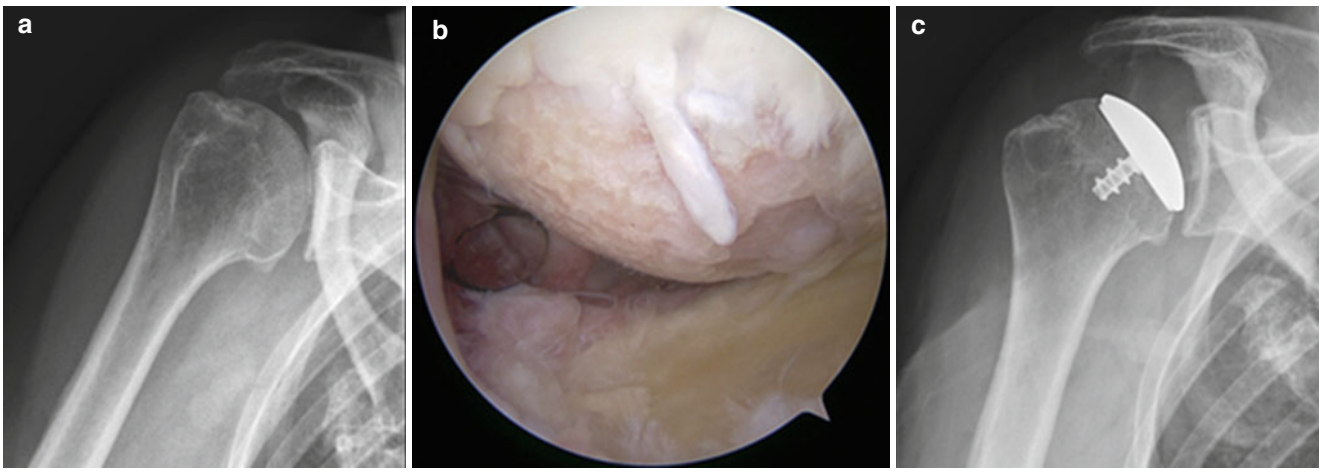

Fig. 43.8 (**a**) Plain radiograph and (**b**) intraoperative detected severe osteoarthritic changes of the humeral head of a 71-year old female patient. The new 35-mm Partial Eclipse prosthesis in situ (**c**) covering at least two-thirds of the humeral head

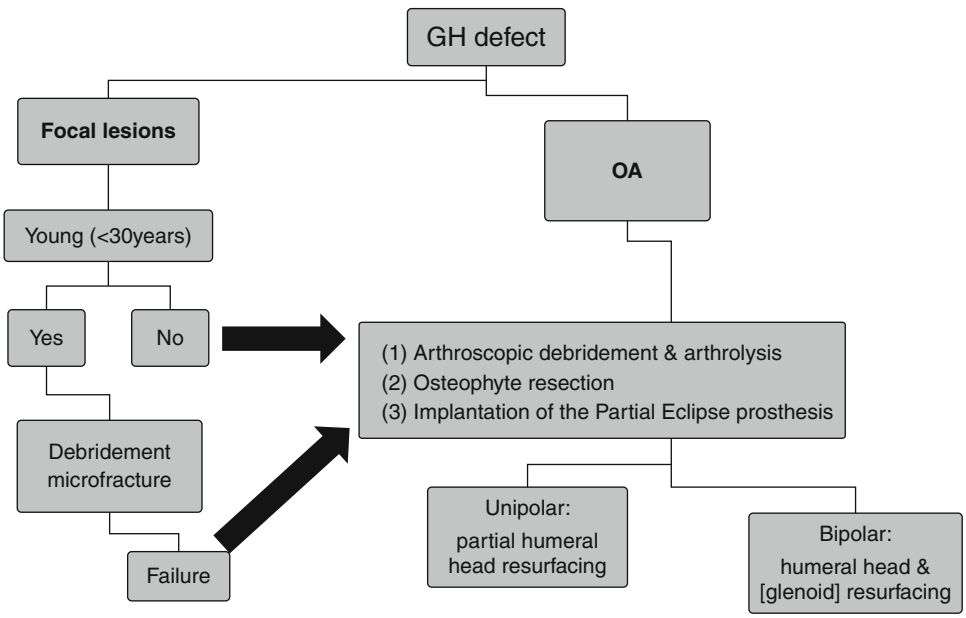

Fig. 43.9 Algorithm for our current arthroscopic approach to treat glenohumeral cartilage defects. *GH* glenohumeral, *OA* osteoarthritis

Table 43.1 Clinical results after arthroscopic resurfacing of the glenohumeral joint

Reference	Nr.	Age[b]	Indication	Surgical technique	FU	Clinical results[b]
Weinstein et al. [8]	25 patients	46 years (range, 27–72)	Early glenohumeral osteoarthritis	Debridement	1–5 years	Pain relief: 20 patients Unsatisfied results: 5 patients
Cameron et al. [10]	61 patients	50 years (range, 21–73)	Osteochondral lesions (grade IV) on the humeral head and/or glenoid	Debridement with/without capsule release	1–7 years	Return of pain: 35% of patients Revision surgery: 6 patients
Kerr and McCarty [9]	19 patients 20 shoulders	38 years (range, 20–54)	Articular cartilage defect (grades II–IV)	Debridement	1–3 years	Post-op ASES = 75 Uni-/bipolar lesions ASES = 93/66 Revision surgery: 3 patients
Van Thiel et al. [12]	71 patients	47 years (range, 18–77)	Glenohumeral degenerative joint disease	Debridement	1–8 years	Pre-op/post-op ASES = 52/73 Revision surgery: 16 patients
Savoie III et al. [22]	20 patients	32 years (range, 15–58)	Osteoarthritis (grade IV)	Biologic glenoid resurfacing with a patch	3–6 years	Pre-op/post-op CS = 26/79 points Revision surgery: 5 patients
De Beer et al. [23]	32 patients	Median 57.5 years (range, 36–69)	Osteoarthritis	Debridement and biological glenoid resurfacing	2–4 years	Pre-op/post-op median CS = 40/64.5 points Revision surgery: 5 patients Unsatisfactory outcome (failure): 4 patients
Millett et al. [13]	30 patients 31 shoulders	46 years (range, 19–59)	Full-thickness chondral lesions of the humeral head and/or glenoid	Microfracture	2–11 years	Pre-op/post-op ASES = 60/80 Revision surgery: 6 patients
Frank et al. [17]	14 patients 15 shoulders	37 years (range, 18–55)	Defect of the humeral head and/or glenoid (pain, injury, avascular necrosis)	Microfracture	1–7 years	Pre-op/post-op VAS = 5.6/1.9 Pre-op/post-op ASES = 44/86 Revision surgery: 3 patients
Gobezie et al. [24]	NA[a]	NA[a]	Large focal defects on the humeral head	Biologic resurfacing with osteochondral allograft	NA[a]	NA[a]
Anderl et al. (unpublished data, 2013)	11 patients 11 implants	60 years (range, 47–72)	Focal chondral defects on the humeral head (grade 4) Osteoarthritis (grades I and II)	Implantation of the Partial Eclipse prosthesis	1–2 years	Pre-op/post-op CS = 53/80 points Revision surgery: 3 patients

Abbreviations: *ASES* American Shoulder and Elbow Score, *CS* Constant score, *FU* follow-up, *NA* not applicable, *VAS* visual analog score
[a]Technical Note
[b]All values, unless otherwise stated, are presented as means

Summary

Arthroscopic management of OA requires both a disease- and patient-based approach. Clinical results of various arthroscopic treatments of OA of the glenohumeral joint are summarized in Table 43.1. Major advantages of all-arthroscopic procedures to treat OA are as follows: (a) excellent diagnostic evaluation of additional pathologies such as instability, rotator cuff lesions, and biceps diseases; (b) the fact that leaving the subscapularis tendon intact; (c) preserving bone stock provides a better starting position in case of later revision surgery; (d) as the surgical trauma is minimal in most techniques, surgery can be performed as an outpatient procedure; and (e) partial humeral head resurfacing with the Partial Eclipse prosthesis promotes immediate full rehabilitation.

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Arthroscopic Management of Tuberosity Fractures

44

Stefan Greiner and Markus Scheibel

Epidemiology

Although proximal humeral fractures are frequent, accounting for 5 % of all fractures, isolated fractures of the greater and lesser tuberosity in terms of a bony avulsion of the rotator cuff are quite rare [1]. 17–21 % of all proximal humeral fractures are isolated fractures of the greater tuberosity. Furthermore, this injury is frequently associated with glenohumeral dislocations, and in 15–30 % of all shoulder dislocations, there is an associated fracture of the greater tuberosity [2, 3].

Isolated fractures of the lesser tuberosity or bony avulsions of the subscapularis are extremely rare injuries. Fewer than 150 cases are described in the current literature, leading to the assumption that this injury is only about 2 % of all proximal humeral fractures [4]. It primarily occurs in young male patients between the second and fifth decade of life and in adolescent patients with an open epiphyseal plate at the proximal humerus [5–11].

Pathophysiology

A malposition of the greater tuberosity often leads to functional limitations and should therefore only be accepted within narrow limits of tolerance [12]. Based on anatomical studies of 140 cadavers, the highest point of the humeral head lies on average 3.2–8 mm above the highest point of the greater tuberosity [13]. Since a position of the greater tuberosity above the highest point does strictly not occur, it is obvious that even very small dislocations in cranial direction can alter clinical function of the shoulder joint. Particularly the function of the greater tuberosity as the insertion zone of the posterior-superior rotator cuff and the limited subacromial space is responsible for reduced flexion and abduction capacity by a cranial displaced fragment. Furthermore, even small dislocations of the tuberosities may lead to biomechanical changes of the rotator cuff and may result in degeneration of the associated muscles [14–16].

This contrasts with the classical Neer classification system of proximal humeral fractures [17], which does not distinguish within two-part fractures between isolated greater or lesser tuberosity fractures and subcapital fractures. According to Neer's recommendations with regard to two-part fractures including fractures of the tuberosities, the need for surgical treatment would only exist at a degree of 1 cm or 45° displacement.

McLaughlin [18], however, reported of functional limitations as a consequence of malunions of the greater tuberosity of more than 0.5 cm. Park et al. [19] recommended surgical reduction and fixation already at 3 mm of dislocation of the fragment, especially in overhead workers and athletes. This work emphasized also the importance of the direction of displacement. In particular, a cranial malunion caused greater functional limitations compared to a posterior or caudal displacement. On the basis of these observations, current treatment recommendation for a displaced greater tuberosity of more than 3 mm is the anatomic or slightly inferior-lateral fixation of the fragment in the active patient.

Since fractures of the lesser tuberosity are rare, they are often diagnosed late after trauma due to chronic shoulder pain [10]. The description of this injury in the literature is mainly based on case reports and case series. Standardized treatment strategies are not available at present. For non-displaced or minimally displaced fractures, primarily a conservative approach is recommended. Displaced fractures of the lesser tuberosity usually lead to insufficiency of the subscapularis muscle requiring surgical treatment. Treatment options range from open reduction and internal fixation to percutaneous procedures and arthroscopically assisted or all-arthroscopic treatment strategies [4, 10, 20, 21].

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History

There are different traumatic mechanisms, which can lead to a greater tuberosity fracture. A retrospective study of 103 patients with greater tuberosity fractures showed that a direct trauma mechanism was evident in 47.6 % and indirect in 32 % [22]. Moreover, an inferior dislocation of the greater tuberosity was observed in 25 % of the cases. This suggests rather an impaction against the acromion, respectively, in case of a shoulder dislocation against the glenoid than a true bony avulsion in pull direction of the rotator cuff. The authors concluded that a greater tuberosity fracture caused by shoulder dislocation can be considered as the maximal variation of a Hill-Sachs defect [22]. In contrast, an abduction and external rotation trauma is considered the reason for a fracture of the lesser tuberosity, which especially in younger patients with a strong inserting tendon of the subscapularis muscle and in patients with an open epiphyseal plate may lead to avulsion of the lesser tuberosity [23]. The previously described trauma mechanisms may also lead to a very rare combination of a fracture of the greater as well as the lesser tuberosity at the same time [22, 24].

Classification

Available classification systems do not sufficiently take into account the clinical significance of fractures of the greater and lesser tuberosities as insertion points of the posterior-superior and anterior rotator cuff. A retrospective study with 610 patients showed that patients with isolated greater tuberosity fractures differ significantly from the overall patient group in terms of gender, mean age, and association with shoulder dislocations and comorbidities [3]. Patients with greater tuberosity fractures compared to patients with proximal humeral fractures were predominantly male, about 11 years younger, and had a higher incidence of shoulder dislocations and significantly fewer comorbidities. The authors concluded that due to obvious demographic differences, it is necessary to establish an own classification system of this entity.

Fractures of the lesser tuberosity are also not well reflected by current classification systems. Corresponding to fractures of the greater tuberosity, slight dislocations are crucial for functional outcome [25]. The least common injury is the combination of both a greater and lesser tuberosity fracture with an intact humeral neck, which is not captured by the current classification systems as well [24, 26, 27].

Imaging

The main objective of diagnostic imaging is to assess the degree of fragment dislocation, as this is crucial for treatment decision.

X-Ray Imaging

Basically true anterior-posterior (a/p) and axillary radiographs are mandatory (Figs. 44.1 and 44.2). True a/p views in internal and external rotation are also useful to assess the

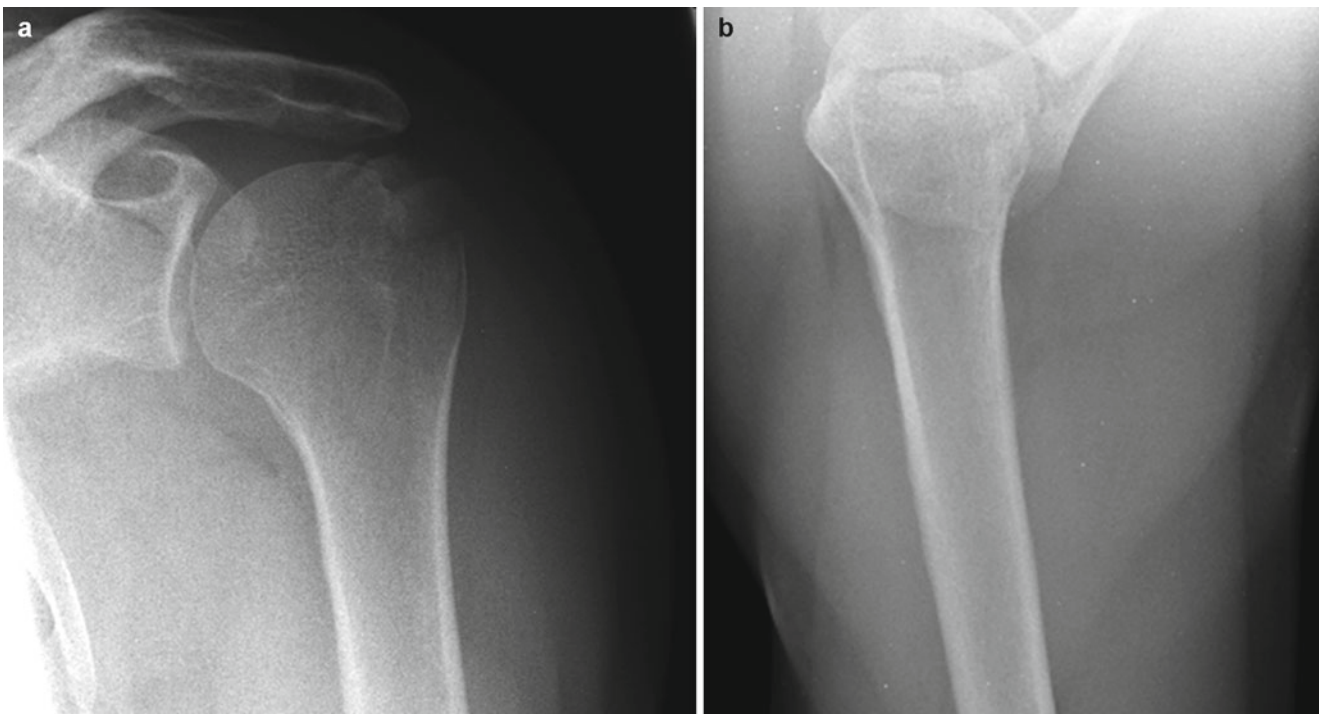


Fig. 44.1 True a/p (**a**) and axillary view (**b**) of a multi-fragmented greater tuberosity fracture

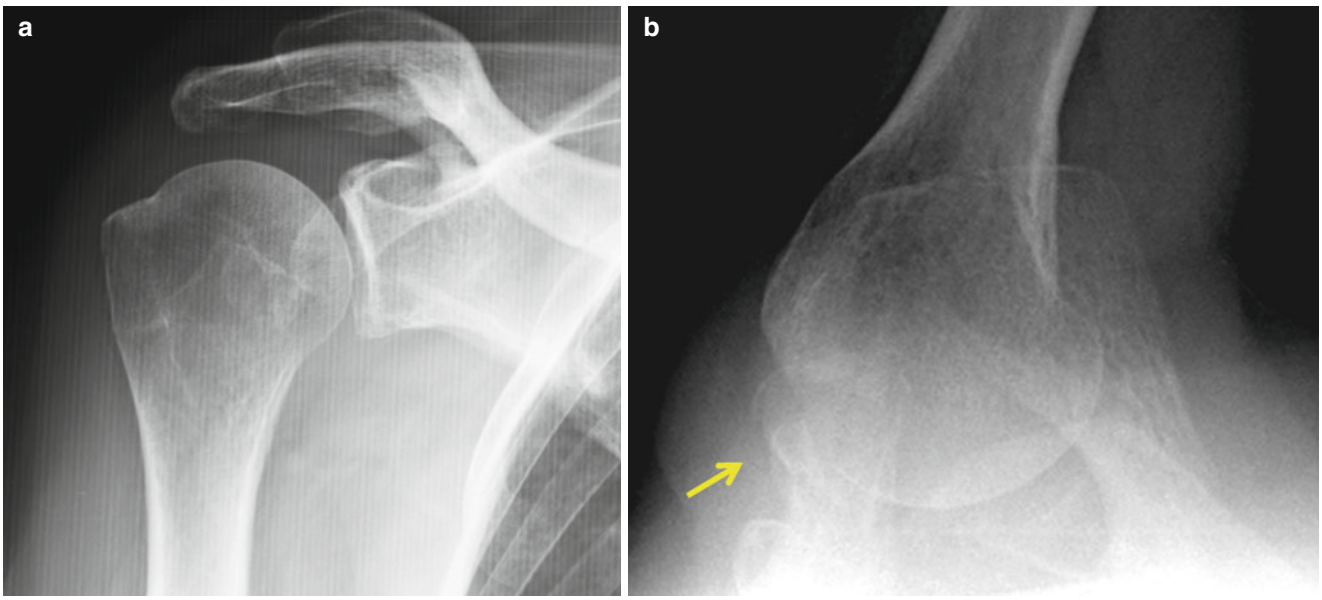


Fig. 44.2 True a/p (**a**) and axillary view (**b**) of a chronic isolated lesser tuberosity fracture. *Arrow* showing the displaced lesser tuberosity fragment

degree of fragment dislocation of the tuberosity more accurately and to eventually exclude an extension of the fracture into the surgical neck. Furthermore, concomitant pathologies such as a dislocation or subluxation and fractures of the glenoidal rim can be diagnosed.

A recent study showed that the critical posterior-superior dislocation of the greater tuberosity is best assessed by using a/p views in external rotation or a/p views with a course of beam 15° caudally directed [28].

Computed Tomography

CT-scan evaluation is recommended if radiographs do not allow an adequate assessment of the displacement relevant for indicating surgical therapy. In axial slices a posterior dislocation of the greater tuberosity or a medial dislocation of the lesser tuberosity can be depicted properly. Coronal and 3-D reconstructions are necessary and helpful to detect shifts in the craniocaudal plane as well (Figs. 44.3 and 44.4).

Magnetic Resonance Imaging

MRI has been shown to be of importance for diagnostics in these injuries also. A recent study showed that all patients with unsatisfactory results after conservative therapy of minimally displaced greater tuberosity fractures showed a partial lesion of the rotator cuff [29].

Basically, in doubt, a CT or MRI scan of the affected shoulder should be performed, especially if radiographs do not allow proper diagnosis despite appropriate clinical signs [30].

Ultrasound examination is useful to diagnose occult fractures of the greater tuberosity but remains investigator dependent in terms of assessing the amount of displacement [31].

Literature Review

Regarding surgical treatment of bony avulsions of the rotator cuff, no consensus can be found in the literature. Various percutaneous and open stabilization methods are described [32].

Due to enhanced development of arthroscopic procedures, they are becoming increasingly important, especially because they allow improved visualization and mobilization of the fragments. Furthermore intra-articular concomitant pathologies can be diagnosed and treated adequately at the same time.

Fractures of the Greater Tuberosity

Initial case reports and descriptions of arthroscopic surgical techniques suggested primarily the arthroscopic mobilization and reduction of the fracture, followed by temporary K-wire fixation and subsequent percutaneous screw fixation under arthroscopic control [33–37]. Common consent in these reports is the improved visualization and mobilization of the fragment, followed by the ability to precisely reconstruct the insertion of the rotator cuff. However, multifragmental fractures, poor bone quality, severe dislocation, and fixed retraction are limitations of this method.

First publications on suture anchor fixation using an open approach showed promising results in long-term follow-up.

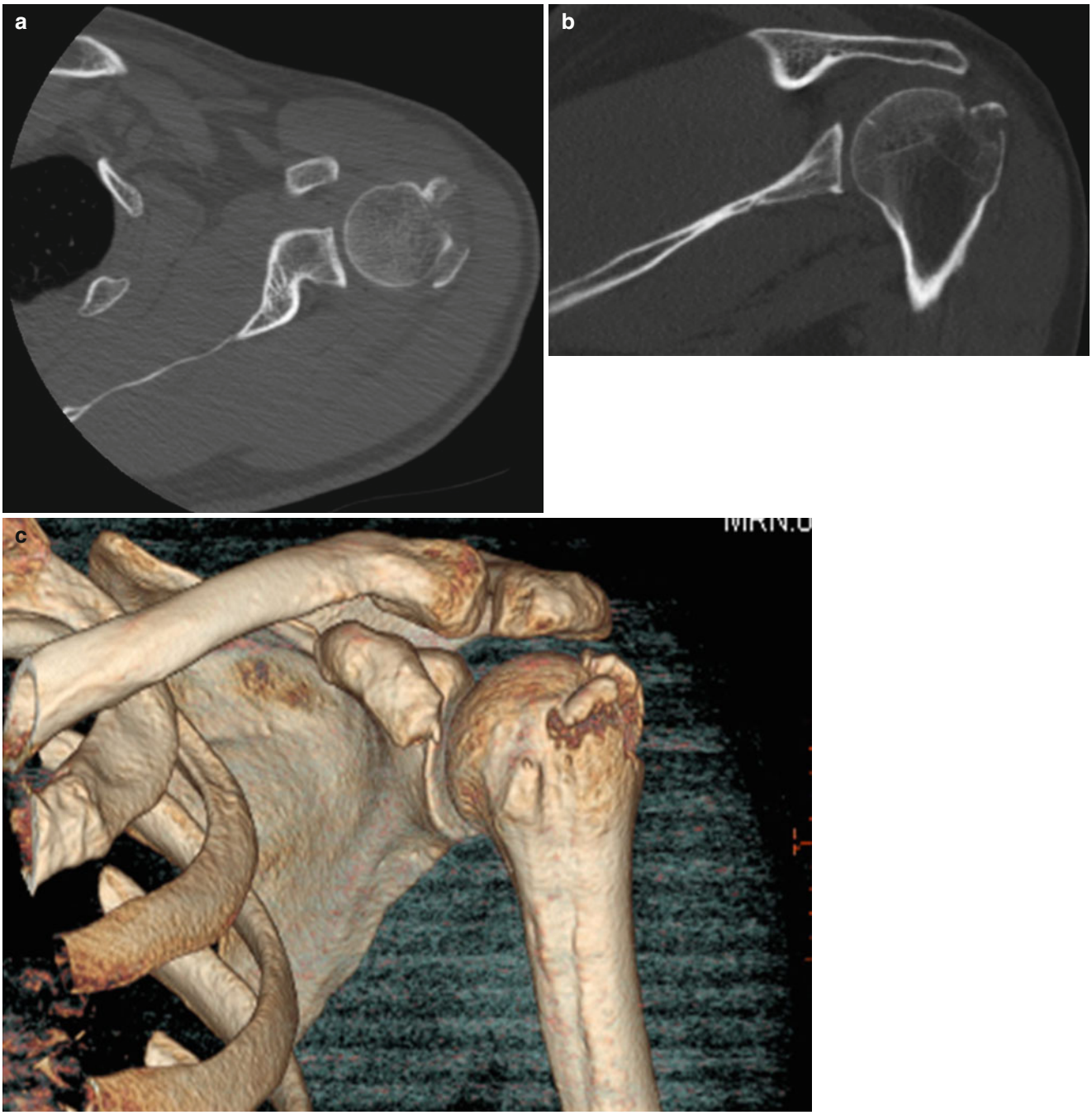


Fig. 44.3 2-D (**a, b**) and 3-D (**c**) CT imaging showing the displacement and size of the fragments

Bhatia et al. [38] treated 21 patients with multi-fragmented fractures of the greater tuberosity with a mean age of 51 years with a double-row suture anchor reconstruction of the rotator cuff-tuberosity complex. After an average of 3.5 (1–5 years), 20 patients showed healing without secondary displacement with 18 very good and good, 2 satisfactory results, and one unsatisfactory result. Two patients showed postoperative biceps tendon pathology, and one patient was revised due to an immunogenic response to the implant.

Ji et al. [39] introduced an arthroscopic technique equivalent to arthroscopic double-row rotator cuff repair. After debridement of the fracture zone using the shaver, the anchor of the medial row is inserted through the intact rotator cuff under intra-articular visualization. Further, under subacromial view, two more anchors are inserted: one anterior and one posterior to the lower edge of the fracture. The sutures are passed through the tendon at the bone-tendon junction. Maintaining the reduction under x-ray control using a blunt

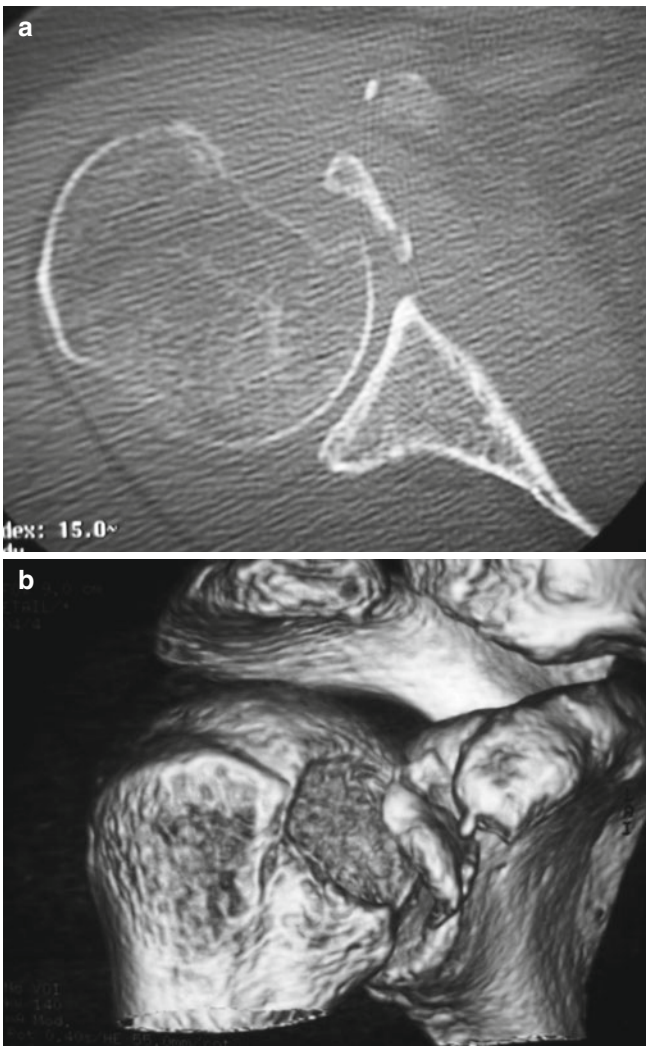


Fig. 44.4 2-D (**a**) and 3-D (**b**) CT imaging showing the chronic displaced and partially absorbed lesser tuberosity fragment

trocar, first the medial row is tied restoring the footprint, followed by the lateral row, which fixes the fragment additionally.

The introduction of knotless anchors allowed development of double-row suture-bridge reconstruction techniques. Kim et al. [40] and Song et al. [41] presented similar procedures where the first two suture anchors are inserted transtendinously through the bone-tendon junction in the humeral head. The medial row is tied and each thread of one medial anchor is introduced in a knotless anchor. The fracture is reduced and sutures are pulled over the greater tuberosity and are locked distally to the fragment with the knotless anchors [40, 41]. Ji et al. [42] presented a prospective case series using this method. They presented the clinical and radiological results of 16 patients with a fracture of the greater tuberosity and a displacement of at least 5 mm with an average follow-up of 14 months after surgery. Postoperative results showed a mean ASES score of 88.1 and an UCLA score of 31 points. Interestingly, the results showed a significant correlation between better score results and anatomical healing of the fragment. Authors report also about limitations of this method: Arthroscopic fixation was not possible in case of a dislocation in posterior-inferior direction in terms of an avulsion of the infraspinatus or teres minor, which then had to be switched to an open procedure [42].

Fractures of the Lesser Tuberosity

Due to the rarity of this injury, existing literature on the treatment of lesser tuberosity fractures consists mainly of case reports, description of surgical procedures, and small case series. The majority of reported cases in the literature were treated conservatively or open using cannulated screws [5, 8, 10, 43, 44]. A case series of ten patients with fractures of the lesser tuberosity demonstrated that clinical outcome after surgical treatment was superior to conservative treatment. In this report three patients were treated conservatively and three patients underwent surgical fixation. Additionally, four chronic cases were treated conservatively. Due to superior results, authors recommended surgical treatment for acute fractures of the lesser tuberosity [10]. In a consecutive series of six cases with fractures of the lesser tuberosity, operative treatment for displaced fractures showed promising results as well [45].

Another case series of 16 patients also showed superior results with surgical treatment. 11 patients were treated surgically. The lesser tuberosity was fixed in five patients. In the remaining six cases, the fragment was excised and reconstruction of the subscapularis muscle was performed. Four patients showed a dislocated long head of biceps tendon which was treated with a tenodesis [46].

Robinson et al. [25] presented the largest published series of 22 consecutive patients with fractures of the lesser tuberosity so far. 17 of those patients were treated surgically. The fragment was fixed in 11 patients by at least two screws using a deltoid-pectoral approach. In two patients the fragment was smaller than 2 cm and fixation was performed using transosseous sutures. One year after surgery, the Constant score was 95 and the DASH score was 12 points. Furthermore, all patients had a negative lift-off test at 2-year follow-up. The authors conclude that in a displaced lesser tuberosity fracture, regardless of the degree of fragment displacement, surgical reconstruction should be favored due to unpredictable results with delayed reconstruction and the possibility of further fragment dislocation due to the pull of the subscapularis [25, 44, 45].

Reports of arthroscopic procedures in lesser tuberosity fractures are rare. Scheibel et al. [21] reported about a 35-year-old patient with bony avulsion of the subscapularis tendon and a 5 mm dislocation of the fragment without

involvement of the bicipital groove. Surgical treatment was all arthroscopic with two suture anchors, which were placed in the fracture zone. The sutures were passed through the bone-tendon junction of the subscapularis and tied as a mattress suture. This allowed an anatomical reconstruction, and 6 months after surgery, the patient showed free active and passive range of motion while having a negative subscapularis signs.

Kowalsky et al. [47] reported about good functional outcome after arthroscopic coracoid plastic in a case of a malunion of the lesser tuberosity, that caused a subacromial impingement. However, at 3 months after surgery, there was a remaining impairment of internal rotation in this patient [47].

Complications

Poor results after conservative treatment are often related to contractures of the capsule. However, symptomatic patients often suffer lesions of the rotator cuff also. Furthermore, even a limited cranial malunion may lead to subacromial impingement, and scaring in the subacromial space may cause impingement symptoms as well. Posterior malunion of the greater tuberosity may limit external rotation.

Medial malunion of the lesser tuberosity may lead to a subcoracoidal impingement. Malunion or nonunion may also attenuate the associated rotator cuff and cause consecutive degeneration of the dependent muscles. Complications of surgery are infection, secondary dislocations, nerve injuries, and postoperative stiffness. Using an open procedure, additionally the morbidity of deltoid split access is a concern. Treatment of malunion and nonunion of the greater tuberosity is challenging, and literature repeatedly pointed out difficulties and limited success after surgical treatment of this entity [18, 48].

Treatment: Indications/Contraindications and Decision-Making Algorithm

Indication is based on true a/p and axillary x-rays. If an accurate assessment of the amount of dislocation is not possible, the extent of infraction is not assessable, or involvement of the anatomical or surgical neck cannot be excluded, a CT scan with 3-D reconstructions is performed. Particularly in fractures of the greater tuberosity after a shoulder dislocation, MRI examination is recommended to evaluate the labrum, capsule, and ligaments. Also attention has to be paid to the subscapularis tendon that can be found torn after a dislocation event in addition to a fracture of the greater tuberosity.

Indication for surgery is, based on the current literature, a dislocation of the greater tuberosity of 5 mm in combination with patient-related factors such as comorbidities, age, and activity level. In overhead athletes or workers, surgical reduction and fixation is performed already with a dislocation of 3–5 mm. Especially with multiple fragments and avulsion of the rotator cuff, an arthroscopic approach is favored. In contrast, an extension of the fragment of more than 3 cm caudally to the upper edge of the tuberosity makes all-arthroscopic treatment more difficult.

Arthroscopic Treatment: Surgical Technique

Patient Positioning and Portal Placement

The patient is placed in beach-chair position, with positioning of the affected limb in a pneumatic arm holder. First, access via a standard posterior portal and diagnostic arthroscopy with particular attention to the footprint of the rotator cuff and the integrity of the long head of the biceps pulley system is performed.

Using an anterior-inferior portal, the insertion of the subscapularis, supraspinatus, and infraspinatus is investigated. The integrity of the long head of the biceps and its stability are examined with a probe, followed by an evaluation of the subacromial space, using the posterior portal. Via an anterior-lateral portal subacromial bursectomy, preparation of the fracture margins and debridement of the fracture zone are performed.

Step-by-Step Procedure

Fractures of the Greater Tuberosity

Under arthroscopic control from an intra-articular view, the fragment is reduced. Then, two suture anchors are inserted transtendinously through the bone-tendon junction directly into the fracture at the border of the cartilage. Sutures are passed medially and laterally of the transtendinous approach through the rotator cuff using a suture shuttle. While visualization from the subacromial space sutures are tied, the fragment is reduced. Subsequently, sutures are passed over the tuberosity and are fixed via one or two knotless anchors caudally of the fracture zone. If the fracture extends distally, direct arthroscopic visualization and image intensifier-controlled positioning of the distal anchor are recommended. Recently, this method has been optimized using suture anchors loaded with Fiber Tapes (Arthrex, Naples, Florida, USA). Anchors with Fiber Tapes are inserted transtendinously or directly through the fracture. Without tying the Fiber Tapes are passed over the tuberosity and are cross-fixed caudally of the fracture (Speed-Bridge technique). This

procedure simplifies anatomic reconstruction with adequate fracture compression (Fig. 44.5).

During the period from 07/2008 to 05/2010, six of our own patients (three women and three men) were treated in the described manner with a mean age of 49 years (26–66 years). In four patients the dominant arm was affected.

The radiological examination showed complete consolidation of the fracture in all patients after 6 months of follow-up (Fig. 44.6). Four patients showed anatomical positioning; two patients had a caudal positioning <5 mm in the true a/p views. All patients were pain-free, and after an average of 16 months of follow-up, they reached an average Constant score value of 84.5 points [49].

Fractures of the Lesser Tuberosity

Basically, all patients with a dislocated fracture of the lesser tuberosity and without contraindication for surgery undergo arthroscopy in order to treat the main pathology and to detect and treat possible concomitant pathologies such as injuries of the long biceps tendon.

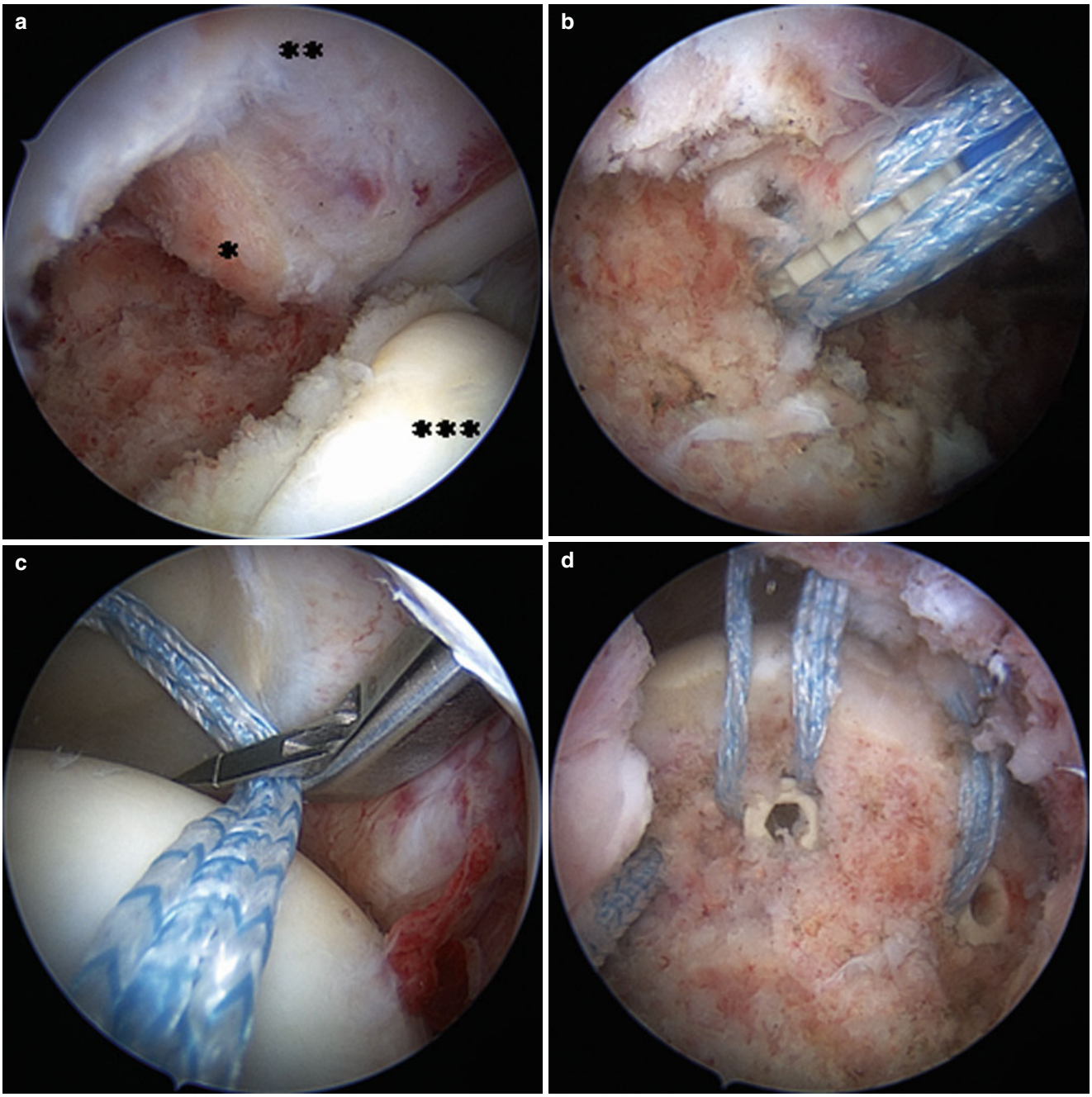
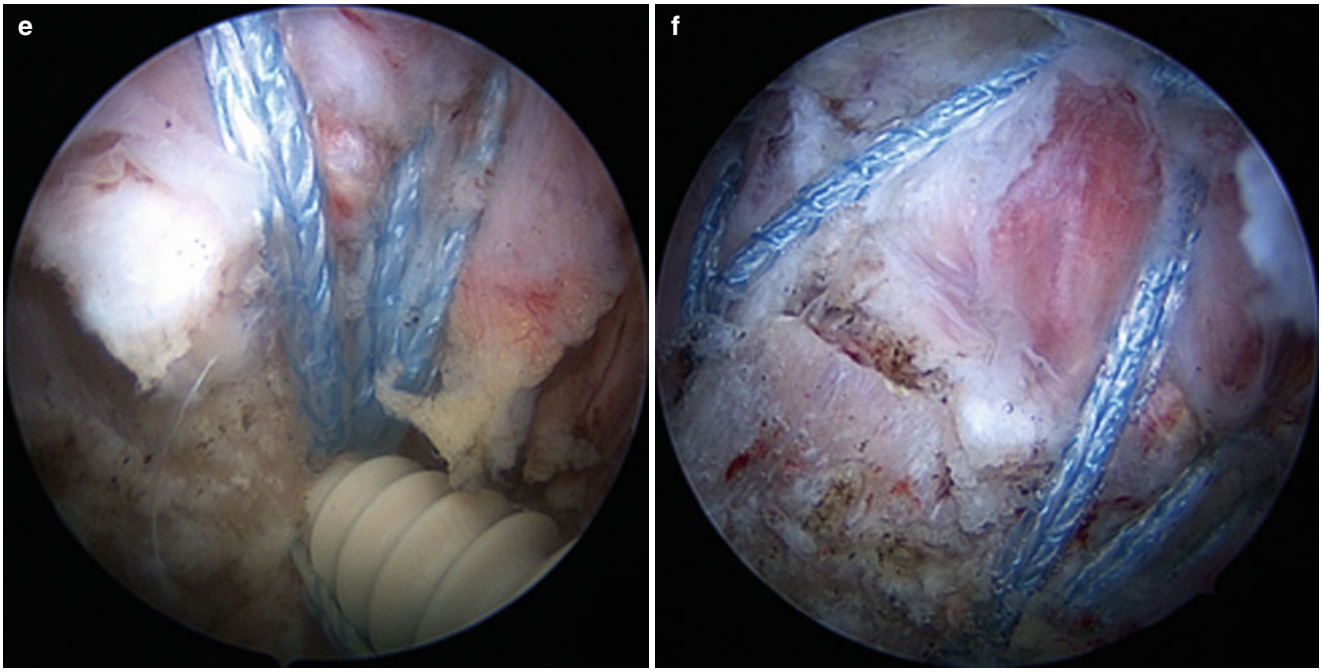


Fig. 44.5 (**a**–**f**) Arthroscopic Speed-Bridge technique for reconstruction of a bony supraspinatus tendon avulsion. (*) greater tuberosity fragment, (**) supraspinatus tendon (***) humeral head



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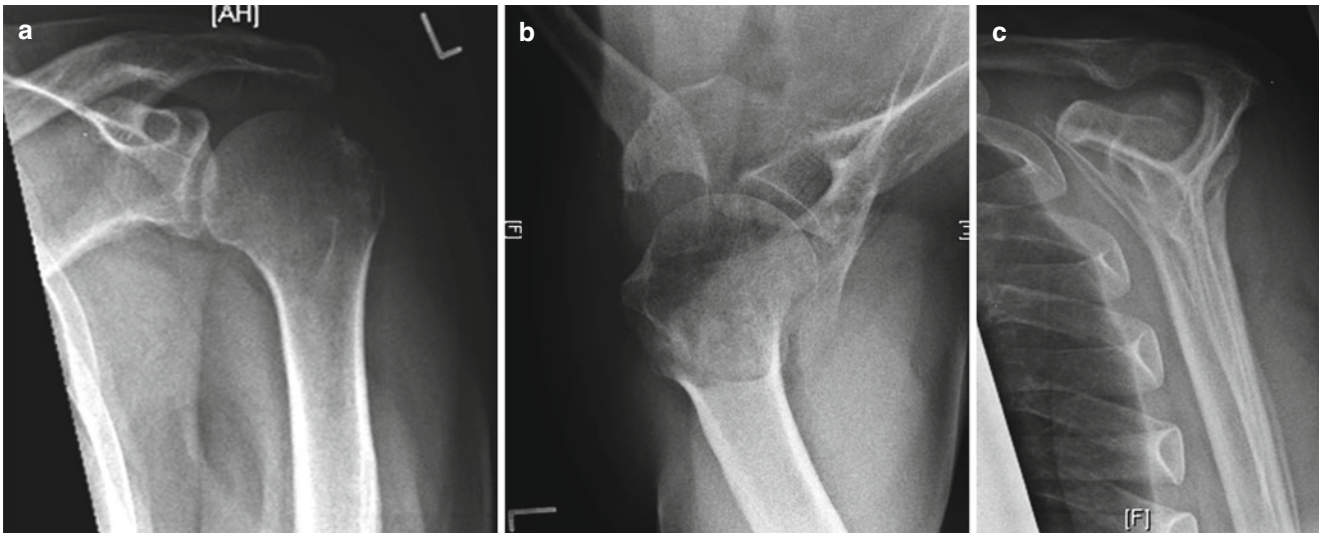


Fig. 44.6 True a/p (**a**), axillary (**b**), and Y-view (**c**) after arthroscopic Speed-Bridge repair

Suture anchor fixation is favored, and depending on the fragment morphology and dislocation, double-row reconstruction techniques may be used. The medial suture anchors are placed at the medial fracture area similar to bony avulsions of the supraspinatus muscle; the sutures are then passed through the subscapularis tendon at the bone-tendon junction and are tied and cross-fixed laterally over the fragment with knotless anchors (Figs. 44.7 and 44.8).

Problems sometimes arise due to poor bone quality in the fractured area. Placing the anchors more distally in the bone is helpful in these cases. Solid fragments can be reduced arthroscopically and fixed with two cannulated screws. Due to the small number of cases and the rarity of this injury, no standardized advice for surgical treatment can be made [49].

Combined Fractures of the Greater and Lesser Tuberosity

Only a few reports regarding combined fractures of the greater and lesser tuberosity exist in the literature [24, 26, 27]. We recently reported about a 28-year-old male patient who suffered a bike accident with sudden forced external rotation and abduction to the right shoulder. 3-D CT-scan

evaluation showed a cranial-medial dislocation of the lesser tuberosity, while the main fragment of the comminuted greater tuberosity was dislocated about 5 mm posterior caudally. The lesser tuberosity was fixed using the above-described method with two suture anchors. The greater tuberosity was addressed accordingly with two suture anchors loaded with Fiber Tapes (Arthrex, Naples/FL). Due to the large distal excursion of greater tuberosity fragment, additionally cannulated screw fixation was performed, and the Fiber Tapes were pulled over the tuberosity and fixed to the screw. Clinical and radiographic follow-up showed recovery of active motion and consolidation of the fragments in anatomic positions 6 months after surgery [50].

Summary

Bony avulsions of the rotator cuff are rare injuries. However, regarding possible impairment of clinical function, they are extremely relevant. Current classification systems capture

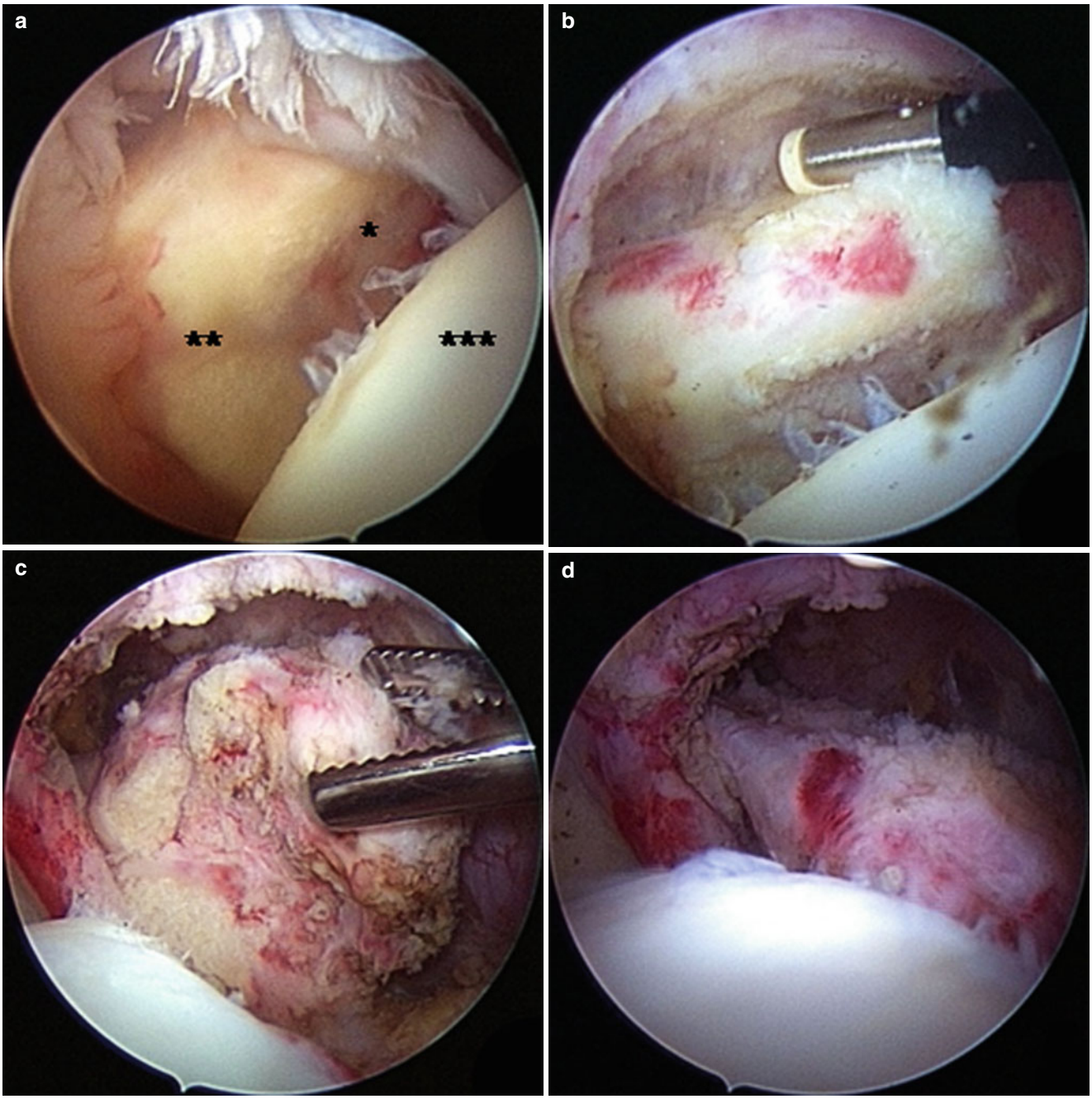


Fig. 44.7 (**a**–**l**) Arthroscopic suture-bridge technique for reconstruction of a chronic bony subscapularis tendon avulsion. (*) Lesser tuberosity fragment, (**) supraspinatus tendon (***) humeral head

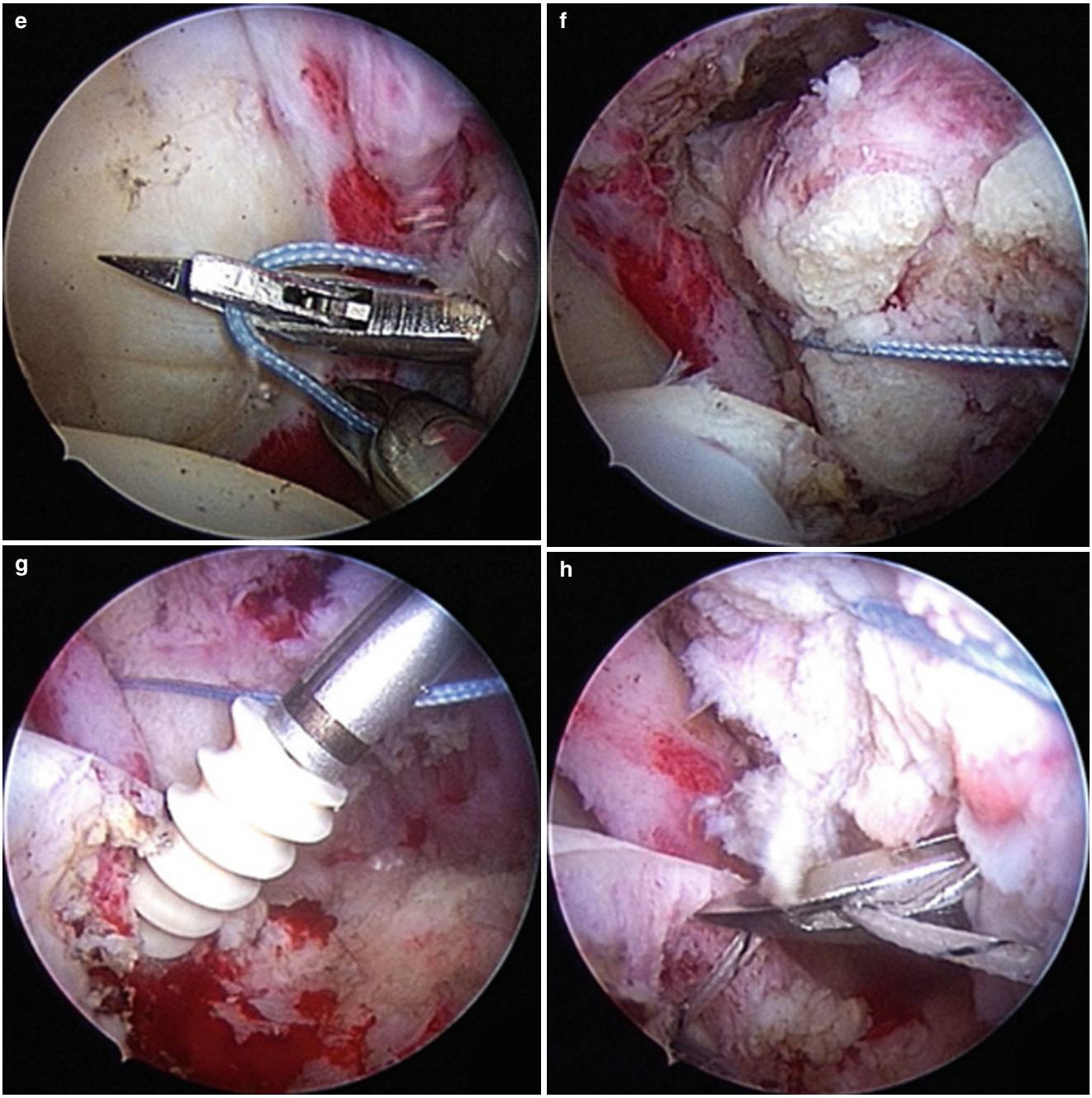
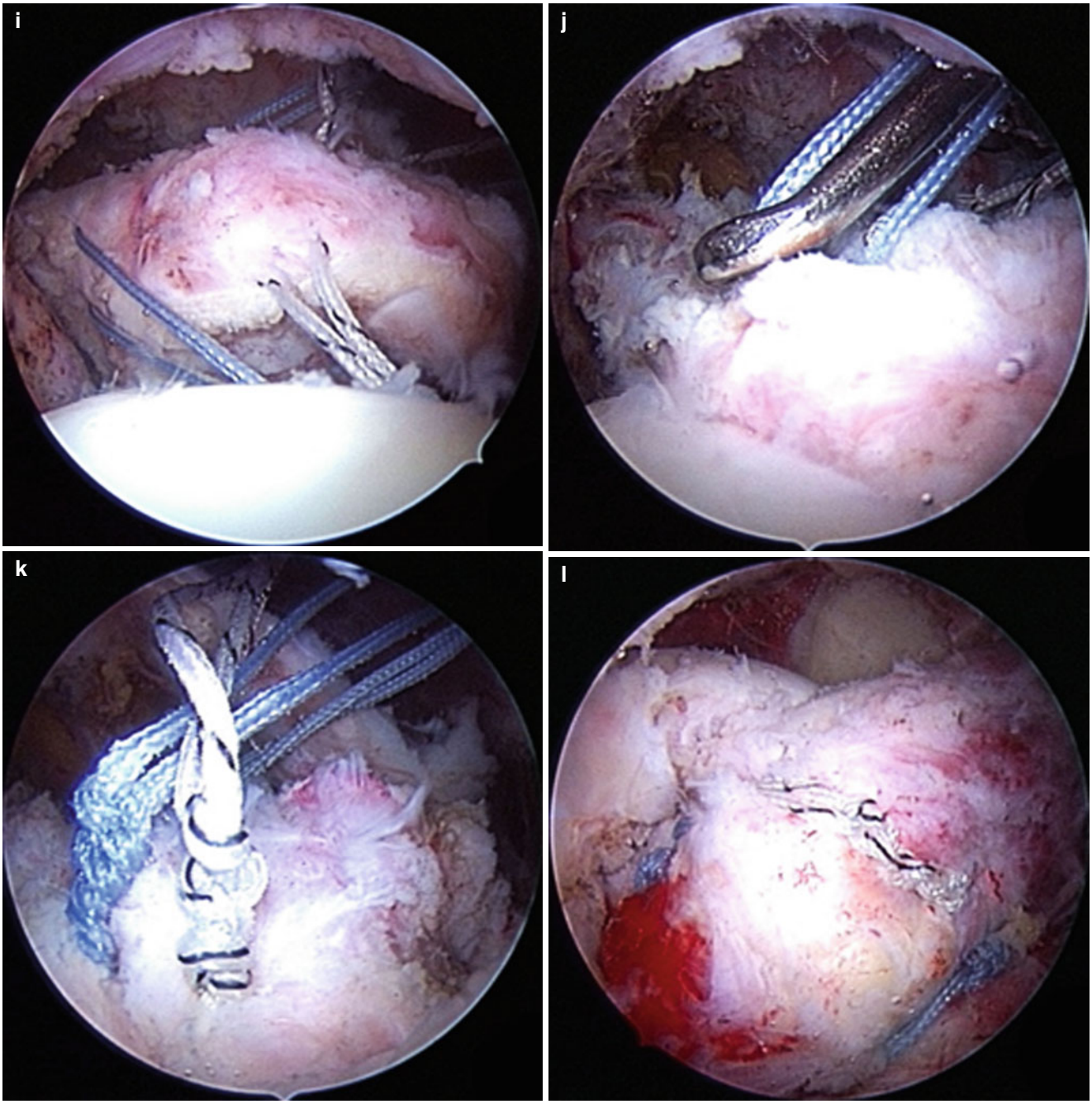


Fig. 44.7 (continued)



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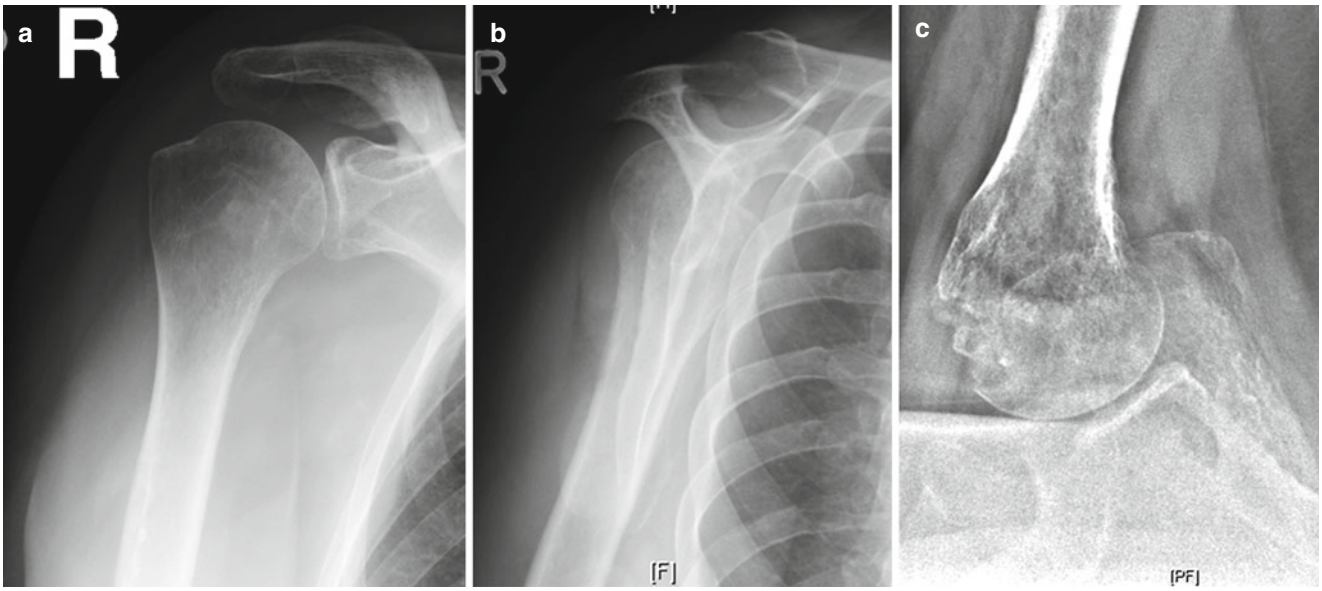

Fig. 44.8 True a/p (**a**), axillary (**b**), and Y-view (**c**) after arthroscopic suture-bridge repair

these injuries only insufficiently. Fragment dislocation should be accepted only within narrow limits, since otherwise functional deficits may occur. Arthroscopic techniques allow better visualization, treatment of concomitant injuries, and secure fixation. However, so far there is a lack of larger case series in the literature.

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Acromioclavicular Joint Instability

45

Frank Martetschläger, Sepp Braun, and Andreas B. Imhoff

Epidemiology

Acromioclavicular (AC) joint injuries are common and account for about 12 % of all shoulder injuries in clinical practice [1]. This number increases to almost 50 % in athletes participating in contact sports. The true prevalence might even be underestimated since many individuals with low-grade (type I or II) injuries may not seek medical attention [2]. A recent longitudinal cohort study reported on an incidence of 9.2/1,000 injuries among young athletes, whereas male patients experienced a significantly higher incidence rate than female patients [3]. This is most likely due to a different risk-taking behavior and contact sports rather than anatomic differences between genders. The most AC joint injuries occur in the third decade, and the sports most likely to contribute to the incidence of AC joint dislocations are football, soccer, hockey, rugby, biking, and skiing [2, 4–6]. The mechanism of trauma is frequently a direct blow to the shoulder with the arm in an adducted position. Due to the excessive strength of the sternoclavicular joint, the AC joint and the clavicle represent the weak points for injury [4, 7].

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Pathophysiology

AC joint stability is provided by the joint capsule, with the superior, inferior, anterior, and posterior AC ligaments and the coracoclavicular (CC) ligaments. Native AC joint structures tolerate displacements of 4–6 mm in the anterior, posterior, and superior planes and under a 70 N loading [8]. Rotary motion of 5°–8° is experienced during scapulothoracic motion and 40°–45° during shoulder abduction and elevation [9, 10].

The four AC joint ligaments are horizontally directed and mainly contribute to horizontal stability, whereas the superior and the dorsal ligaments contribute the most to anterior-posterior stability [8], whereas the superior AC ligament is the largest and strongest ligament of the AC joint complex [11].

The CC ligaments, namely, the conoid (anteromedial) and trapezoid (posterolateral) ligaments, span from the inferior surface of the flattened distal clavicle to the base of the coracoid process. They mainly contribute to vertical stability. Rios et al. [11] determined a ratio of the CC ligament insertions to total clavicle length (17 % trapezoid, 31 % conoid), which appeared to be more accurate for AC ligament reconstruction compared to actual distance measurements, regardless of gender. The trapezoid ligament shows a quadrilateral shape, and the conoid ligament takes a conical shape with its base facing superiorly [7]. The ultimate failure load of the native AC capsule ligament complex during superior loading has been shown to be 590 ± 95 N [12], whereas a different study has reported the ultimate failure load of the separated CC ligaments to be 500 ± 134 N [13]. From ligament sectioning studies we know that the inferior AC capsular ligament is the primary restraint to anterior translation, while the trapezoid ligament primarily prevents posterior translation [14]. However, other studies have suggested the posterior and superior AC ligaments to primarily contribute to posterior stability [15, 16]. Restriction of superior translation and rotation appears to be mainly provided by the conoid ligament [8, 15].

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Since in type II injuries the AC ligaments fail before the CC ligaments, one can conclude that the AC ligaments resist quantifiably smaller displacement moments than the CC ligaments. Therefore, complete disruption of the AC ligaments renders the CC ligaments the primary restraint for AC stability [8, 15].

The knowledge of this anatomy/pathoanatomy of the mechanical stabilizers is mandatory for the correct classification and surgical treatment, especially when using modern anatomic reconstruction techniques.

History

Patients with AC joint injuries typically complain of a generalized shoulder pain. Therefore, a thorough history is mandatory for correct diagnosis and treatment. A complete patient history includes a trauma anamnesis with exact mechanism of trauma and onset of symptoms. Usually the pain is acute with a history of trauma, typically including a direct force to the lateral aspect of the lateral shoulder [4, 7]. In more chronic cases with a trauma lying further in the past, the thorough anamnesis might be more difficult, but since symptoms can be unspecific, it is even more important for correct diagnosis. In the chronic setting patients typically complain of superior shoulder pain, which can be provoked when the arm is brought across the body or during weight lifting activities such as the bench press.

Clinical Examination

Inspection of the shoulder girdle may reveal abrasions of the shoulder and apparent prominence of the distal clavicle resulting from inferior displacement of the scapula. The palpation of the AC joint will reveal tenderness in the acute setting and the direction of instability can be detected. Range of motion exercises typically show an impaired shoulder function limited by pain [17–20].

Clinical provocative tests for AC joint pathology (O'Brien, Paxinos, and scarf tests) might be helpful to localize anterior/superior shoulder pain to the AC joint. These tests are especially useful in patients with low-grade injuries (types I and II) in which palpable deformity may not be present [2, 4].

Since it has been shown that concomitant intra-articular injuries frequently occur in high-grade AC separations (types III–VI), it is important to rule out these injuries in addition. A study by Tischer et al. [19] demonstrated the presence of ancillary intra-articular injuries in 14 of 77 patients with type III–VI injuries, whereas 11 of 77 patients also had superior labral anterior-posterior (SLAP) lesions. In carefully selected cases an AC joint injection with lidocaine may be helpful in discriminating AC joint pain from other pathologies causing anterior/superior shoulder pain.

Imaging

The standard radiographic examination when detecting for AC joint injuries includes anterior-posterior (AP), scapular Y, and Alexander or Zanca films. The AP view allows for identification of the vertical displacement of the distal clavicle, whereas the Alexander view is used to identify displacement in anterior-posterior direction. The Zanca view, an AP view that is tilt 10–15° cephalad, is helpful in giving a clear view of the AC joint without superimposing structures [21]. Bearden et al. [22] found that a 25–50 % increase of the CC interval was indicative of complete CC ligament disruption. Therefore, the CC interval can be measured and compared to that of the contralateral shoulder in cases of uncertain degree of severity. Weighted stress radiographs have been used to distinguish type II from occult type III injuries [23, 24]; however, it has been shown that these films do not improve the diagnostic accuracy and cause needless patient discomfort [25, 26].

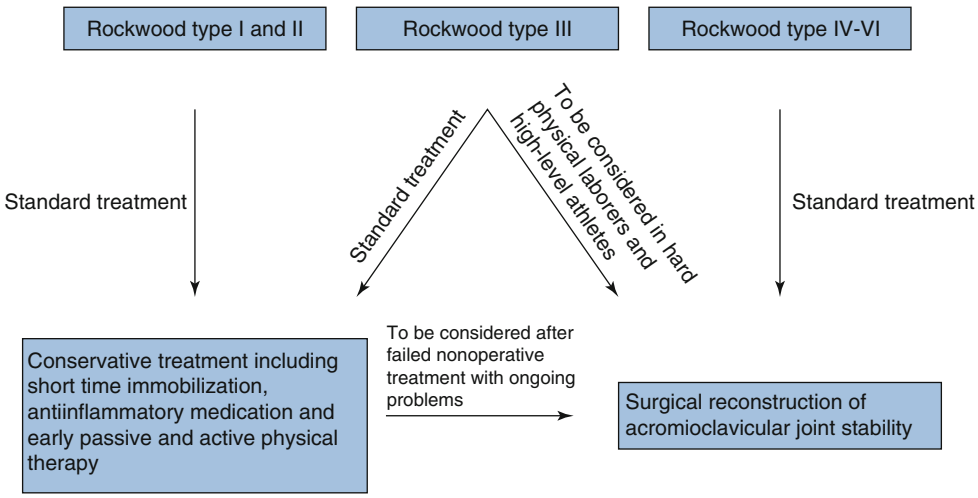
Treatment: Indications and Contraindications

Today, nonoperative treatment is generally recommended for type I and II injuries since several studies have shown satisfactory results [27–31]. This treatment typically includes a brief immobilization (1–3 weeks) of the shoulder followed by early range of motion exercises.

However, in contrast, several studies have described that persistent symptoms are common even after nonoperative treatment of low-grade injuries [27, 28, 32, 33]. Furthermore, data by Song et al. [34] suggest that early distal clavicle excision might be beneficial in some patients with type II injuries. However, to date there is no hard evidence for indicating surgical treatment for type I and II injuries.

Treatment of type III AC injuries is still controversial. Since clinical studies could not show significant advantages for either treatment, a trial of conservative treatment is typically recommended [35–38]. However, other studies suggest that early surgical treatment of type III injuries may result in better clinical outcomes compared to patients undergoing surgery at a point greater than 3 months beyond the injury after unsuccessful nonoperative therapy [39, 40]. Therefore, early surgical repair of type III AC lesions might be considered in manual workers or overhead athletes [35, 36, 40]. Type IV through VI lesions are typically treated surgically in order to avoid the reported long-term sequel [6, 28, 35, 36, 40].

Table 45.1 Decision-Making Algorithm for Treatment of AC Joint Instability



Possible contraindications or limitations for surgical interventions include concomitant acute fractures of the coracoid process or the distal clavicle and the common general contraindications for surgical treatment.

Decision-Making Algorithm

A decision-making algorithm based on the review of the current literature is shown in Table 45.1. Primarily, the correct diagnosis has to be established and the lesion is graded according to the Rockwood classification [41]. Based on this classification, the lesions are divided in low-grade (types I and II), type III, and high-grade (types IV through VI) injuries. Patients with low-grade injuries are treated with conservative therapy, which includes a short period of immobilization (1–3 weeks) and early passive and active therapy. Nonsteroidal anti-inflammatory drugs (NSAIDs) are prescribed as necessary. The treatment of most type III injuries is identical; however, the time of immobilization in a sling may be extended as needed. Surgical treatment might be considered for hard laborers and high-level athletes as discussed above. Furthermore, persistent pain, discomfort, and impairment of shoulder function may be an indication for surgery, which has to be discussed with the patient. Acute surgical stabilization is typically recommended in high-grade injury separations.

Clinical Case/Example

A 22-year-old male suffered a traumatic AC joint dislocation during a handball match. The mechanism of injury was a direct force from a fall on the lateral aspect of the shoulder with the arm in an adducted position. Right after the fall the patient reported to have shoulder discomfort and a painfully restricted range of motion. Therefore, he presented to our department seeking medical treatment. Prior to the match, the patient was completely asymptomatic without a history of injury or trauma.

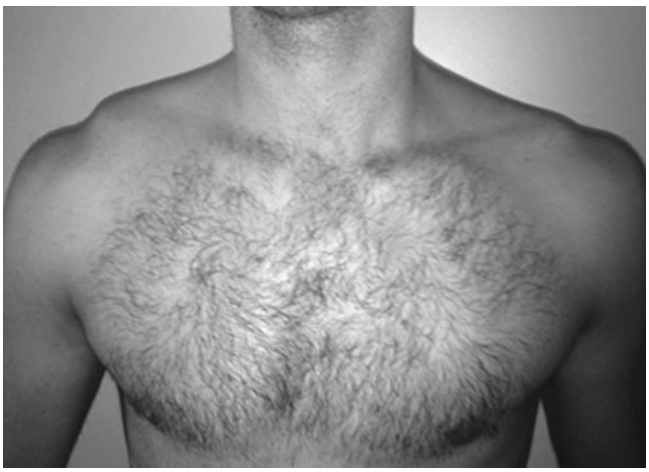


Fig. 45.1 Preoperative photograph of the shoulder girdle revealing a distinct prominence of the right lateral clavicle when compared to the contralateral side

Inspection of the shoulder girdle revealed a distinct prominence of the lateral clavicle when compared to the contralateral side (Fig. 45.1).

During initial physical examination of the shoulder, there were tenderness to palpation over the AC joint and a significant vertical instability of the lateral clavicle. Range of motion exercises showed an impairment of active shoulder function limited by pain. Global testing for rotator cuff function and strength was uneventful and neurovascular examination was within normal limits.

Radiographs of the affected shoulder revealed no bony lesions. There was a significant displacement of the lateral clavicle, corresponding to a Rockwood type V lesion (Fig. 45.2).

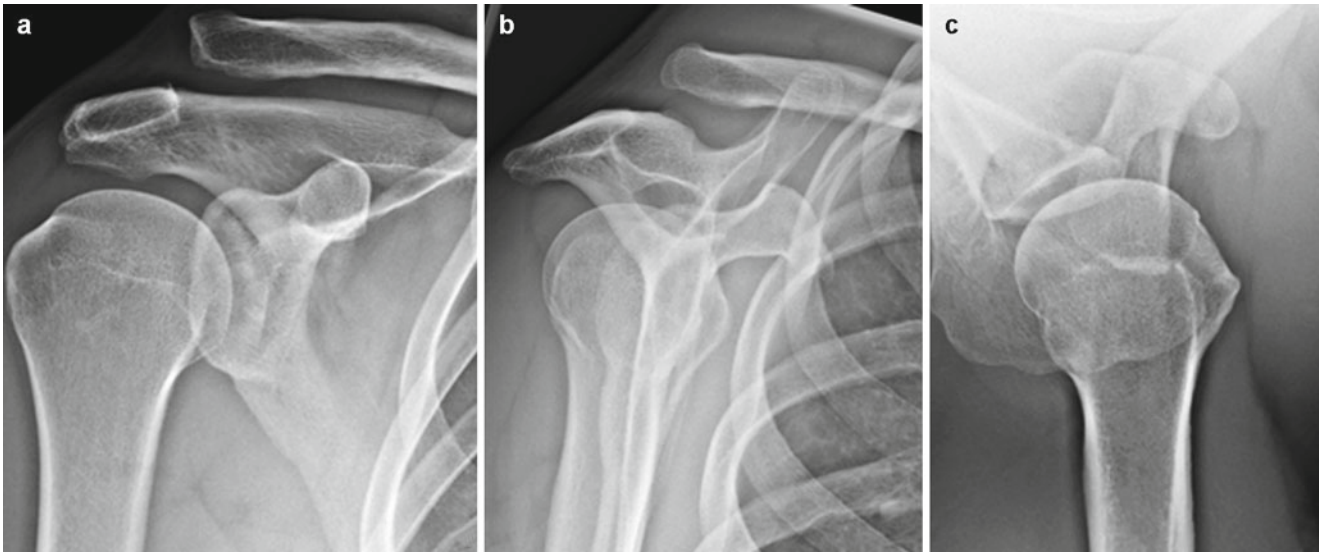


Fig. 45.2 Anteroposterior view (**a**), Y-view (**b**), and axial view (**c**) showing the severe displacement of the lateral clavicle, corresponding to a Rockwood type V lesion

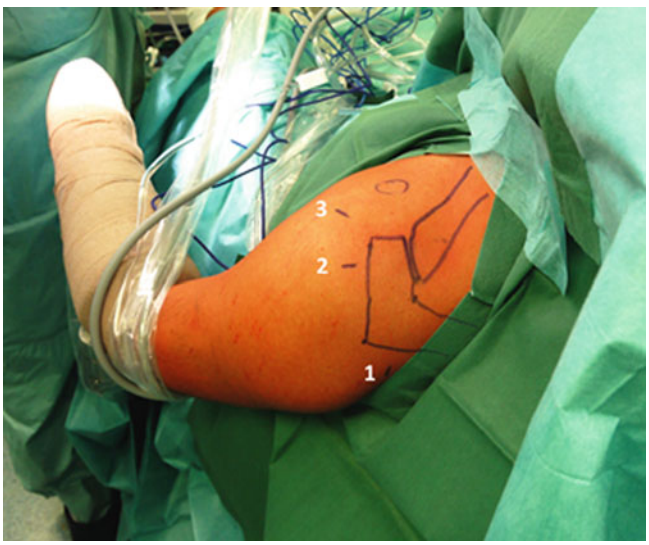


Fig. 45.3 Preoperative photograph showing the standard portals marked on the skin: *1* posterior portal, *2* lateral viewing portal, *3* anterolateral working portal

Discussion of the risks, benefits, and alternatives of each therapy modality was undertaken, and largely due to the high-grade instability, the patient decided to undergo AC joint reconstruction. Diagnostic arthroscopy revealed no concomitant injuries and the AC joint was repaired as described below.

Arthroscopic Treatment: Surgical Technique

Improvement of instruments and techniques within the last decade has enabled the orthopedic surgeon to perform acromioclavicular reconstructions mainly arthroscopically. Today, arthroscopic anatomic reconstruction techniques of the CC ligaments are state of the art. Typically, tendon grafts [42–44] and/or suture button devices [43–47] are used to reduce and stabilize the AC joint. Recent biomechanical studies have shown excellent mechanical properties for either technique [42, 47]. The authors' preferred techniques for arthroscopic anatomic AC reconstruction will be described in this section.

Patient Positioning

For the arthroscopic techniques, the patient is placed in the beach chair position. A mechanical arm holder (Trimano, Arthrex Inc., Naples, FL, USA) is used for easier management. After general anesthesia is induced, a thorough examination of both shoulders is performed. The operative shoulder is then prepared and draped in a standard fashion. The anatomic landmarks are marked on the skin after reduction of the AC joint with the mechanical arm holder (Fig. 45.3).

Portals

Diagnostic arthroscopy is performed through a standard dorsal viewing portal. A working portal is established under arthroscopic visualization through the rotator interval as a modified anterolateral portal with a spinal needle parallel to the subscapularis tendon. The arthroscope is switched to a lateral trans-supraspinatus viewing portal dorsal to the long head of the biceps tendon. Additional portals may be needed to address any concomitant intra-articular lesions, e.g., SLAP lesions. The deep anterolateral portal is secured with a

flexible cannula (PassPort Cannula 8 mm×4 cm, Arthrex) and is used to expose the base of the coracoid (Fig. 45.3).

Diagnostic Arthroscopy: Understanding and Recognizing the Pathology

In patients with AC joint separations, the main pathology is located extra-articularly. However, since a high number of concomitant intra-articular lesions have been described for AC joint separations [19], a thorough diagnostic arthroscopy of the entire glenohumeral joint is mandatory. Especially in high-grade injuries, an incidence of SLAP lesions up to 20 % has been reported, and moreover, rotator cuff tears have been found.

Step-by-Step Procedure (Box 45.1)

After the diagnostic arthroscopy and addressing potential concomitant lesions, the focus is moved towards the actual reconstruction of the AC joint.

Box 45.1: Tips and Tricks

Arthroscopic stabilization of the AC joint can be challenging even for the experienced arthroscopic surgeons. The following recommendations can aid to facilitate the procedure and avoid complications:

- Make sure you are familiar with the instruments and implants needed for this procedure.
- Use additional portals for optimum visualization.
- Avoid dissection medial to the coracoid process in order not to jeopardize the brachial plexus.
- Allow enough time for a thorough subcoracoid debridement and exposure of the coracoid process which will then facilitate the rest of the procedure.
- Avoid tunnel placement within the coracoid process too close to each other and too close to the cortex in order to minimize risk for fracture and breakout of the tunnels.
- Use fluoroscopy to control the position of the drill tip guides before over-reaming.
- Control AC joint reduction and position manually and under fluoroscopy.

Coracoid Exposure

A trans-articular approach is used for exposure of the coracoid process through the rotator interval. The coracoid process is identified just anterior to the subscapularis tendon. For better visualization, the arthroscope is switched to a trans-subscapularis viewing portal as described above. A radio-frequency device is used through the deep anterolateral portal to open the anterior joint capsule and expose the coracoid process from the tip to the base by removing soft tissue carefully with a radio-frequency device. The attachment of the pectoralis minor and the conjoined tendons is preserved. Dissection medial to the coracoid process is avoided not to injure the neurovascular structures. This step has to be performed thoroughly since good exposure and visualization of the subcoracoid space is mandatory for the following drilling and placement of any implant or graft.

Superior Approach to the Distal Clavicle

For exposure of the distal clavicle, a 3–4 cm skin incision is made within Langer's lines perpendicular to the clavicle approximately 40 mm medial to the AC joint. Next, the trapezius-deltoid fascia is exposed and incised in line with the fibers of the trapezius muscle and the clavicle. Thus, the anterior and posterior cortical margins of the clavicle can be exposed. The AC joint capsule is carefully mobilized elevating the anterior and posterior flaps subperiosteally as a single layer. By doing so, one facilitates the later repair of the joint capsule over the reconstructed AC joint. The AC joint can now be directly visualized.

Tunnel Placement

For arthroscopic anatomic AC joint reconstruction, two suture button devices (TightRope, Arthrex) are used in order to separately reconstruct the conoid and trapezoid ligaments as previously described [45, 48]. Two 4 mm drill holes are established through the clavicle and coracoid according to the attachments of the native CC ligaments as described by Rios et al. [11] and Salzmann et al. [48]. This step is performed under direct visualization from intra-articular using a special drill guide, which is inserted through the anterolateral portal. A 2.4 mm drill tip guide is placed approximately 4.5 cm medial of the AC joint transclavicular and close to the base of the coracoid. After this, a second 2.4 mm drill tip guide is introduced in the same way with the drill guide with approximately 2 cm distance lateral in the clavicle and lateral in the coracoid. Correct positioning of the two drill tip guides is verified under fluoroscopy with a C-arm (Fig. 45.4). Subsequently, the 2.4 mm drill tip guides are overdrilled starting medial with a cannulated 4 mm drill while protecting the tip of the 2.4 mm drill with a drill stop or curette. A SutureLasso wire loop (Arthrex) is inserted through the cannulated drill bit before it is removed. The second lateral 2.4 mm drill tip guide is overdrilled next and the cannulated drill is left in place.

Button Placement

The two suture button devices can now be inserted through the superior approach by the use of the wire loop, again

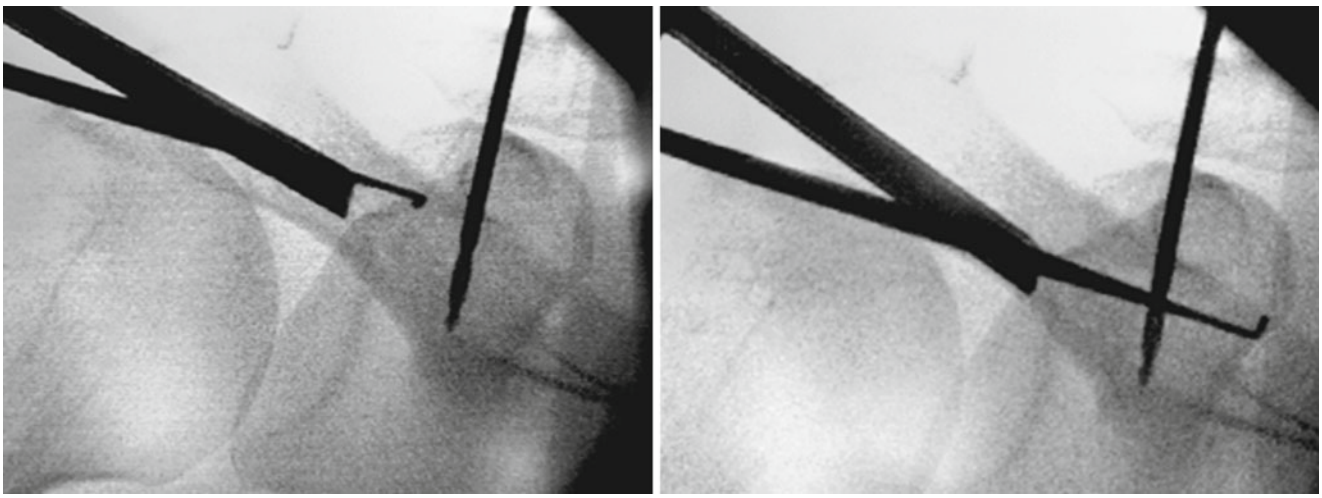


Fig. 45.4 Intraoperative fluoroscopy used to verify correct positioning of the two drill tip guides. A hooked probe is used to verify the lateral (*left*) and medial (*right*) borders of the coracoid process

starting medial. The SutureLasso is then introduced in the 4 mm cannulated drill that is still in position and the second suture button device is pulled in. Correct placement of the implants under the coracoid is controlled by direct visualization (Fig. 45.5). The AC joint is then manually reduced by elevating the arm against the scapula. When anatomic reduction is achieved, the clavicular buttons are placed on top of the clavicle. The medial and finally the lateral device is tightened using the pulley system and secured by alternating knots (Fig. 45.6).

Tendon Graft Augmentation

In revision or chronic AC joint separations, an autologous tendon graft augmentation is performed in order to add stability and enhance biological healing. For this reason, we prefer to use the gracilis tendon as a graft. The graft is typically harvested from the ipsilateral knee in a standard fashion and prepared with sutures on both ends.

The technique is modified, and we use FiberTape (Arthrex) with bigger buttons for the medial tunnels to support the graft in this tunnel. Therefore, the graft and one limb of FiberTape are pulled in the tunnel and out of the anterolateral working portal. Outside the joint, the bigger button (Dog Bone, Arthrex) is clipped on the FiberTape, and the free limb is pulled back superior in the joint and back through the coracoid and clavicular tunnel. A second Dog Bone button is used superior of the clavicle. The graft itself is pulled out with a grasper that is introduced anterior of the clavicle and finds the graft sutures lateral of the coracoid. By doing so, the graft forms a figure of eight with one limb through the bony tunnels and one around. The graft is then knotted around the clavicle and secured under tension with additional sutures, and the free ends of the graft are cut off.

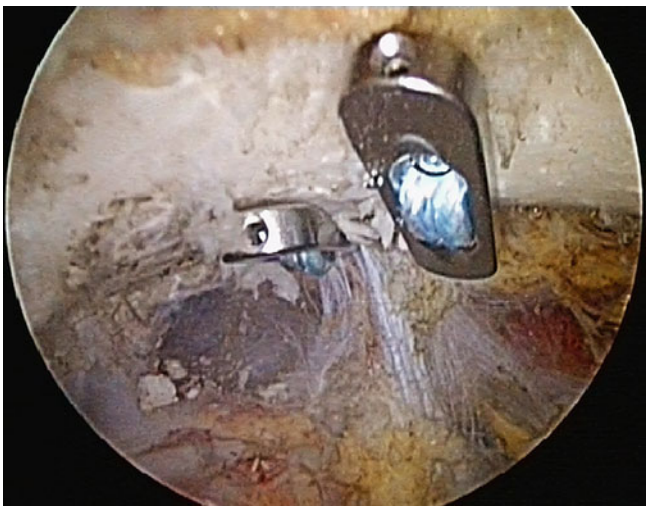


Fig. 45.5 Intraoperative view through the lateral portal of a right shoulder showing the base of the skeletonized coracoid process with the two button devices in correct position

Finally, the trapezius-deltoid fascia and the joint capsule are repaired meticulously and the skin is closed in a standard manner.

Postoperative Care

The shoulder is immobilized in a sling for 6 weeks postoperatively to minimize strain on the CC ligament reconstruction. Patients are allowed full active elbow, wrist, and hand exercises. Within the first 2 weeks, passive motion exercises are performed limited to 30° of flexion and abduction as well as to 80° internal rotation and 0° external rotation. Within weeks three and four, range of motion exercises are performed up to 45° flexion and abduction in an active-assisted

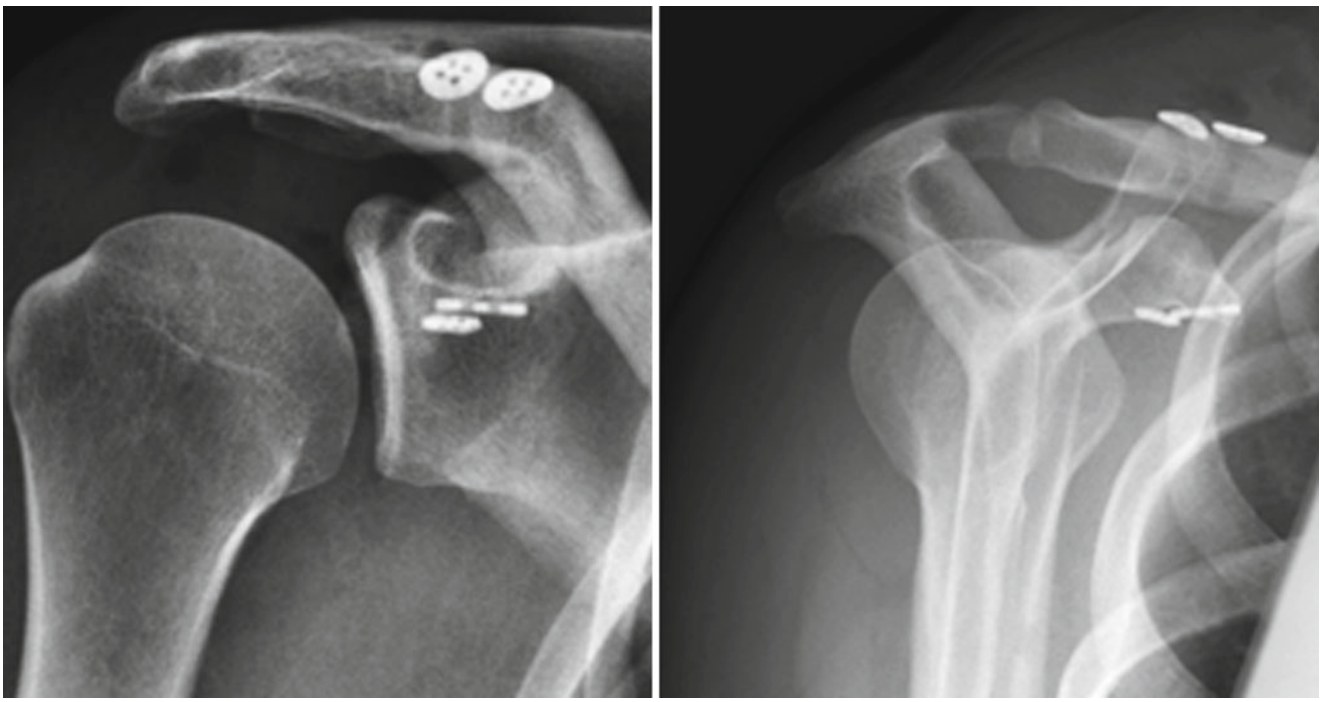


Fig. 45.6 Postoperative radiographs showing anatomic reconstruction of the AC joint and the TightRope devices in correct position

manner. Within weeks five and six, the range of motion exercises are advanced to 60° of flexion and abduction with an unlimited rotation. Active motion in the upright position is then advanced per the patient's tolerance. After regaining pain-free full active range of motion, strengthening exercises, which primarily focus on scapula stabilization, can start around the twelfth postoperative week. Return to work without any restrictions is typically allowed at 12–16 weeks after surgery. The patients are usually allowed to go back to full-contact athletics after 5–6 months, assuming the range of motion and strength are within 90 % of the unaffected shoulder [4].

Literature Review

Since Weaver and Dunn [49] published their popular technique in 1972, a vast number of different open and arthroscopic techniques have been described for surgical treatment of AC joint reconstruction. In order to improve the techniques and decrease the reported high failure rates, which were reported to be as high as 30 % [40, 49], new techniques have been evolved continuously. Furthermore, several biomechanical studies have been conducted showing the advantages and downsides of current AC repair techniques [12, 47, 50–56]. In 2008, Walz et al. [47] have reported on the biomechanical strength of an anatomic suture button repair, which showed comparable stability to the native ligaments. Also, excellent biomechanical properties have been shown for different graft reconstruction techniques [42, 52, 54, 55, 57, 58].

Anatomic reconstruction techniques have already shown good clinical outcomes; however, high complication rates have also been described [44, 45, 59, 60]. Since these techniques typically use tunnels through the coracoid and/or the distal clavicle for suture button or graft fixation, complications like fractures of the coracoid process or the clavicle have been described [44, 45, 61]. Coale et al. [62] showed in a recent CT-based study that an anatomic graft reconstruction with transclavicular-transcoracoid drilling (6 mm) significantly increases the risk of cortical breach and fracture of the coracoid process and in some cases may be not feasible. These finding are supported by recent clinical studies showing high complication and failure rates when the small coracoid process is weakened by a 6 mm drill hole [44, 60]. Using an anatomic double TightRope suture button technique, Scheibel et al. [46] reported on good to excellent early clinical results (mean follow-up: 26.5 months) in 37 patients without any coracoid fracture or early loss of reduction (within 6 weeks). Using a similar technique, Salzmann et al. [45] showed satisfactory clinical results in 23 patients with acute AC injuries after at least 24-month follow-up. However, they described a revision rate of 11.5 % (3/26) in this first published series of the double TightRope technique. Causes for revision surgery included one coracoid fracture, one cranial button slippage, and one wound infection. The caudal migration (four patients) or breakout (one patient) of the clavicular buttons,

noted in 22 % of the patients, has not shown to have a negative influence on the clinical outcome.

Summary

AC joint separations are common injuries of the shoulder girdle and numerous treatment options have been proposed in literature. Low-grade injuries (types I and II) should be initially managed nonsurgically. Surgical treatment is typically reserved for high-grade lesions (types IV through VI) and might be beneficial in some type III lesions for heavy laborers or high-level athletes. Due to recently published biomechanical data, there is a current trend towards an anatomic reconstruction of the CC ligaments. While some reports have shown encouraging results using these techniques, relatively high complication rates have been reported at the same time. Moreover, anatomic reconstructive techniques have introduced a new complication profile including migration of suture buttons and coracoid or clavicle fracture. Therefore, the ideal technique for AC joint reconstruction has yet to be firmly established.

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Scapulothoracic Disorders

46

John B. Hubbard and Gary G. Poehling

Epidemiology

Subscapular bursitis or "snapping scapula syndrome" is an inflammatory condition of the subscapular bursae that results in pain and crepitus. It was originally described by Boinet [1] and Milch [2], who noted an abnormal forward curvature of the superomedial border of the scapula as a possible culprit. Morse [3] later described two causes of subscapular irritation: soft tissue and bone. The soft tissue component consisted mostly of bursitis or muscle irritation, while the bony causes dealt more with articular incongruence of the scapulothoracic joint. Osteophytes, abnormal scapular shape, or space-occupying lesions can all result in compromised scapulothoracic articulation.

Today, subscapular bursitis is seen as an uncommon condition. For example, shoulder impingement, arthritis, rotator cuff tears, and labral pathology are all seen more commonly. Often, subscapular bursitis is found in the setting of concurrent pathology. For example, pathology relating to the glenohumeral joint and subacromial space such as rotator cuff tears can cause symptoms of subscapular bursitis. Furthermore, pain or weakness in the shoulder area can contribute to scapular dyskinesia which can cause subscapular bursitis. Tightness or contracture of the shoulder capsule and surrounding structures may contribute as well. Finally, other conditions well outside of the shoulder anatomic area such as cervical pathology or diseases of the chest wall can also result in retroscapular symptoms.

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Pathophysiology

Normal Anatomy

The scapulothoracic joint consists of the concave anterior aspect of scapula articulating with the thoracic wall. It accounts for one-third of total shoulder motion, while the glenohumeral joint accounts for the remaining two-thirds. The scapulothoracic joint helps to position and stabilize the glenohumeral joint in space.

The structures around and within the scapulothoracic articulation are richly innervated and vascularized (Fig. 46.1). The suprascapular artery and nerve traverse through the suprascapular notch laterally. The transverse cervical artery and dorsal scapular artery run along the medial border of the scapula.

The serratus anterior and subscapularis are found between the scapula and thoracic wall. Two spaces, the subscapularis space and the serratus anterior space, exist between the scapula and chest wall.

Two major (scapulothoracic and subscapularis) and four minor bursae have been described (Fig. 46.2). The major bursae are reproducibly found in their respective locations on anatomic dissection and are the two most common bursae to become inflamed [4]. The scapulothoracic or infraserratus bursa lies between the serratus anterior muscle and chest wall, along the superior angle of the scapula. This bursa is most commonly symptomatic. The subscapularis or supraserratus bursa is located between subscapularis and serratus anterior muscles [5]. This bursa is more laterally positioned and less commonly a source of symptoms. The minor bursae are adventitial and not consistently seen in cadaveric models. They typically arise in the setting of abnormal or pathological mechanics. Of these, the inferior scapulothoracic bursa, when present, can be a cause of pain in patients with subscapular complaints. The scapulotrapezial bursa lies at the base of the scapular spine medially and is also thought to contribute to pain [6].

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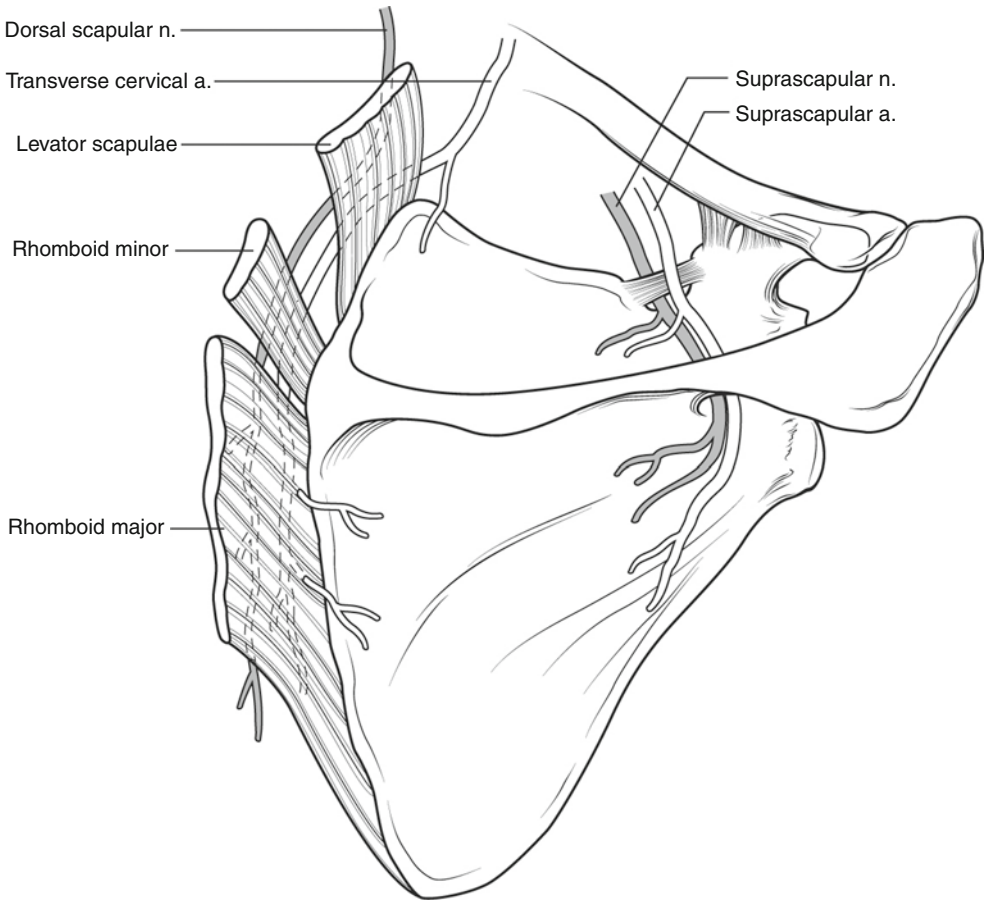

Fig. 46.1 Normal scapular anatomy (From [4]. Copyright: Arthroscopy; published by Elsevier 2009. Reproduced with permission)

The causes of pain and crepitus with scapulothoracic motion are many and include both soft tissue and bony abnormalities. As discussed above, bursitis can develop in a variety of anatomic locations, resulting in pain and crepitus. An osteochondroma of the scapula, Luschka tubercle, fractures of the ribs or scapula, postural incongruity from kyphosis or changes in thoracic alignment, and scapular dyskinesia can all cause secondary subscapular bursitis.

Oftentimes, it can be difficult to pinpoint an exact mechanical or pathoanatomic reason for periscapular pain [7]. In such cases, the diagnosis of snapping scapula is made on patient history and clinical findings.

Soft Tissue Abnormalities

Bursitis can be the result of muscle imbalance and soft tissue malalignment. Shoulder stiffness can result in scapular dyskinesia which can lead to bursitis and pain along the medial border of the scapula and the scapulothoracic articulation. Additionally, soft tissue tumors can lead to bursitis. Elastofibroma dorsi, for example, is a slow-growing tumor most commonly seen in elderly women, which occurs in the subscapular and infrascapular areas [8]. Advanced imaging such as MRI can be especially helpful if there is a concern for soft tissue mass.

Bony Abnormalities

Osteochondromas are the most prevalent benign tumor of the scapula [9]. Lesions of the undersurface of the scapula may cause pain or crepitus in conjunctions with scapulothoracic articulation. The superomedial aspect of the scapula may have a prominence (Luschka tubercle) or an excessively hooked surface. Old, malunited fractures of the scapula or ribs or postural changes of the thoracic spine such as kyphosis can lead to crepitus and secondary bursitis as well.

History

Patients with subscapular bursitis will often complain of pain at the superior and/or inferior angles of the scapula, as well as pain medial and deep to the scapula. The pain is often activity related and associated with crepitus and may occur more often with overhead motion or activity. Oftentimes, patients will complain of pain that is worse at night. The onset is typically insidious, may also occur after change in

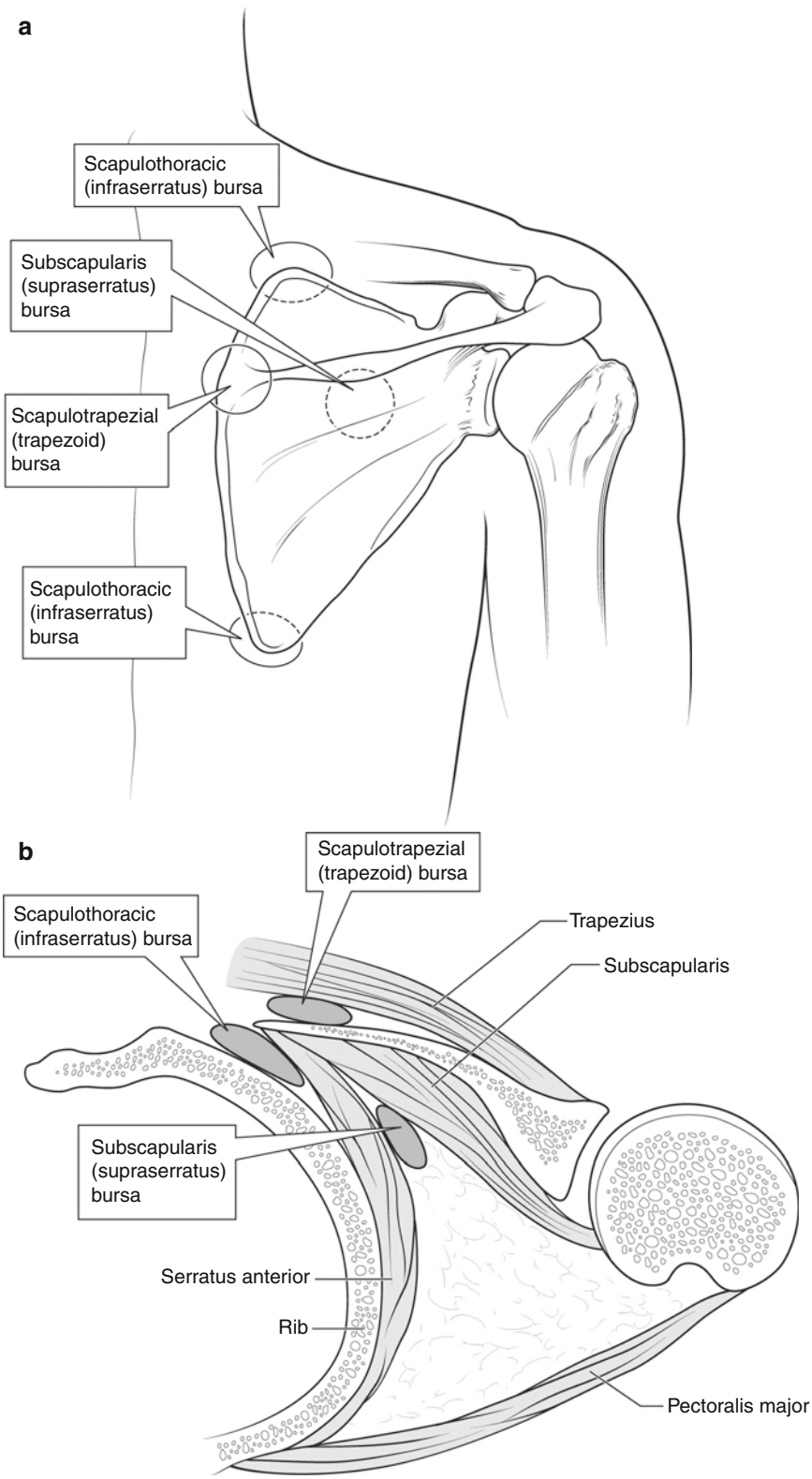

Fig. 46.2 The subscapular bursae. (**a**) Posterior coronal view. (**b**) Cross-sectional view (From [4]. Copyright: Arthroscopy; published by Elsevier 2009. Reproduced with permission)

activity, or may be associated with trauma [10]. Interestingly, many patients may have crepitus with no pain. In this setting, the symptoms are not necessarily deemed pathological, and a conservative course is favored.

Athletes involved in repetitive motion, especially overhead, may be at risk. Sports such as baseball, swimming, gymnastics, and weight lifting in particular seem to be susceptible [11].

Many patients will present with a component of chronic pain. It is not uncommon for patients to have seen a number of medical providers prior to orthopedic evaluation, and long-term narcotic use can be prevalent.

In addition to subscapular symptoms, it is important to inquire about neck and glenohumeral complaints as well. Any numbness, weakness, impingement, or biceps pathology, for example, should be worked up. Complaints such as new onset shoulder stiffness (adhesive capsulitis) or radicular symptoms (cervical pathology) may cause secondary bursitis.

Clinical Examination

The clinical exam begins with inspection. It is important to examine both sides of the patient, preferably without overlying garments. Muscular tone is inspected, and any asymmetry is noted. The presence of fullness or swelling should alert the examiner to areas of inflammation, muscular spasm, or even a possible mechanical obstruction such as an osteochondroma. Any tenderness to palpation is documented, paying special attention to the superior and inferior angles of the scapula. The medial border of the scapula may be isolated by extending and internally rotating the shoulder, which makes the medial border more prominent. Crepitus is a common finding, even when patients do not complain of pain. Painless crepitus in the absence of other symptoms is not a reliable sign of subscapular bursitis. The spine should be carefully inspected for increased thoracic kyphosis and poor posture.

Shoulder strength and motion is tested and compared to the contralateral side. Careful attention is paid to kinematic abnormalities. The scapular position in space and response to cardinal motions of the shoulder are noted. Careful attention is paid to the presence of scapular winging. The patient is tested for both medial winging (serratus anterior weakness) and lateral winging (trapezius weakness). The patient is asked to lean into a wall with both shoulders flexed to 90° and push. Many patients will have a degree of scapular dyskinesia, and this is documented. Any side-to-side difference in scapular mechanics should be identified. Many instances will respond to focused physical therapy and conservative management.

Specific muscle groups may be isolated and tested. Strength testing is carried out for trapezius by shrugging against resistance. The rhomboids and levator scapulae are tested by having the patient put her or his hands on her or his hips and then actively push elbows backward against resistance. As mentioned above, the serratus anterior is tested by examining for scapular winging while pushing against a wall (Fig. 46.3). Finally, weakness of the latissimus dorsi is evaluated by downward/backward shoulder motion while palpating the inferior angle of the scapula [12].

The area of interest is then palpated. Special attention is paid to any fullness or swelling. The location of any tenderness to palpation (muscular, fascial, bony) is documented.

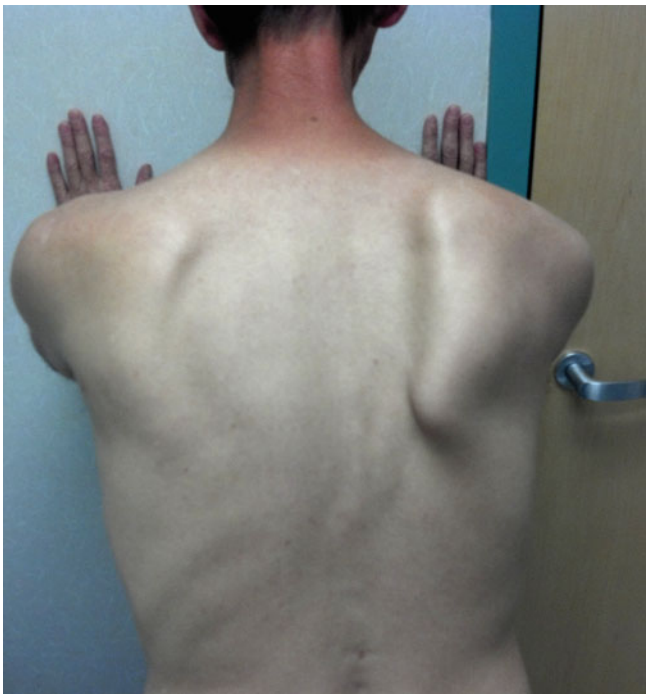

Fig. 46.3 Scapular winging

Adduction and internal rotation of the shoulder can allow for full examination of superomedial and inferomedial angles and medial border of the scapula.

Injections can be a helpful modality. A steroid injection combined with a local anesthetic can offer diagnostic and therapeutic value. Immediate relief of pain after a targeted subscapular steroid injection can help confirm the diagnosis. Additionally, patients may get long-term pain relief from the steroid. Steroid injections combined with a focused physical therapy program can offer significant relief.

Imaging

Different imaging modalities can be helpful when treating patients with subscapular bursitis. These include x-rays, computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound (US).

Imaging work-up for a patient complaining of subscapular bursitis begins with standard shoulder radiographs. A shoulder x-ray series, including anteroposterior (AP), true AP, scapular Y, and axillary views, is obtained. Bony abnormalities such as a subscapular osteochondroma or Luschka tubercle may be detected. Additionally, other causes of shoulder pain may be apparent such as abnormal acromial morphology and arthritis of the acromioclavicular and glenohumeral joints. A cervical spine x-ray series may be helpful for patients complaining of neck pain or cervical complaints.

CT can be helpful when concerned about bony causes of posterior shoulder pain. CT can clearly define lesions such as a subscapular osteochondroma as well as help with preoperative planning. When bony pathology is not suspected, CT scans are not routinely obtained.

The usefulness of MRI in the setting of subscapular bursitis is debated. Increased signal in the superior medial or inferior medial scapula may be present on T2-weighted sagittal images. Fluid within the subscapular bursae may also be present. Finally, MRI may show less common reasons of subscapular bursitis, such as an elastofibroma or osteochondroma. MRI of the shoulder or cervical spine may be helpful to evaluate for pathology in these neighboring areas. If the clinical picture is clear and there are no signs or symptoms of glenohumeral or cervical pathology, an MRI is not routinely ordered.

US can be a useful imaging modality for shoulder pathology. US offers distinct advantages over other imaging modalities in that it can be done in a static or dynamic fashion. Specific muscle groups can be isolated and tested by the surgeon herself or himself, with real-time feedback. It can often be performed in the clinician's office rather than an outpatient imaging center and so has the added benefit of convenience. Subscapular bursitis is still a fairly new application for US, and at this time its efficacy has not been fully elucidated.

Treatment: Indications and Contraindications

The initial treatment for subscapular bursitis is nonoperative. A multifaceted approach is utilized, typically for a 4–6-month time period. Most patients get better with the following nonoperative management.

Physical therapy is an important component in the nonoperative treatment of subscapular bursitis. Scapular dyskinesia is very commonly seen in this condition, and therapy is focused on restoring normal scapular mechanics. Specific muscle groups such as the serratus anterior or trapezius can be isolated and worked up. Periscapular muscle strengthening focuses on improving the ability of the subscapularis and serratus anterior muscles to elevate the scapula off of the chest wall [5]. Strengthening of the serratus anterior will also help to keep the scapula from tilting forward and restore normal scapular mechanics.

Therapy is also focused toward strengthening and conditioning of core muscle groups and improvement of posture. The cessation of any offending activity is important as well. Activities such as overhead lifting and sitting for a long time in certain positions may exacerbate this condition. Therapy and work hardening activities can focus on ameliorating these exacerbating traits.

Different medications can be used during the nonoperative period as well. A short trial of nonsteroidal anti-inflammatory drugs (NSAIDs) can be helpful in curbing the inflammatory component of bursitis. Selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs) are also commonly used. It is important to note that chronic pain syndrome is commonly seen in this patient population, and it is not uncommon for patients to have been on chronic pain medicine including narcotics previously.

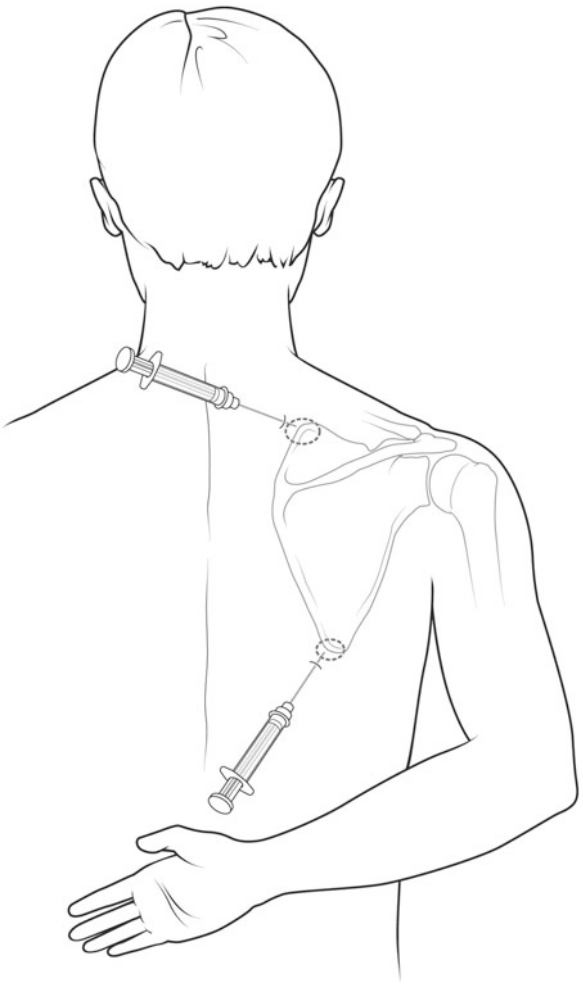

Fig. 46.4 Location of steroid injections into infraserratus bursae ([From [4]. Copyright: Arthroscopy; published by Elsevier 2009. Reproduced with permission)

Subscapular steroid injections can be a useful adjunct to nonoperative care. It is important to clearly define where the source of pain is; typically, it is associated with the superior or inferior scapular angles. After determining the location of pain, the patient is placed in the prone position, and the shoulder is internally rotated and extended into the "chicken wing" position. This is the same position used for arthroscopy and exaggerates the medial border of the scapula. The injection is then directed toward the symptomatic bursa, most commonly the superior or inferior infraserratus bursae (Fig. 46.4). Care is taken not to direct the needle too deeply as pleural damage, bleeding, or even a pneumothorax could result.

When used in conjunction with NSAIDs, steroid injections, physical therapy, and activity restrictions, we have had significant success with these medications. We do not routinely use narcotics in the nonoperative setting of subscapular bursitis.

Surgery for subscapular bursitis is indicated after a 4- to 6-month period of nonoperative treatment. The patient should consistently complain of pain that is related to a subscapular bursa. If an injection was utilized, it is reassuring if it provided at least temporary relief.

It is important to exclude other sources of pain which may mimic subscapular pathology or result in referred pain to the area. The examiner should investigate for cervical pathology. In particular, C7 radiculopathy can cause medial scapular pain which may mimic the symptoms of subscapular bursitis. Glenohumeral pathology can be the cause of posterior shoulder pain as well, both directly and indirectly. Thorough examination of these areas, including radiographic work-up if indicated, should be performed.

Other contraindications to surgery include persistent abnormal shoulder mechanics, refusal to participate in therapy, and secondary gain. Much like volitional shoulder dislocators, patients who volitionally snap their scapulae should be approached with trepidation.

Decision-Making Algorithm

Once the patient has completed a full course of therapy and failed medical management, subscapular bursoscopy is considered. Both open surgery and bursoscopy are efficacious, although we prefer endoscopic surgery and have largely abandoned open procedures for this problem. Surgery involves a thorough bursoscopy, followed by bursectomy and bony removal of implicated structures. Any bony prominence such as an osteochondroma can then be removed.

If the patient has concurrent glenohumeral or subacromial pathology, then a standard shoulder scope can be performed in the same setting. Typically, this will be performed first, either in the lateral or beach-chair position. The patient can then be re-prepped and draped for the subscapular portion of the case.

Clinical Case/Example

A 20-year-old female presented with activity-limiting left-sided superomedial scapular pain associated with crepitus. It bothered her all the time; her pain was worse with overhead motions and at night. She was otherwise healthy.

On exam, she was found to have full range of motion with superomedial scapular pain and audible crepitus, with no winging and no scapular dyskinesia. She was neurovascularly intact distally.

Arthroscopy of the subscapular space showed a bony excrescence causing impingement along the superomedial angle. This was decompressed with a shaver and burr.

Postoperatively, the patient was placed in a simple sling and advanced motion to tolerance. We limited resistive activities for 4 weeks.

Arthroscopic Treatment: Surgical Technique

Patient Positioning

The patient is positioned prone, and the operative arm is draped free with a stockinette. Extension and internal rotation (the "chicken wing" position) are utilized to exaggerate bony landmarks, including the medial border of the scapula and superior and inferior angles. The acromion is palpated and bony landmarks are marked. The arm is placed in the chicken wing position, and oftentimes a non-penetrating clamp can be used to hold the arm temporarily in place by clamping the stockinette to the drapes (Fig. 46.5). The surgeon and assistant stand on the contralateral side of the operating table (i.e., left side for a right shoulder). The viewing screen and arthroscopic towers are placed on the operative side of the OR table (Fig. 46.6).

Portals

Establishing the portals in subscapular arthroscopy can be challenging. This should be done with extreme care, given the neighboring neurovascular structures and chest wall. First, both medial portals are established concurrently. The viewing portal is established three fingerbreadths medial to the medial border of the scapula, just below the level of the scapular spine. The working portal is established inferior to the viewing portal at the midpoint between the scapular spine and inferior scapular angle, again three fingerbreadths medially (Fig. 46.7). By keeping the portals medial to the medial scapular border and inferior to the scapular spine, the dorsal scapular neurovascular structures are protected (Fig. 46.8a). Switching sticks or blunt trocars can be helpful to develop the potential space as well as triangulate. A 4 mm 30° arthroscope is used with 60 mmHg pump pressure. This may need to be elevated periodically throughout the case if bleeding is encountered, although we do not recommend using elevated pump pressure for significant amount of time.

A third portal can be established with an inside-out technique to help with visualization and triangulation in

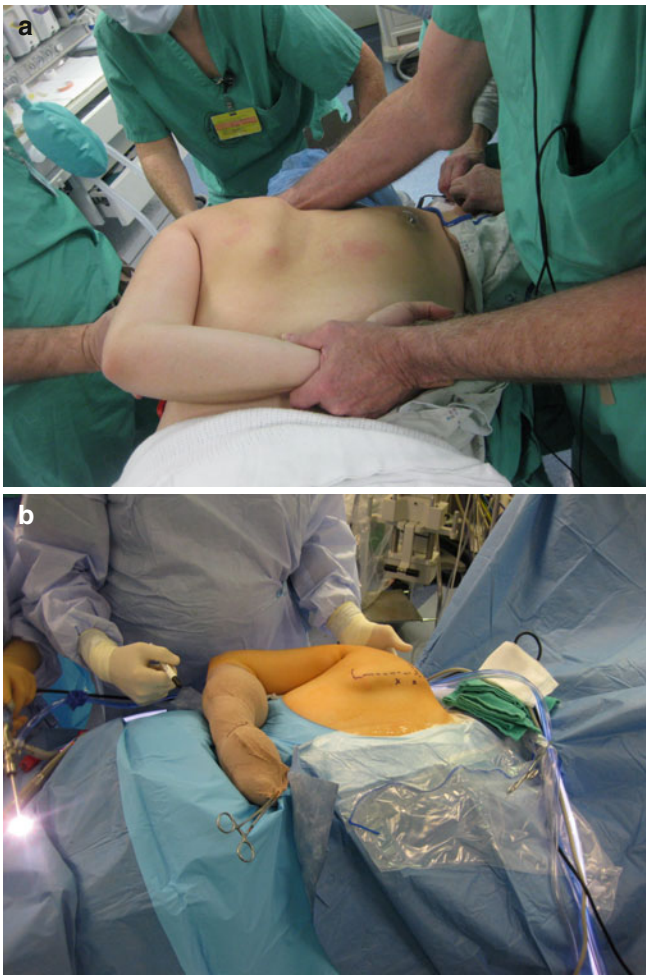

Fig. 46.5 (**a**) The patient is positioned in the prone position. The arm is placed in the "chicken wing" to exaggerate bony landmarks, including the medial border of the scapula and superior and inferior angles. (**b**) The field is draped widely to allow for full access. A non-penetrating clamp can be used to hold the arm temporarily in place by clamping the stockinette to the drapes with the operative extremity draped free. Bony landmarks are marked

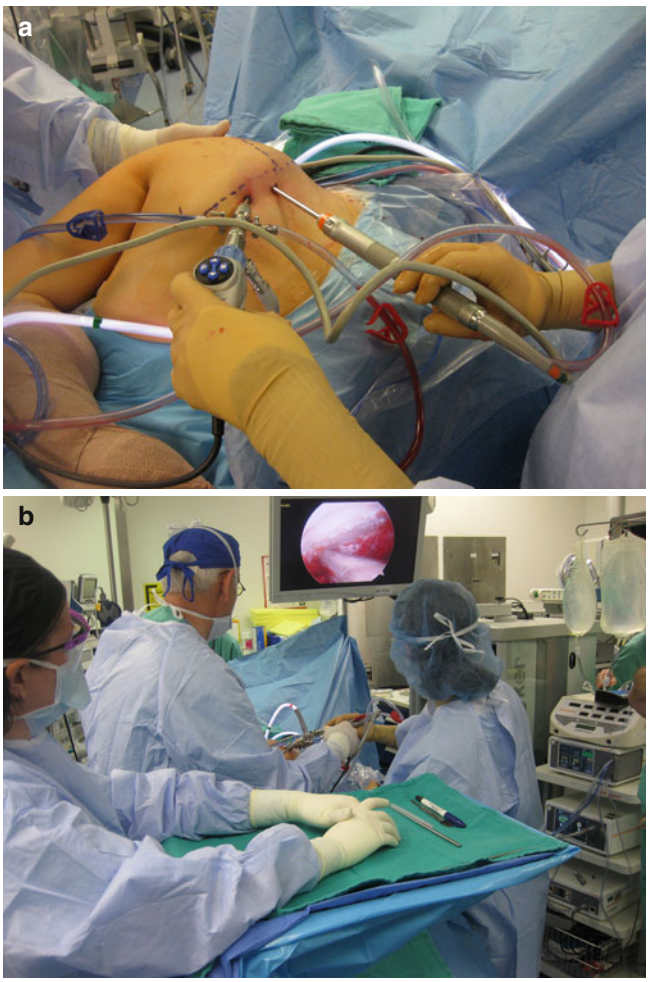

Fig. 46.6 Arthroscopic room setup. (**a**) The surgeon and assistant stand on opposite sides. (**b**) The viewing screen and arthroscopic towers are placed superiorly on the operative side of the OR table

the operative space. The portal is located on the superior border of scapula, one-third of the distance from the superomedial angle of the scapula to the acromion (Fig. 46.8b) [12].

Step-by-Step Procedure (Box 46.1)

Once the arthroscopic procedure is initiated, the bordering structures are ribs and intercostal muscles inferiorly, subscapularis muscle laterally, and rhomboids and levator scapulae medially. When starting out, the space is quite small. Any exaggerated motion with the arthroscope or instrument may compromise visualization. The surgeon is

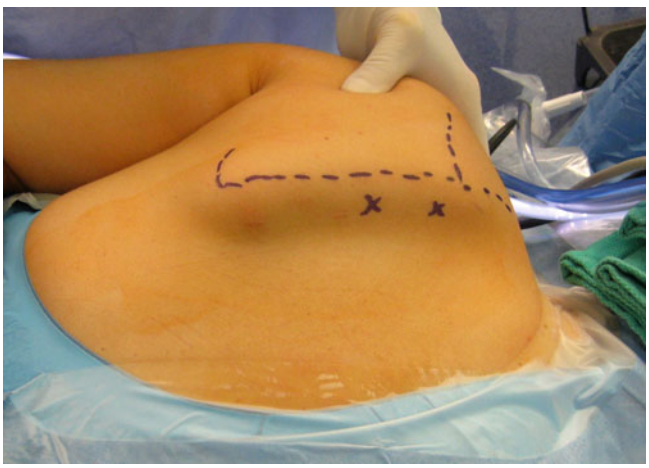

Fig. 46.7 Standard arthroscopy portals were marked on the skin 1 cm medial to the scapula starting just below the scapular spine to allow access to the superior subscapular recess

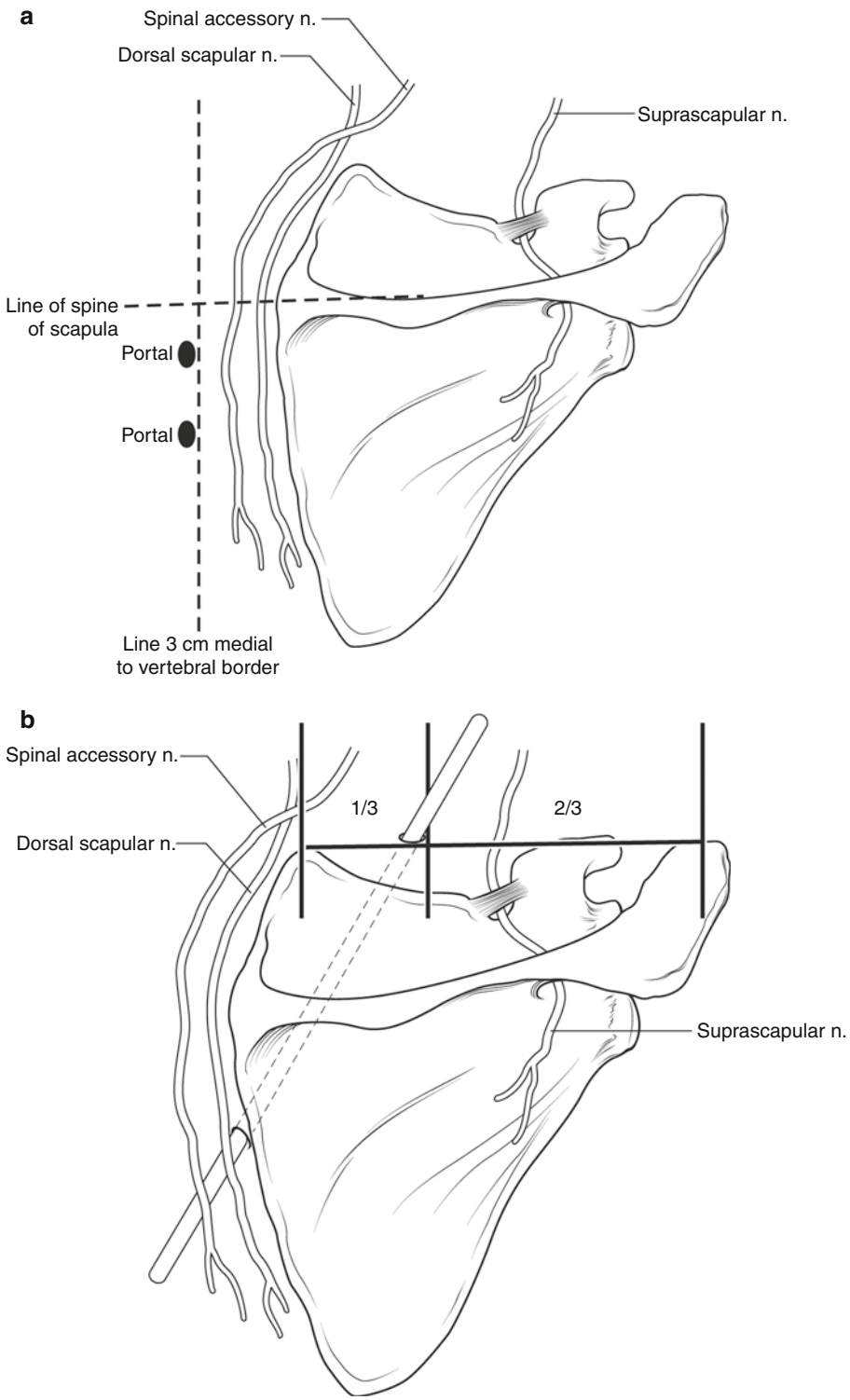

Fig. 46.8 (**a**) Standard arthroscopic portals. (**b**) Superior portal (From [4]. Copyright: Arthroscopy; published by Elsevier 2009. Reproduced with permission)

encouraged to work slowly and deliberately to define the space and identify landmarks and pathology.

Bleeding can obscure the visual field, just as in traditional glenohumeral or subacromial arthroscopy. To help avoid bleeding, the surgeon is encouraged to take as little muscle as possible and have direct visualization of instruments at all times. Occasionally increasing the pump pressure (to 80 or even 100 mmHg) can be helpful. This

Box 46.1: Tips and Tricks

- The stockinette can be clamped to the drapes using a non-penetrating clamp to hold the arm in the chicken wing position.
- When establishing the portals, blunt trocars or switching sticks can be used to develop the potential subscapular space and triangulate.
- When injecting symptomatic bursae or placing arthroscopy portals, make sure that the angle is not too steep. The trajectory should be more lateral than deep to avoid injury to underlying pleura.
- Dissect the medial border of the scapula subperiosteally to define arthroscopic position and help to develop the space.
- Avoid excessive resection of muscle fibers and deep fascia or detaching muscle insertions of levator scapulae and rhomboids.
- Avoid injury to dorsal scapular nerve/artery. Place arthroscopic portals three fingerbreadths (or 3 cm) medial to medial border of scapula and at level or inferior to scapular spine.
- Avoid injury to supra scapular nerve/artery. Do not operate too lateral; also do not operate on the vicinity of the coracoid process.
- Avoiding hazards:
  - Dorsal scapular nerve
  - Spinal accessory nerve
  - Suprascapular nerve and artery
  - Chest wall, pleural tissue
  - Axillary space

should be done for brief periods of time only. It is imperative that good communication is maintained with the anesthesiologist to try and keep blood pressure down as safely as possible. Finally, some advocate the use of epinephrine in the arthroscopy fluid, although we do not have experience with this.

The best landmark at the beginning of the procedure is the medial border of the scapula which is skeletonized of bursal tissue. Minimal muscle is resected, and careful attention is paid to not detach the muscular insertions of the rhomboids and levator scapula. Once it is clear that the surgeon is in the subscapular space, it is important to not operate medial to the scapular border or deep to the space. At this point, a third, superior portal can be developed using spinal needle and outside-in approach or inside-out technique, as described above. Triangulation with this portal is often simpler than with the first two portals, as the third portal is developed at more of a 90 angle to the viewing portal.

At this point in the procedure, any masses such as an osteochondroma should be identified and excised. A thorough bursectomy of the involved bursa is carried out. It is important to not drift laterally to avoid damage the suprascapular nerve and vessels. If orientation is obscured, the surgeon should always return to the skeletonized medial aspect of the scapula for direction.

After bursectomy, a scapuloplasty may be considered, which involves resection of the superomedial angle of the scapula. Although traditionally described as an open procedure, arthroscopic resection can be performed without significant difficulty. Instruments such as a motorized burr and an arthroscopic rasp are helpful in resection and smoothing out the remaining bone. Typically 20 × 20 mm is resected in this fashion (Fig. 46.9) [6]. The surgeon should be cognizant that the scapular bone is actually quite thin, and care should be taken to not plunge or jeopardize deep structures.

Once the scapulothoracic articulation is sufficiently decompressed, the shoulder is taken through a range of motion under direct arthroscopic visualization. The surgeon can place the shoulder and scapula in the positions that were most symptomatic to check for remaining impingement. If still present, the debridement steps are repeated.

Complications of this procedure can be quite serious. Damage to the chest wall or pleural tissue can result in significant bleeding as well as possible pneumothorax or hemothorax. Careful attention should be paid at all times to not drift deep. There are many nerves in the vicinity of the surgery which can be damaged as well. The dorsal scapular nerve is avoided by placing the medial stab incisions three fingerbreadths (3 cm) medial to the medial border of the scapula. The suprascapular nerve is avoided by keeping the dissection from too far laterally in the subscapular space.

Postoperative Care

Arthroscopic procedures allow for an earlier return to function and typically less postoperative pain. Patients recover more quickly and can usually start immediate postoperative activity. Sling for comfort, start immediate motion to tolerance; strengthening can begin at 4 weeks. Return to sport is expected at 2–3 months. Physical therapy, when needed, starts with passive motion followed by active motion and strengthening.

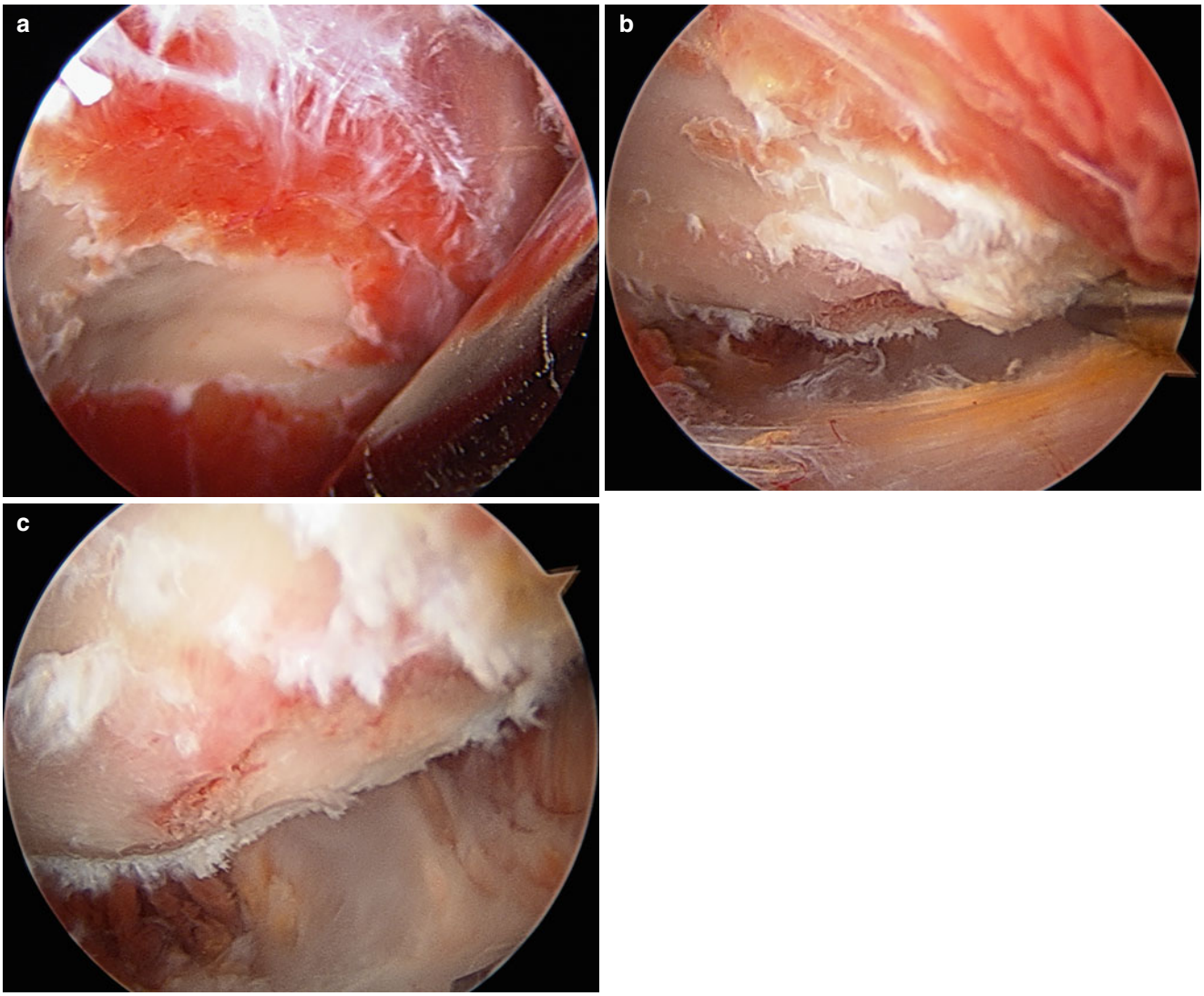

Fig. 46.9 Arthroscopic pictures show the subscapular space with a bony excrescence causing impingement along the superomedial angle. This was decompressed with a shaver and burr

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Complications in Shoulder Arthroscopy

47

Gregor Szöllösy and Bruno Toussaint

Complication (n): A secondary disease or condition that develops in the course of a primary disease or condition and arises either as a result of it or from independent causes [1].

Introduction

Shoulder arthroscopy showed a progressive technical improvement and widening of indications. This led to increased rate and change in surgical complications.

Many complications described in the early days of shoulder arthroscopy have disappeared along with improved or abandoned techniques. In the first large survey by the members of the Arthroscopy Association of North America [2], the highest complication rate was associated with staple capsulorrhaphy (5.3 %) – a procedure that to our knowledge is not usually performed anymore [3].

Overall complications of shoulder arthroscopy are rare, and even if the exact number cannot be given due to intrinsic reasons, it is safe to assume that they are well below 5 %. The reason why the number can only be an estimate is that there is no clear and widely accepted definition of what a complication is. For example, is loss of 20° of external rotation after instability surgery a complication or a part of the natural course of a shoulder capsulorrhaphy? Is it a "secondary disease" because it reduces range of motion or a "desired condition" because it prevents the shoulder from going into a position risking a dislocation? Furthermore, we must realize that there is a significant publication bias in orthopedic (and other) science [4], which is difficult to assess in its extent [5] and certainly leads to underestimating the quality and quantity of complications.

We would like to focus this chapter on the complications that are likely to be encountered in current arthroscopic shoulder surgery. Complications associated with specific procedures are discussed where the procedure is described.

Generic Complications

Infection

Although a dreaded complication due to its often devastating outcomes, infection is a rare complication. D'Angelo and Ogilvie-Harris [6] reported an infection rate of 0.23 % and recommended the use of routine antibiotic prophylaxis. Bigliani et al. [7] noted an infection rate of 0.04–3.4 %. This very low infection rate kept constant over the years and was confirmed by many other authors. Murray et al. [8] advocate the use of 2 % chlorhexidine gluconate, proving that it reduces the number of skin bacterial colonies by one-third. Randelli et al. [9] confirmed the advantage of preoperative antibiotic prophylaxis. In an unmatched series of 9,385 surgeries, the relative risk of infection was 6 times higher without antibiotics. Athwal et al. [10] observed that Propionibacterium acnes was the most common organism isolated, infecting 20 of 39 cases (51 %). Schneeberger et al. [11] showed the connection between persistent pain and subclinical infection with Propionibacterium acnes. They also pointed out the long incubation time (average: 8 days; maximum: 17 days) and the often frustrating antibiotic treatment.

Venous Thrombosis and Pulmonary Embolism

Deep vein thrombosis (DVT) and pulmonary embolism (PE) are feared complications due to their lethal potential, particularly in elderly patients. Therefore, a lot of attention has been paid to this entity. Nevertheless, the risk remains very low,

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and in-hospital anticoagulation treatment of patients having DVT or PE is effective. In the United Kingdom, the rates of DVT and PE are below 0.01 % for arthroscopic shoulder surgery and similar to a background nonsurgical population [12]. Reported rate for fracture surgery and total shoulder replacement is around 0.2–0.4 %. Chemoprophylaxis does not affect the number of events. Ojike et al. [13] reviewed 8 articles with a total of 40,000 shoulder surgeries including 16,000 arthroplasties and found an overall incidence of 0.24 % for DVT and 0.11 % for PE. Kuremsky et al.[14] reported six cases on 1,900 shoulder arthroscopies over a period of 4 years, resulting in an incidence of 0.31 %. All of these patients required in-hospital treatment. Randelli et al. [9] reported 0.6/1,000 events with no influence of chemoprophylaxis, but they were aware of a possible bias of the study due to surgeon-reported survey design.

Pain and Chronic Regional Pain Syndrome (CRPS)

Pain not only is one of the leading symptoms before shoulder surgery but is often the leading symptom after surgery and a patient's "tool" to assess the surgeon and the quality of his/her work. Although pain is individual and sometimes difficult to assess [15, 16], it is important to know which procedure will hurt how much. Stiglitz et al. [17] showed a pain peak at days 1–2 after surgery and then a constant improvement until day 30. They found rotator cuff repair being the most painful surgery and instability surgery the least painful one. Buess et al. [18] found arthroscopic surgery to be less painful than open surgery with an average pain reduction in the VAS from 8.0 to 1.6 points postoperatively. Kasten et al. [19] found that patients after arthroscopic rotator cuff repair used fewer painkillers in the early postoperative stage but had higher pain scores (VAS) in the time 4–8 weeks postoperatively compared to patients undergoing a mini-open rotator cuff repair. These results may be biased due to different types of repair in the two groups, as suture anchors and transosseous sutures were used in the arthroscopic and mini-open group, respectively. In a review paper, Lindley et al. [20] somewhat questioned these findings, stating that there only might be decreased short-term pain in patients undergoing arthroscopic repair. Sultan et al. [21] reported 18 % of unplanned hospital readmission after day-case arthroscopic surgery due to wound conditions and pain.

The complex regional pain syndrome (CRPS) is characterized by diffuse pain, edema, reduced range of motion, and changes in temperature and skin color of the affected extremity. These signs occur for the most part at the distal part of the extremity [22]. It has to be discerned from the stiff or frozen shoulder which is an inflammatory process [23]. The incidence of CRPS after shoulder arthroscopy is not known, and diagnosis of this complication is difficult [24]. It has been suggested to measure the shoulder surface temperature, but other than the scintigraphy, it has not been proven a useful diagnostic tool [25], as the skin temperature changes according to activity, daytime, and underlying pathology [26]. As little as we know about pathology and etiology, as little evidence there is for treatment options. Some authors [27–29] reported CRPS as an indication for brachial plexus block; however, recent reviews did not support this therapeutic option [22, 30, 31]. Savas et al. showed that only 10 % of the patients with CRPS can expect complete regression of symptoms and majority of patients have persisting symptoms, mostly pain [32].

Complications from Anesthesia

General anesthesia, as much as surgery, has become much safer over the decades, and severe complications are actually very rare. However, there are still devastating complications in respect to the surgery and expected outcome, which should be considered in decision-making process around a surgery. This cannot be the place to completely list all the possible complications associated with anesthesia, and we kindly refer to the specific literature dealing with the matter (see Chap. 9). Nonetheless, we would like to focus on some complications that may arise from brachial plexus block.

Block failure was noted by Weber and Jain in about 18 % of the cases [33]. Brull reported more than 90 % of successful procedures [29]. Bloc et al. obtained similar results with an infraclavicular technique [34]. The neurostimulator seems to have a positive influence on the results only if single nerves are blocked and it does not reduce the total amount of complications [35]. Indeed, the use of ultrasound is improving the results.

Phrenic nerve blocks appear in almost 100 % of brachial plexus blocks due to the close vicinity of the phrenic nerve to the plexus. The majority of patients well tolerate the hemidiaphragmatic paresis resulting from phrenic nerve block, and only 2–6 % of the patients complain of respiratory distress. Nevertheless, case reports on dramatic situations of respiratory distress [36–39] make us aware about this complication. Neuraxial blockade happens in 0.4–4 % but remains mostly transient. However, symptoms vary from loss of consciousness to respiratory and cardiac arrest. Unintentional anesthesia of the contralateral side has also been described [29, 40–42]. Intravascular injections are avoided if aspiration is performed prior to injection. The effects of local anesthetics on the central nervous system (CNS) are well known and include convulsions, seizures, and even postictal hemiparesis [43]. It must be emphasized that even local application of local anesthetics results in a dose- and time-dependent systemic distribution. Continuous interscalene infusion of bupivacaine at a rate of 0.25 $mg \times kg^{-1} \times h^{-1}$ for 48 h is considered

a safe dose [44]. Between 5 and 20 % of patients in seated position make a vasovagal response of varying degree to anesthesia. The triggers are venous pooling in upright position, catecholamines, pain, and fear. The triggers tend to cumulate. The treatment is to restore adequate venous return, the absolute circulating volume, and the early use of ephedrine [45].

Complications Associated with Shoulder Arthroscopy

Patient Positioning: Beach Chair vs. Lateral Decubitus

Best patient placement is an ongoing and probably never-ending debate, since each surgeon has her or his preferences and customs that influence the weighting of any argument towards one of the two placements. Along with Rains et al. [46], we would like to emphasize that almost all complications can be avoided if surgeon and anesthesiologist are vigilant and careful in patient positioning.

The complications that arise from patient positioning are traction nerve injuries, hypotensive-bradycardic episodes with impairment of cerebral perfusion, nerve damage through portal placement, irrigant-related complications, and intraoperative technical ones such as orientation, ease of conversion to open procedure, and ease of portal placement. In addition, Peruto et al. [47] found the beach-chair position to be much more expensive than the lateral decubitus, albeit the issue is questionable due to different operating room setups.

Overall transient nerve injuries occur in almost 10 % of lateral decubitus shoulder arthroscopy procedures, but very much less in beach-chair position [46]. They are due to arm positioning and traction used. Klein et al. [48] measured the least strain on the brachial plexus and best visibility in 45° forward flexion with 0° or 90° of abduction. Pitman et al. [49] found that patients placed in lateral decubitus position regularly had abnormal evoked potentials in their musculocutaneous nerve, but only 10 % of them had paresthesia, and they resolved after 48 h. Persistent clinical paresthesia is infrequent and restricted to lateral decubitus position. Rodeo et al. found no persistent paresthesia after beach-chair positioning [50].

Hypotensive-bradycardic episodes are actually not uncommon and were reported in up to 30 % of cases [51, 52]. Generally, these episodes are transient and have no clinical implication. However, they might produce disastrous consequences to all neurovascular structures even with permanent deficits. Pohl and Cullen [53] reported one case of death and three cases of permanent brain damage. Lee et al. [54] showed that the mean arterial pressure (MAP) and cerebral oxygenation decreased during anesthesia in beach-chair position. Papadonikolakis et al. [55] showed that blood pressure measurements are not reliable to assess brain perfusion and oxygenation. Dippman et al. [56] suggested avoiding deliberate hypotension along with beach-chair position and advocated the use of regional cerebral oxygenation measurement during anesthesia. However, Gillespie et al. [57] in a prospective study showed that the tolerance to hypotension was greater than generally believed.

Portal Placement

Nerve injuries can occur through traction but also through direct injury via portal placement. Segmuller et al. [58] described this complication in 7 % of 304 shoulder arthroscopies and stated that the lateral portal is responsible for injuring the cutaneous branches of the axillary nerve. The posterior portal must be placed carefully as it might damage the suprascapular nerve when placed too high and the axillary nerve when placed too low [59]. Lo et al. [60] showed that all the neurovascular structures are more than 20 mm away from any portal except for the cephalic vein.

Bone and cartilage injury can result from improper portal placement [61] even when using a blunt trocar. Tendon injuries due to trocar placement have been also described [62, 63].

Fractures

Overly zealous resection of the acromion can lead to its fracture and should therefore be avoided [64, 65]. There is a report of a clavicular fracture due to a misinterpretation of the anatomy [66], but even humeral shaft fractures are possible when stiff shoulders are manipulated [23], as much as greater tuberosity avulsions can occur.

Stiffness

Postoperative stiffness of the shoulder is a common and unpleasant complication, even if in most of the cases a second intervention is not necessary. The overall incidence is not precisely known and seems to be between 4 and 15 % [67], whereas in the general population it is around 2 % [23]. Transient stiffness after arthroscopic rotator cuff repair occurs in 10 % of the cases, albeit it requires capsular release in only 3 % of the cases. Prevalence of stiffness seems to be negatively correlated with the size of cuff tear size [68]. Gleyze et al. [69] prospectively analyzed nonsurgical, surgical, and combined treatments including self-rehabilitation below and above the pain threshold. They found that supervised self-rehabilitation was more effective than traditional assisted

rehabilitation. Capsular distention improves short-term results, but is ineffective in the long term [70, 71]. Along with the French Society for Arthroscopy, Gleyze et al. [72] interestingly found that informed and active patients who perform self-rehabilitation beyond the pain threshold suffer less from pain than patients who respect the pain threshold. Conservative treatment of postoperative shoulder stiffness has significantly higher failure rate (14–17 %) than capsular release (0 %), though function improvement and overall results are similar. Therefore, self-rehabilitation under supervision is proposed as the first step of treatment followed by distention (optional) or capsular release if there is no improvement after 6 months of conservative treatment.

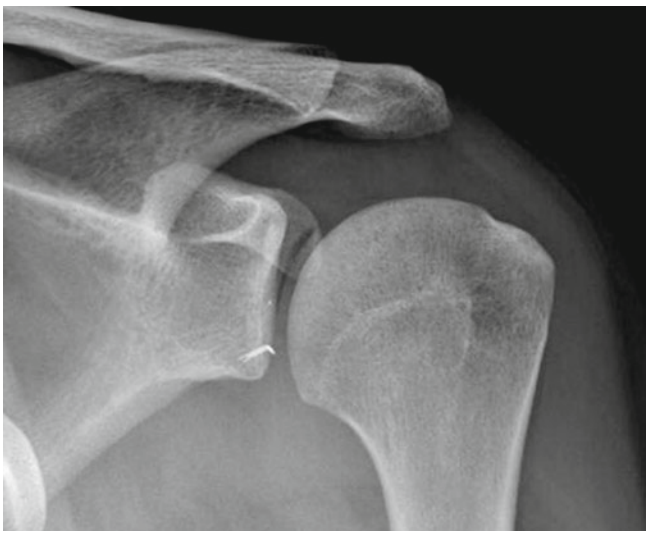

Fig. 47.1 Intra-articular metallic foreign body: an anchor introducer device broken inside the joint

Chondrolysis

Post-arthroscopy chondrolysis is a rare but devastating complication, since it almost always concerns young patients [73, 74]. The incidence is not known as there are mostly retrospective studies dealing with the subject [75, 76]. It must be assumed that patient factors play a role in post-arthroscopy chondrolysis [75] and athletes are more at risk for traumatic cartilage injury [77]. However, main ethological factors are iatrogenic. The most often mentioned cause of chondrolysis is the intra-articular injection of local anesthetics. Particularly, bupivacaine, lidocaine, and ropivacaine have been proven to be chondrotoxic and to induce in vitro and in vivo chondrolysis in animal and human cells [76, 78–85]. Their negative effect is dose and time dependent [86], and their use is strongly discouraged since there seems to be no beneficial effect on the long-term outcome [87, 88]. Further causes of chondrolysis are heat as applied through radio-frequency devices [75, 76, 89]. An intra-articular temperature of more than 45 °C has been proven to damage chondrocytes [90, 91], and the manufacturers started to produce devices with inbuilt thermometers and alerts. Thermal capsulorrhaphy as treatment of shoulder instability was discouraged due to the devastating effects on the cartilage [92]. Loose bodies in the shoulder, such as implants, sutures, or bone fragments, can mechanically destroy cartilage and thus lead to chondrolysis [75, 76]. Bioresorbable anchors can get loose when they are partially absorbed and no bone ingrowth has taken place. There have been reports of Gentian Violet being chondrotoxic [76]. Although there are a number of treatment options for cartilage damage in shoulder surgery, including microfractures, osteoarticular transfer (OATS), remplissage, and lesser tuberosity transfer [77], in bupivacaine-induced chondrolysis, a total shoulder arthroplasty often remains the only option [93].

Implants

Complications from implants are known in any type of surgery that uses implants and can roughly be divided into implant failure and biocompatibility problems. In arthroscopic shoulder surgery, the use of bioabsorbable implants has become more and more popular since they do not interfere with any later surgery (e.g., total shoulder arthroplasty) [94]. However, these implants have their own spectrum of complications. Whereas metallic implants can break and/or migrate (Fig. 47.1) [95], bioabsorbable implants walk a thin line between keeping the strength (too quick degradation) and not being absorbed at all [96]. In addition, absorbed implants are not replaced with bone (Fig. 47.2), and adipose tissue can be found at the site of implantation after degradation. Also aseptic osteolysis around the implant has been described

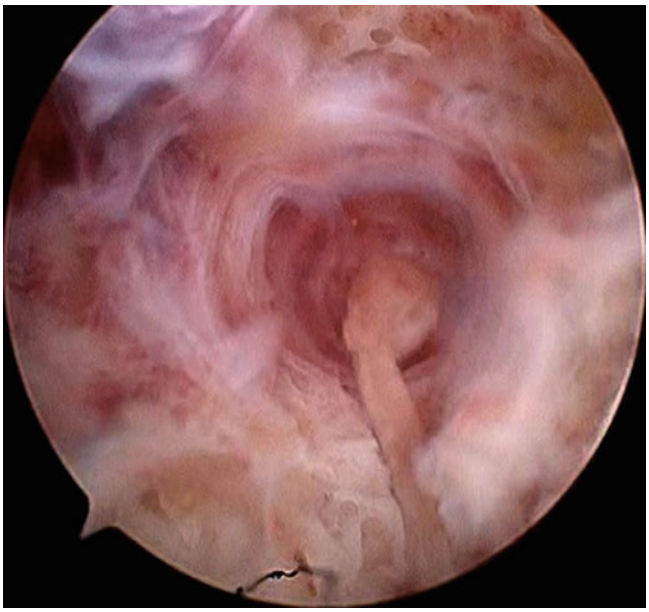

Fig. 47.2 Anchor resorption without bone integration

(Fig. 47.3). All of these sequelae result in implant loosening and migration (Figs. 47.4) and finally in mechanical cartilage damage and synovitis [97–101]. A rare complication to bioabsorbable implants is an allergic reaction [94]. Nonetheless, in their level I prospective studies, Milano et al. found no difference in the short-term outcome between metallic and bioabsorbable anchors for rotator cuff and instability repair [102, 103].

Radio Frequency

Radio frequency has been associated with rather uncommon complications, such as skin burns due to overheated fluid loss [104, 105]. Certainly, major concern with this device is the risk of overheating the glenohumeral joint, as heat levels of more than 45 °C are dangerous for the chondrocytes and can induce thermal chondrolysis [90, 91]. Although McKeon et al. [106] found in a cadaver study that during intra-articular use of radio frequency, temperature never exceeded 43 °C, however, it is reasonable that excessive use of radio frequency inside the glenohumeral joint can be dangerous [75, 76, 107]. Indeed, when there is enough irrigation, the temperature is quickly reduced [90, 108].

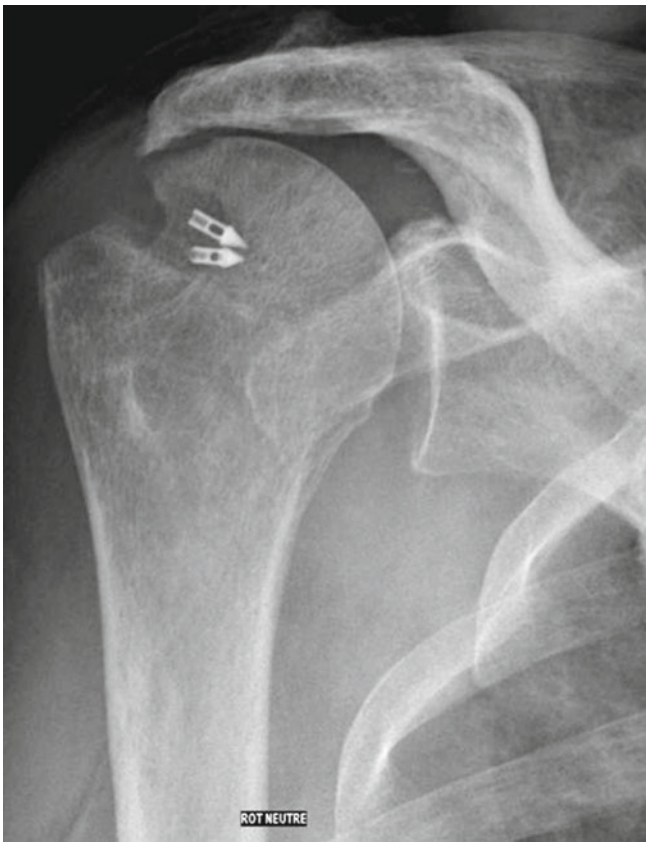

Fig. 47.3 Osteolysis around two anchors. The missing bone is clearly visible

Liquid Diffusion

The net weight gain during a shoulder arthroscopy is time dependent and related to the amount of fluid used [109]. Lo and Burkhart [110] used up to 100 L of irrigation fluid during their procedures. Although they noticed a postoperative weight gain of up to 8.5 kg (average: 4 kg), no irrigation-related complications were reported.

During shoulder arthroscopy, the deltoid muscle pressure can rise up to 100 mmHg, and a case of rhabdomyolysis with consecutive kidney failure was described [111].

The main concern of extra-articular fluid gain is the risk of respiratory distress, which has been reported in case descriptions [112–115] and is mostly associated with the lateral decubitus position [46]. In our experience, one case of fluid extravasation caused respiratory distress in a beach-chair position (unpublished data).

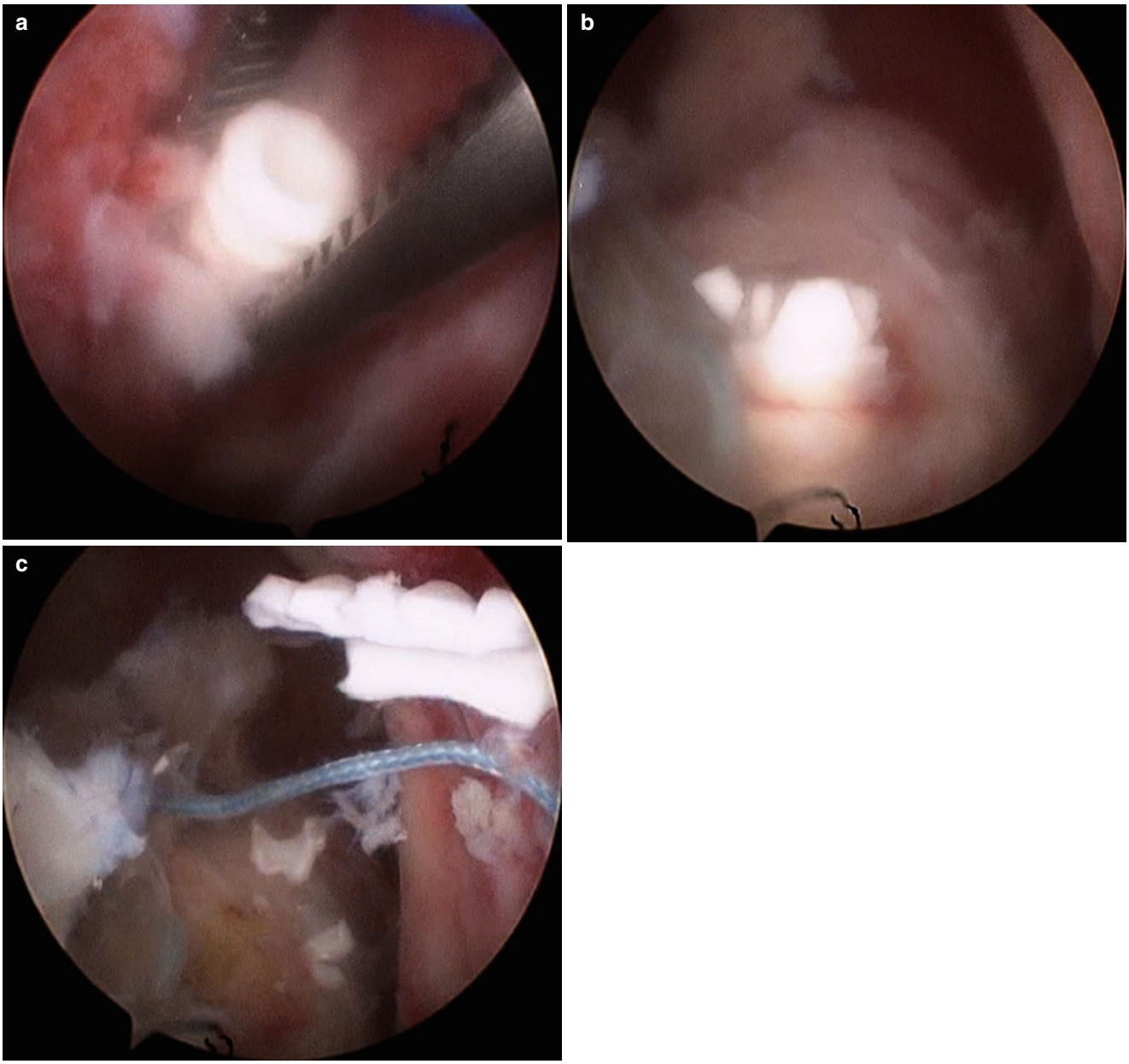

Fig. 47.4 (**a**) Loose resorbable anchor. The anchor can easily be removed with the clamp. (**b**) Loose and partially broken resorbable anchor. (**c**) Broken resorbable anchor

Summary

When we take surgical decision, select patients for surgery or talk to the patients and get their informed consent; we must consider the possible complications. Knowledge of the incidence of a complication is important but not sufficient. We need to know and to explain to what extent a complication can affect the outcome and thus the patient's (and surgeon's) satisfaction. Both incidence and severity of a complication must be in relation to the achieved goal of an operation.

Complications after arthroscopic shoulder surgery are rare and mostly of low impact. Furthermore, most of them can be avoided or greatly reduced when all the involved personnel is aware and cautious.

It is our duty as responsible physicians to make sure we set up a work environment that allows everyone, including ourselves, to perform at our peak.

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Part V

Outcome Research in Shoulder Arthroscopy

Outcome Research in Shoulder Arthroscopy: An Overview

48

Roberto Padua and Enrico Ceccarelli

Outcome research is no news. At the end of the twentieth century, it is understood that this set of research methodologies represent an evolution and extension of the kind of clinical research that was already familiar to physicians [1–3].

Outcome research, in its most complete form, may contain a number of different components such as analysis of large databases; organized or structured reviews of the literature, known as meta-analysis; small-area analysis of healthcare utilization; prospective clinical studies emphasizing patient-oriented outcomes of care; and development of decision-making analytical models, cost-effectiveness studies, and practice guidelines [1–3]. Today, all these issues are endorsed, and most of the papers follow the outcome research concepts. However, not even the outcome research goal is reached especially for patient's perspective data.

In the last 20 years, the use of validated patient-oriented measures has added another dimension to clinical outcome evaluation. These measures, which focus on functional status and symptoms, are more relevant to patients [1–3].

Self-administered questionnaires have been shown to be valid and reliable methods that should be used to determine the effects of treatment on the health and the quality of life of the patient. However, questionnaires to be used in scientific literature should show the following characteristics: reliability, sensitivity or responsiveness, and validity [4].

Reliability is a measure of consistency or degree of dependability. It can be divided into two major classes. Internal consistency, which is a measure of equivalence, is the ability of a scale to measure a single coherent concept. Reproducibility, or test-retest reliability, which is a measure of stability, is the ability of a scale to give the same results when administered on separate occasions [5].

Responsiveness or sensitivity is the ability to detect clinical change, while validity is an index of how well a test measures what it is supposed to measure [6, 7].

In order to obtain significant data, the questionnaire must be widely accepted and used in literature, so that the extract data could be considered a "standard" and the paper data might be used in secondary publication. Nowadays, in shoulder assessment, there is not a "standard" widely accepted being more of 20 different questionnaires published. This fact is harmful for patient's perspective dissemination [8–12].

Experimentation and the statistical validation of some of these tools makes the patient's point of view, rigorously obtained, a fundamental requisite for conducting any accurate clinical trial [12–14]. The development of subjective evaluation questionnaires on medical and surgical treatment depends on economic motivations and on the scientific need to evaluate results in a codified manner.

To detect the patient's perspective tools, we can rely on generic questionnaire, disease-specific questionnaire, and site-specific questionnaire.

The generic questionnaire, such as the SF-36, generically analyzes the quality of life; instead, the disease-specific questionnaire analyzes symptoms and function in a specific pathological condition, while the site-specific questionnaire is based on the concept of a single functional unit, such as the upper extremity [4].

The generic questionnaires, validated for assessment of general health status, may not be specific enough to provide an accurate, comprehensive description of symptoms and function of an individual joint [2, 3, 10].

As a consequence, numerous specific questionnaires have been proposed for each different disease [15–18]. These analyze some of the characteristic aspects of the disease, while others are more related to the general health status and the patient's satisfaction with the treatment. On the other hand, questionnaires of this type are limited because of their

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extreme focus on specific diseases and can therefore be used to analyze only the more frequently observed diseases and those of greater scientific interest.

In order to find a compromise between accuracy and feasibility, the American Academy of Orthopedic Surgeons has established a specific committee whose efforts have led to the development of four basic questionnaires that analyze different areas of interest (upper limb, lower limb, spine, pediatric orthopedics) rather than single diseases [19, 20].

During the 63rd AAOS Annual Meeting (Atlanta, 1996), the results of the first 3 months of experimentation on 39,000 patients who had filled out these forms were reported, and now some of these became a standard in scientific world [19, 20].

The data collected may be stored in an intentionally created computer software program and may therefore be easily elaborated statistically. In this manner, the data may be compared to those of other centers.

The use of general or specific self-administered questionnaires allows comparing the results of different surveys, introducing the meta-analysis in orthopedics. It must be underlined that this statistical analysis of results reported in single studies in which the same measures of evaluation and rigorous inclusion criteria are employed permits combining the information and greatly increasing the statistical value of the conclusions [13, 14].

The use of systems of evaluation on the state of health, based on the patients' perceptions, would be useful in developing the meta-analysis as an instrument to provide the orthopedist with valid motivations to follow a particular therapy rather than another, justified by statistically high numbers and not influenced by the opinion of the individual author.

Which Questionnaire Must We Use in Shoulder Assessment?

Firstly, the ideal questionnaire depends on the goal of our intention. If we want to study all our shoulder patients, we must use one site-specific questionnaire, eventually associated by generic health status one (outcome assessment). If we study a specific and limited population (e.g., in an RCT or cohort study), we can use a disease-specific questionnaire (as WOSI or Rowe for instability).

The "ideal" questionnaire should have a validation process, a cross-cultural validated version in most countries, and be widely used in literature [21].

In order to use a questionnaire with different language groups and in different cultural settings, the questionnaire must not only be translated into the new language but also be adapted to the local culture. It must then be validated against the original version. The cross-cultural adaptation guidelines described by Guillemin are widely accepted and used for the translation and adaptation of questionnaires [21–23].

The cross-cultural adaptation consists in a translation and back-translation of the questionnaire, which must be reviewed by an expert committee and test of the prefinal version to establish as better as possible proper correspondence with the original version. Once more, the validation of the questionnaire must be detected, and the reliability and validity must be assessed through a cross-sectional study [21–23].

A recent literature research, conducted in the most representative databases available (Medline, CINAHL, and EMBASE), following the keywords for the search, identified through the National Library of Medicine's medical subject heading (MeSH) database, showed lots of questionnaires about shoulder. A hundred of articles, regarding shoulder diseases and including patient's perspective, have been collected to identify the "best" questionnaire. The articles were focused on nonoperative and operative treatment of common diseases. Questionnaires used in the papers and related patient number are reported in Table 48.1.

The *ASES subjective form* is a site-specific questionnaire. It has 11 items that can be used to generate a score. These are divided into two areas: pain (one item) and function (ten items). The response to the single pain question is marked on a 10-cm visual analog scale (VAS), which is divided into 1-cm increments and anchored with verbal descriptors at 0 and 10 cm. The ten items in the function area of the ASES include activities of daily living such as managing toileting and putting on a coat. There are more demanding activities such as lifting 10 lb above shoulder height and throwing a ball overhand. Finally, there are two general items: doing usual work and doing usual sport. There are four categories for response options from 0 (unable to do) to 3 (not difficult). The final score is tabulated by multiplying the pain score (maximum 10) by 5 (therefore, a total possible 50) and the cumulative activity score (maximum 30) by 5/3 (therefore, a total possible 50) for a total of 100. Some validated versions of the questionnaire are published in different languages [24].

The *UCLA shoulder scoring scale* was described initially as a method to assess the outcome of shoulder arthroplasty. However, the UCLA scale has been used to describe the outcome of intervention for many shoulder conditions, including rotator cuff tears. The UCLA scale has been modified to include an additional five points for patient satisfaction. Parameters include pain (10 points), motion (10 points), function (10 points), and patient satisfaction (five points). Subjective criteria are responsible for 15 points of a total of 35 points, and examination findings are responsible for the remaining 20 points. The UCLA score was converted to a 100-point scale for comparison with the other shoulder assessment systems [25].

Table 48.1 Shoulder questionnaires used in the literature to assess outcome and related patient number

	ASES	UCLA	Rowe	DASH	SST
N. of papers	27	21	19	13	13
N. of patients	1,115	913	852	439	531

The *Rowe score* was initially described in 1978 as a method to assess the outcome of treatment for anterior shoulder instability after Bankart repair, so is a condition-specific questionnaire. Out of a total of 100 points, 50 points are for stability, 20 points are for range of motion, and 30 points are for function. Some validated versions of the questionnaire are published in different languages [26].

The *DASH questionnaire* is a site-specific questionnaire, and it consists of 30 questions that inquire about symptoms and functions of the upper limbs which are affected by orthopedic or neurologic disorders. These provide a single main score, the DASH function/symptom (DASH-FS) score, which is basically a summation of the responses on a one-to-five scale, after transformation to a zero (no disability) to 100 (severe disability) scale. In addition to the 30-item score, there are two optional four-item question sets, the DASH sport/music (DASH-SM) and DASH work (DASH-W), which are scored similarly. The questions test the degree of difficulty in performing a variety of physical activities because of arm, shoulder, or hand problems (21 items). They also investigate the severity of pain, activity-related pain, tingling, weakness and stiffness (five items), and the effect of the upper limb problem on social activities, work, sleep, and self-image (four items). The two optional domains contain activity-specific items concerning the performance of sports and/or the playing of musical instruments and the ability to work. All the items have five response choices ranging from "no difficulty or no symptoms" (scores 1 point) to "unable to perform activity or severe symptoms" (scores 5 points). The questionnaire is the most widespread for validated version in different languages [23, 27–29].

Simple Shoulder Test (*SST*) is a series of 12 "yes" or "no" questions. The patient answers about the function of the involved shoulder. The answers to these questions provide a standardized way of recording the function of a shoulder before and after treatment. Also in this case, validated versions of the questionnaire are published in different languages [30].

Some words must be spent for the *Constant-Murley shoulder scoring system*, the most commonly used international shoulder scoring scale. This questionnaire is attractive because it has been the subject of extensive psychometric validation, but since it does not collect patient's perspective, it has not been included in this chapter.

Summary

The author's opinion, based on the questionnaire-specific characteristics and its spread, is that the "ideal" questionnaire does not exist yet. Considering the data found in literature, the compromise between diffusion and validation, the DASH and the ASES are the best questionnaires to be used in shoulder outcome assessment [30, 31]. To have all paper data in univocal manner, a bigger effort should be done by the scientific world selecting a unique questionnaire as standard to evaluate shoulder. For outcome research in a specific population, the choice depends on the focus of the study.

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Experimental Models in Shoulder Research

49

Leonardo M. Cavinatto and Leesa M. Galatz

Introduction

Animal models represent a very important tool for the advancement of orthopedic research. These models enhance the understanding about the natural history of diseases, contribute to the development of new clinical treatments and surgical techniques, and serve as a bridge between in vitro studies and human clinical trials. The use of animal models permits testing emerging theories and concepts in a coherent and controlled environment, with consistent approaches at specific time points.

In orthopedic research, there are many well-established animal models that closely reproduce human conditions. These include stress fractures, cartilage degeneration, knee instability, spinal cord injury, and arthritis. Many pathological conditions of the shoulder lack a validated animal model. Nevertheless, several shoulder conditions have been the subject of experiments using animal models, including shoulder contracture [1], shoulder arthroplasty [2, 3], shoulder instability, and neonatal brachial plexus palsy [4]. In recent years, most shoulder experiments using animal models focused on rotator cuff disease, the most prevalent shoulder orthopedic condition (Table 49.1).

Cadaveric studies provide an appropriate tool to study some of the basic concepts of the shoulder, such as anatomy and biomechanics. Regarding rotator cuff disease, cadaveric studies precisely test muscle function, shoulder biomechanics, and strength of various repair techniques, but are not suitable for analyzing the healing process after rotator cuff repair. A biological scenario is necessary to study gene expression, extracellular matrix production, and tissue viscoelastic properties. In order to try to identify the best animal model for the study of rotator cuff disease, Soslowsky et al. [5] compared 33 animals using 34 different criteria. As a result, they suggest the rat as the most appropriate animal model for the study of rotator cuff disease. This chapter will review the various models used for the study of shoulder disorders and discuss their strengths and weaknesses with regard to clinical relevance.

Rat Model

The rat is the most commonly used animal model for the study of rotator cuff disease. As shown by Soslowsky et al. [5], the rat has numerous similarities in both bone and soft tissue anatomy to the human (Fig. 49.1). In fact, the rat is one of rare animals that has a well-developed acromion that orients anteriorly and articulates with the clavicle, forming an enclosed bony arch over the supraspinatus and infraspinatus tendons. When the rat walks, burrows, or reaches overhead, there is an excursion of these tendons under the coracoacromial arch. This particular anatomy, similar to the human condition, makes this a relevant model to study extrinsic mechanisms involved in rotator cuff disease, such as impingement and overuse [6, 7].

A wide variety of different biologic reagents are available for the rat, making broad biological analysis feasible [8]. Because of the large number of primers and antibodies already commercially available, gene expression, protein quantification, and immunohistochemistry assays are easily accessible. The rat is one of the animals with the greatest percentage of genetic similarity to humans. One study of comparative genomics showed a 80–90 % of genetic semblance between the rat and the human [9]. This similarity enables the rat to provide very useful translational data.

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Table 49.1 Comparative analysis of reported models for shoulder research

	Bony anatomy	Biologic reagents	Rotator cuff repair	Cost	Ease of handling	Arthroplasty
Mouse	+++	+++	No	$	++++	No
Rat	+++	+++	Difficult	$	++++	No
Rabbit	++	+	Yes	$$	+++	No
Dog	+	–	Yes	$$$	++	Yes
Sheep	+	–	Yes	$$$	++	Yes
Primate	+++		Yes	$$$$	Very challenging	Potential

The rat is also one the rare animals that can tolerate bilateral surgery. This unique characteristic makes paired analysis possible, which enhances the statistical power of the study. Additionally, rats have a short gestational period with multiple offspring, a rapid growth rate, and a short life span [8]. As opposed to other animals used in shoulder research, rats rarely get infected or sick, tolerate anesthesia well, are relatively inexpensive, and are very docile.

However, the rat and the human rotator cuff have considerable differences. In the rat, the tendons are not interdigitated together, and they are blended to the underlying joint capsule only very close to their bone attachment. In the human shoulder, the rotator cuff tendons are interdigitated together and blended to the capsule throughout the whole trajectory [10]. Additionally, rodents have a growth plate at the proximal humerus that remains open well into maturity. The clinical significance of this is unknown, but suggests a higher capacity for certain remodeling and regenerative processes compared to humans.

Another obvious disadvantage of the rat model for shoulder research is the small size of the rotator cuff tendons, making standard-of-care repair techniques virtually impossible. The rat, like all other animals used for shoulder research, generates a massive scar response between the stump of the tendon and the bone where the tendon was previously attached. Therefore, the rat generates scar tissue even in the absence of repair [11]. However, unlike large animal models, tendon repairs rarely fail in the rat, making the study of repaired tendon-to-bone insertion sites possible [12, 13].

In the following sections, the important contributions from rodent models concerning shoulder pathology and development will be briefly described.

Mechanobiology

The rat model has opened new avenues for studying mechanobiology during tendon development and rotator cuff disease and has provided insight for clinical application.

Muscle paralysis during tendon development in neonatal mice, either by botulinum toxin injection or superior trunk neurotomy, results in delayed tendon formation, delayed fibrocartilage maturation, and impaired mineralization of the enthesis [4, 14]. For the first 2 weeks of life, the effects of paralysis are minimal, and the effects escalate beyond that time point. These findings suggest that early development is under genetic control, while later changes are greatly influenced by the mechanical environment. These experiments lead the way for developing a useful animal model for future research examining musculoskeletal changes secondary to neonatal nerve injury, including neonatal brachial plexus palsy (NBPP).

The rat model has been instrumental in studying tendon-to-bone healing of the rotator cuff. This model has been used to develop and improve repair and rehabilitation strategies. In an acutely repaired rat rotator cuff, immobilizing the operated limb leads to improved mechanical properties, smaller volume of scar tissue, and greater collagen organization when compared to the rats allowed normal cage activity or exercised on a treadmill [15, 16]. These data show a potential negative effect of excessive motion and force at the repair site.

Similarly, another study demonstrated that high load and excessive motion across the repair site during tendon healing leads to inferior mechanical properties and poor healing outcomes. Passive motion and exercise following tendon-to-bone repair led to significant decreases in mechanical, structural, and compositional properties at the repair site. Exercise and passive motion also led to reduced range of motion compared with rats exposed to immobilization or regular cage activity [15, 17, 18].

Although some stress deprivation by immobilization or short-term paralysis can potentially benefit recovery and enhance mechanical properties of the enthesis in certain scenarios [19, 20], complete unloading has also been shown to be detrimental for tendon-to-bone healing. In two experiments where rat supraspinatus muscles were injected with botulinum toxin after repair, biomechanical testing demonstrated significantly inferior structural properties, specifically ultimate stress and stiffness, in experimental rats compared to controls [12, 21]. Therefore, similar to other musculoskeletal tissues, some controlled force is beneficial to healing. There is an optimal level of tension for tendon-to-bone healing, and both the complete lack of force as well as excessive loads are detrimental [22]. This research provides insight for rehabilitation protocols in clinical practice.

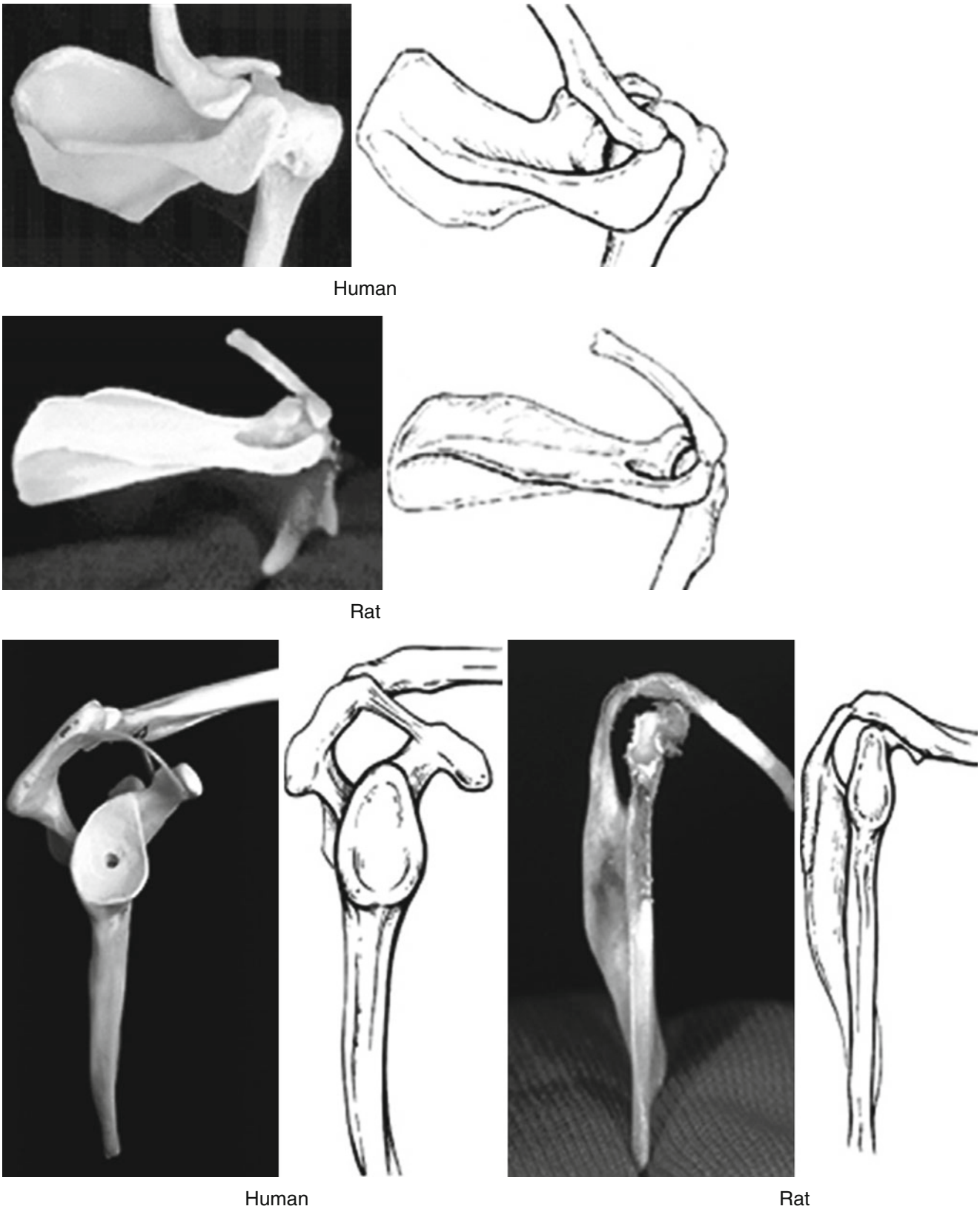

Fig. 49.1 The bony anatomy of the human and the rat shoulder is very similar. A well-developed acromion and coracoacromial ligament forms a rigid arch over the rotator cuff (From Soslowsky et al. [5]. Copyright: Journal of Shoulder and Elbow Surgery; published by Elsevier 1996. Reproduced with permission)

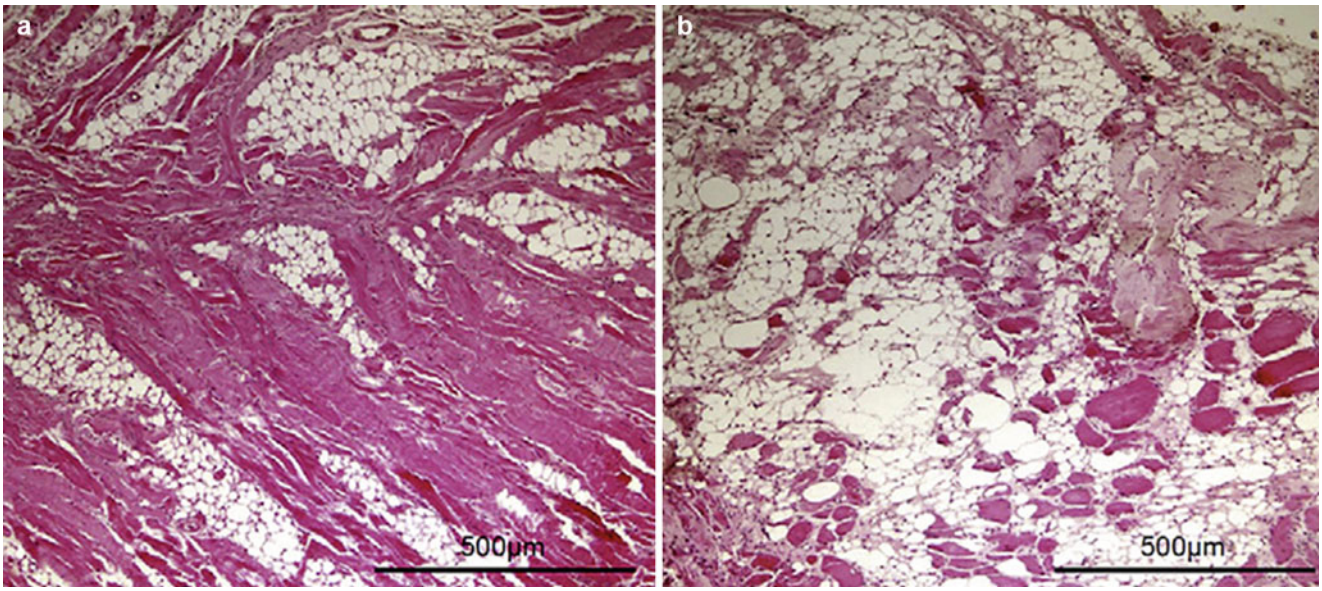

Fig. 49.2 A mouse supraspinatus (**a**) and infraspinatus (**b**) muscles at 8 weeks after tenotomy of the two tendons (supraspinatus and infraspinatus) and neurotomy of the suprascapular nerve shows substantial muscle degeneration, characterized by fatty changes and atrophy (From Kim et al. [30]. Copyright: Journal of Shoulder and Elbow Surgery; published by Elsevier 2012. Reproduced with permission)

Biological Studies

The rodent model has enabled the study biological adjuvants to the repaired rotator cuff, in an effort to improve healing. Augmentation of the tendon-to-bone environment with growth factors [23], stem cells [24, 25], or bioengineered scaffolds [26, 27] has been used to enhance healing of the rotator cuff to bone. Although some improvement could be seen with the addition of some specific growth factors, the biological process is complex and involves a multitude of growth and transcription factors. Ideal timing for delivery and maximally effective agent is still under investigation [22].

Cellular and gene transfer approaches have shown to promote some improvement in the rotator cuff repair. Although mesenchymal stem cells (MSCs) alone delivered at the repair site did not result in improvement [28], MSCs transfected with scleraxis, a transcription factor involved in tendon development, resulted in improved structural properties of the newly formed repair [29]. A similar study with MSCs transfected with type-1 matrix metalloproteinase (MMP-1) also resulted in significant improvement in rat rotator cuff healing compared to controls [25].

Scaffolds

The rat provides a live model to test bioengineered scaffolds. Newer scaffolds that recreate the native tissue mineral gradation between the compliant tendon and the stiff bone are beginning to be tested in the rat shoulder [22]. These scaffolds are either being implanted alone, with MSCs, or with MSCs transfected with bone morphogenic protein-1 (BMP-1). Further studies are ongoing.

Chronic Model

Recently, the rat was validated as a model for massive, chronic rotator cuff tear [30]. It was shown that the detachment of both the supraspinatus and infraspinatus from the greater tuberosity alone or combined with suprascapular neurotomy resulted in fatty muscular degeneration, atrophy, and stiffness. All of these changes are clinical characteristics of chronic degenerated rotator cuff. A preliminary unpublished study comparing acute and chronic repairs using this new chronic rotator cuff model did not show any difference between the two groups by analyzing histology and mechanical properties, and a higher failure rate correlated with tear size and gap formation.

Mouse Model

Recently, a mouse model for rotator cuff tears was developed [31]. In addition to all the advantages of using a small rodent model, the mouse model can take advantage of the vast array of transgenic mouse strains (Fig. 49.2). However, the surgery to repair a rotator cuff tear is vastly more difficult, creating limits as to its utility as a healing model.

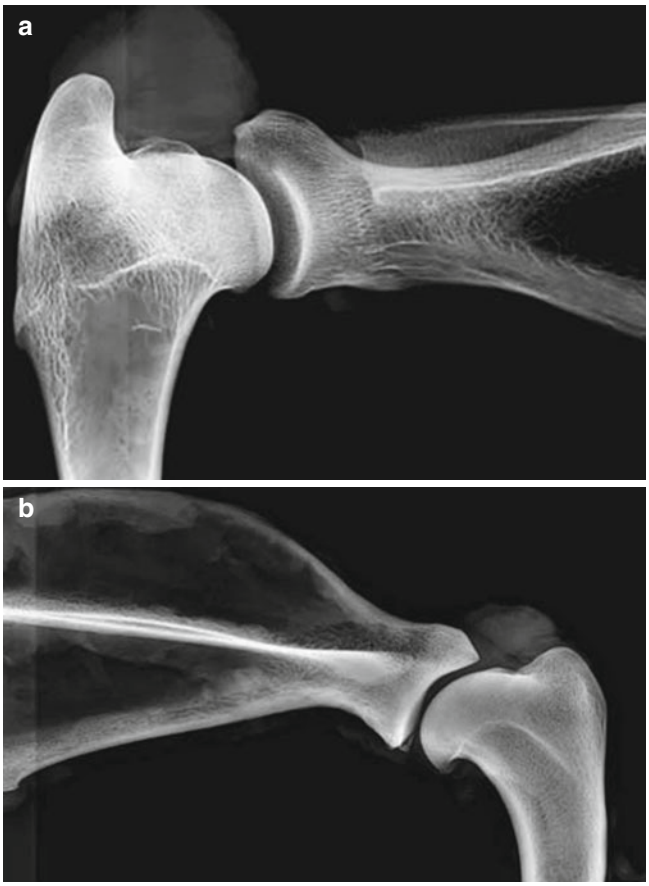

Fig. 49.3 Shoulder radiograph of a sheep (**a**), and of a dog (**b**), showing on both shoulders an elongated humeral head, a deep glenoid, and a prominent greater tuberosity (From Turner [32]. Copyright: Journal of Shoulder and Elbow Surgery; published by Elsevier 2007. Reproduced with permission)

Large Animal Models

Large animals have shoulder joints and rotator cuff tendons that are similar in size to the human (Fig. 49.3). This allows more accurate and reproducible studies of repair techniques of the rotator cuff tendons to the proximal humerus insertion footprint. The smaller the animal, the more accelerated the healing process. Therefore, studies evaluating the strength and mechanical properties of a repair over a given time course, potentially provide more applicable translational data.

Novel shoulder prosthesis designs and techniques have been tested in large animal models [2, 33]. The use of large animal models for the study of prosthesis design and wear characteristics is potentially useful, but its use has not been validated or maximized at this time point.

Notwithstanding, large animals have several disadvantages for studying shoulder pathology. All the large nonprimate animals used for shoulder research, including the sheep, the goat, the dog, the rabbit, and the calf, have very different bone and soft tissue anatomy compared to the human shoulder [10, 34]. In these quadruped animals, the acromion, the clavicle, and the coracoid process are generally nonexistent and do not cover the rotator cuff. In the rabbit, specifically, the acromion directs inferiorly and partially covers the infraspinatus and the teres minor tendons. The rabbit subscapularis tendon passes through a bone tunnel in the anterior aspect of the joint. The soft tissues in the shoulder are also significantly different from humans, as the animal rotator cuff tendon fibers are very aligned, analogous to the Achilles tendon in the human. The tendons do not have fibers crossing in different directions and are not blended with the underlying capsule. In large animals, the rotator cuff tendons are mainly extracapsular, preventing contact with the synovial fluid.

In large animal models, among the rotator cuff tendons, the most utilized is the infraspinatus. This tendon has a size very similar to the human supraspinatus, is easily accessible after the shoulder approach, and when injured, gait is significantly affected.

One of the biggest disadvantages of the use of large animals for the study of rotator cuff disease is the ubiquitous rate of early tendon repair failure, often in the first few days. Because re-tears always happen, what it is really being studied is the scar tissue that forms between the stump of the tendon and the greater tuberosity and not the newly formed enthesis itself [35–38]. One may argue that is embodies the character of rotator cuff tendon healing; however, the universal retraction presents a limitation. This phenomenon is seen even when the postoperative limb is protected by immobilization or by a softball affixed to the operated paw postoperatively.

Canine Model

The canine shoulder has been used as a model for rotator cuff injury and repair, as well as other shoulder pathologies in a broad range of experiments. In one study, tantalum bead markers were placed on the injured tendons of adult mongrel dogs after full-thickness supraspinatus injury and repair. The repairs showed a 100 % failure rate, as evaluated by bead displacement (Fig. 49.4). These failures occurred early in the postoperative period, regardless of the suture type, suture configuration, or postoperative protocol [38]. To reduce the incidence of re-tears in the canine model, another experiment performed by the same group tried a partial-width lesion and subsequent repair, detaching only the upper two thirds of the infraspinatus tendon [39]. Although the retraction distance was reduced compared to the full-thickness injury model, a 100 % repair failure rate was still observed. The authors concluded that the canine model represents a rigorous test for new sutures and repair techniques in rotator cuff repair, but is not suitable for studying the newly formed enthesis.

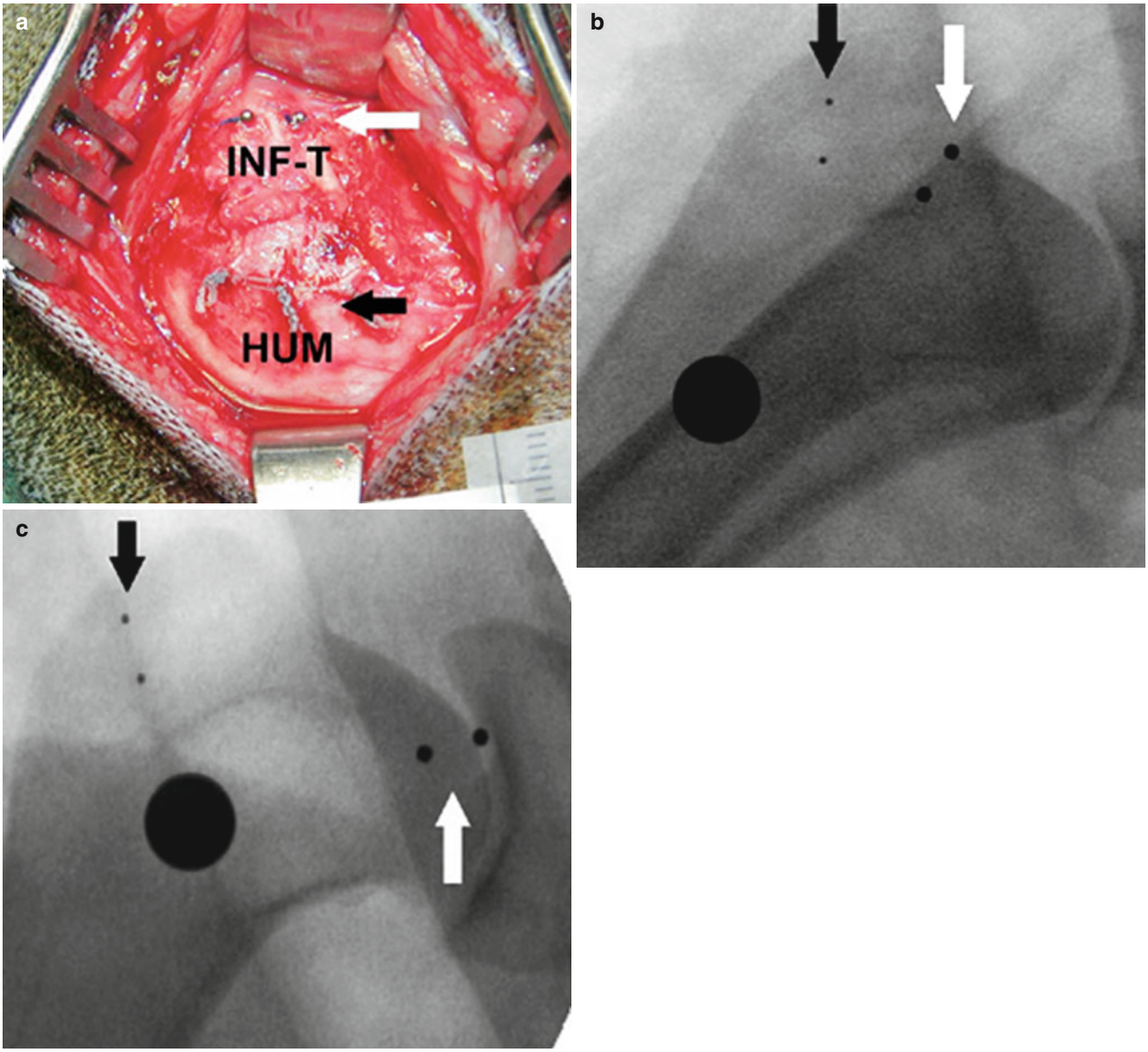

Fig. 49.4 (**a**) Tantalum bead markers were placed on the injured infraspinatus tendon (IFT-T, *white arrows*) and on the greater tuberosity of the humerus (HUM, *black arrows*) of adult mongrel dogs after full-thickness supraspinatus injury and repair. Fluoroscopy was done intraoperatively (**b**) and 5 days after repair (**c**) (From Derwin et al. [38]. Copyright: Journal of Shoulder and Elbow Surgery; published by Elsevier 2007. Reproduced with permission)

The dog shoulder has some peculiar differences compared to the human shoulder. Canines have a flattened humeral head, a prominent greater tuberosity, and a deep glenoid [40]. There are also important differences in the biomechanics of the dog shoulder compared with the human, as the dog uses the forelimbs for weight bearing and has very limited overhead activity [8, 10, 34].

On the other hand, the canine model accurately reproduces the degenerative changes observed in the chronically ruptured human rotator cuff tendons. In fact, the canine model simulates muscle stiffness, atrophy, and fatty degeneration in the chronic and massive rotator cuff tears observed in the human clinical scenario [38, 41]. Additionally, dogs have been used to test scaffolds for augmenting tendon-to-bone repair [39].

The canine shoulder also has served as a model to test other clinically relevant experiments, such as shoulder reconstruction and arthroplasty. Wirth et al. tested a new glenoid implant in the canine [33], and Matsen et al. experimented with a novel technique for shoulder arthroplasty, where a prosthetic humeral component articulated in a reamed, non-implanted glenoid [2]. The authors from this last experiment subsequently applied the technique to clinical practice [42].

Sheep Model

The sheep model has become a convenient large animal for the study of orthopedic diseases, including shoulder pathologies. The sheep has a well-developed and easy accessible infraspinatus tendon with size comparable to the human supraspinatus tendon. These features make the sheep infraspinatus tendon a reasonable option for investigations involving rotator cuff repair (Fig. 49.5). Sheep are also easily handled animals and well accepted by society as a research animal [32].

The sheep has been using to test different suture techniques and configurations [37], novel suture anchors [43], bioengineered scaffolds, and biological aids to try to enhance tendon-to-bone healing [44] in the setting of rotator cuff repair. It has also been used to study the degenerative consequences after tendon unloading and to investigate the findings after tendon reloading [45].

The sheep model has two major shortcomings. Similar to other large animal models, a huge scar forms between the stump of the tendon and the bone after detachment. This is in direct opposition to the lack of healing evidenced in the clinical scenario, especially in chronic, degenerated, and massive tears. Additionally, in this model, it is virtually impossible to keep the repaired tendon attached to the bone, regardless of the type of immobilization used in the postoperative period. The high loads imposed by muscle activity and weight bearing result in the formation of a gap between the distal end of the tendon and the proximal humeral attachment.

Another distinct disadvantage of this model, especially when comparing to the murine model, is the lack of probes and reagents for the sheep, limiting the variety of biological assays, like PCR, immunohistochemistry, and in situ hybridization [32]. Although limited, some research was conducted in the ovine model in regard to glenohumeral instability. Because of the major anatomic differences, these studies have limited clinical applicability [46].

Rabbit Model

The rabbit is another animal commonly used for shoulder research. Because of the larger size of the tendons compared to the rat, the repair is ultimately more manageable for surgical manipulation and for biomechanical testing. The rabbit model has been primarily used for the study of rotator cuff disease.

The rabbit model has been extensively used to study muscle response after tendon injury and unloading. Muscular fatty degeneration and atrophy were observed after supraspinatus detachment, and the changes were shown to arise within the first 3 months after unloading [47, 48]. In an attempt to reverse the muscle pathology after unloading, Uhthoff et al. repaired rabbit supraspinatus tendon after 6 and 12 weeks after detachment [49]. The investigators showed that early supraspinatus repair did prevent an increase in muscle fat. Interestingly, reloading via repair did not reverse muscular degeneration, even when repaired as early as 6 weeks after detachment.

The rabbit model has also provided new information about tendon-to-bone re-formation after cuff repair. Chondrocytes, non-chondrocytic cells, extracellular matrix, and collagen organization at the healing enthesis were quantified longitudinally from 2 to 24 weeks after the repair [50]. Although both cell lines were restored by 24 weeks, extracellular matrix formation and collagen organization did not reach normal levels. These results suggest that at 24 weeks, the re-formed enthesis is still mechanically weaker than an original age-matched enthesis.

Longitudinal investigations analyzing the temporal expression of metalloproteinases and tissue inhibitions of metalloproteinases, as well of growth factors and proinflammatory markers, were also performed using the rabbit model. These experiments help determine the temporal expression of the biological factors involved in tendon-to-bone healing and provide preliminary data for future experimentation using biological adjuvants to enhance healing [36, 51].

A histological investigation comparing acute and delayed supraspinatus repair was also conducted in the rabbit model [52]. Interestingly, the authors found no difference between the tendons repaired immediately after detachment and after 6 or 12 weeks. The enthesis histology depended only on the time lapsed from repair to ultimate sacrifice or follow-up and not on the period between detachment and repair.

The rabbit supraspinatus tendon was used in several other rotator cuff pathology experiments, including tendon-to-bone healing after the delivery of growth factors, and augmentation with scaffolds [53]. Recently, the rabbit subscapularis tendon, because of its particular anatomy passing through a bone tunnel in the anterior region of the shoulder, has been used in rotator cuff experiments [54] in order to attempt to replicate the potential effect of a coracoacromial arch.

Primate Model

Only one study to date has used the primate model to investigate rotator cuff healing [55]. The authors analyzed histology to show that the new enthesis was not completely matured even after 15 weeks after repair – the furthest time point analyzed. These results support the notion that the rotator cuff healing process is a reparative, rather than a regenerative process. While the primate model is desirable in terms of its human applicability, the challenges with regard to cost and animal handling present major limitations. Use of primates also raises ethical issues surrounding care and use of experimental animals.

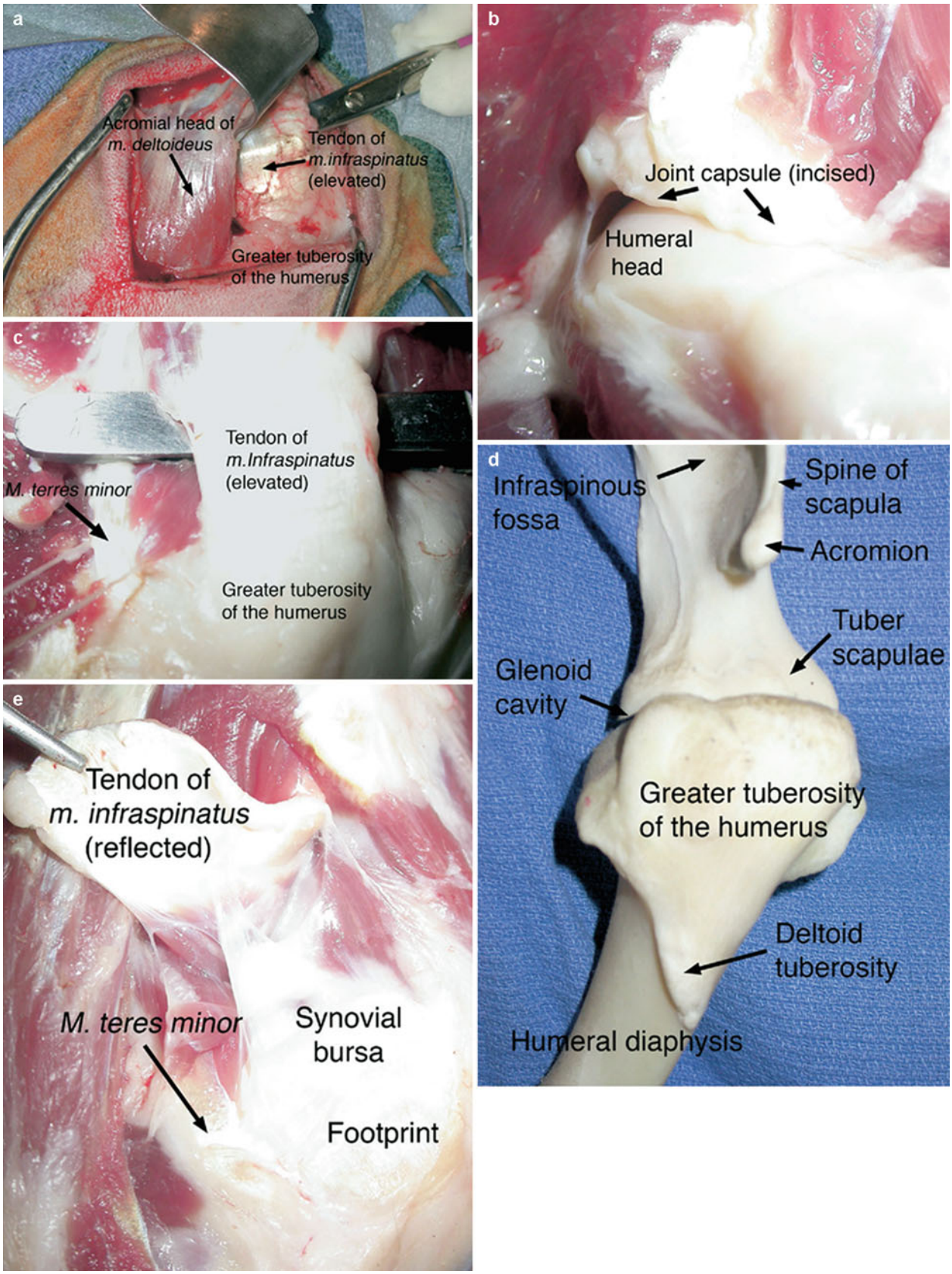

Fig. 49.5 (**a**) Intraoperative view of the surgical approach to the sheep shoulder, showing the infraspinatus tendon insertion on the greater tuberosity of the right humerus. (**b–d**) Anatomic dissection shows the anatomic location of the teres minor muscle, infraspinatus tendon and footprint insertion, humeral head, and joint capsule. (**e**) Bony anatomic landmarks (From Longo et al. [8]. Copyright: Sports Med Arthrosc; published by Lippincott Williams & Wilkins 2011. Reproduced with permission)

Conclusions

No single animal model perfectly represents the clinical condition of a given clinical condition in the shoulder. However, each model has strengths and weaknesses that should be considered in determining appropriate applicability for answering a given scientific question. Smaller animals offer the advantage of low cost, anatomic similarity, and availability of biologic reagents. They are better for evaluating gene expression and protein production. The larger models lend themselves to reproducible surgery but high retraction rates. They are better utilized for evaluating surgical techniques. Both can be utilized for evaluating the utility of biologic augmentation strategies. As always, ethical and appropriate use of animals should be carefully considered.

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Outcome Measurement Tools for Functional Assessment of the Shoulder

50

Warren R. Dunn and James P. Leonard

Introduction

The impact of shoulder injuries and treatments on patients is mostly measured during the clinical evaluation. Initially, simple questions inquire about pain, other symptoms, influence on function and treatment satisfaction. Next, a physical examination assesses the shoulder's range of motion, strength, and stability before performing different provocative maneuvers evaluating for different pathologies. Finally, diagnostic imaging data is obtained and appraised first for injuries and deformity, and then later for signs of healing and prosthetic alignment and stability. The physician deciphers through all the clinical information to evaluate how the shoulder pathology is affecting the patient, determine treatment, and then gauge the effectiveness of their treatment on the patient.

This modality of physician-based outcome measurements has demonstrated some inconsistent results. A study comparing self-reported and observer-reported disability in an orthopedic trauma population found that disability level rating varied greatly, with observers consistently rating disability levels lower than participants [1]. Two other studies evaluating anterior cruciate ligament reconstruction 1 year postoperatively found the patients' ratings of satisfaction, activity level, and function on their self-administered questionnaire to be significantly lower than the surgeons' rating after patient interviews [2, 3]. Indeed, whenever an observer questions a patient with regard to function and then records a response, the possibility of observer bias is introduced [4]. In addition, several studies have demonstrated unreliable results in "objective" measures such as range of motion [5–8], rotator cuff function tests [9], and shoulder instability tests [10]. Such "objective" measures correlate poorly with patient satisfaction when compared with outcome measures that focus on subjective symptoms and function [11]. Overall, physician-directed outcome measures have inherent biases and inconsistencies that adversely affect their results, as well as ignore or marginalize the patients' perspective.

Patient-directed outcome measures focus mostly on domains that directly impact the health and quality of life of the patient. These domains include pain and physical symptoms, sports and recreational function, occupational function, mental health, social issues, emotional issues, and impact on general health. As the health-care system of the twenty-first century continues to evolve, the utilization of these outcome measures is becoming increasingly important. Progressively more focus is being placed on health-care interventions in terms of their effectiveness, their impact on the general health of patients, and their overall cost-effectiveness. Consequently, the use of patient-directed outcome measures that can evaluate these criteria is increasing in the orthopedic literature [12], with some publications making their inclusion a requirement [13]. Furthermore, the orthopedic community has placed emphasis on the utilization of these outcome measures in clinical practice as well as research [14].

Categories of Shoulder Outcome Measures

There are a multitude of different shoulder outcome measures available for patient evaluation, each with specific strengths and weaknesses. These shoulder instruments can be subdivided into two broad categories: general health outcome measures and shoulder-specific outcome measures.

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Table 50.1 Categories of outcome measures to evaluate the shoulder

Category	Definition	Advantages	Disadvantages	Examples
General health	Evaluates the impact of any medical condition on physical, mental, and emotional aspects of general health	Determines impact on quality of life	Outcome scores affected more by lower extremity function than upper extremity function	Medical outcomes study 36-item short form (SF-36)
		Allows comparison of different diseases and conditions across a medical spectrum	Limited responsiveness to changes in shoulder function	Medical outcomes study 12-item short form (SF-12)
Limb specific	Evaluates the physical function, symptoms, and psychosocial factors of a single or multiple disorders of the upper extremity	More specific questionnaires to upper extremity than general health outcome instruments Evaluation of a single or multiple disorders Useful if diagnosis to upper extremity unknown Allows comparison between different upper extremity conditions	Less responsive than shoulder-specific or condition-specific measures	Disabilities of the Arm, Shoulder, and Hand (DASH) QuickDASH
Joint specific	Questionnaires specific to shoulder symptoms, function, and their impact on different activities	More sensitive than general health and limb-specific outcome measures	Less able to evaluate effect on overall health Unable to compare outcome across different conditions, populations, or interventions	American Shoulder and Elbow Surgeons (ASES) Constant-Murley Simple Shoulder Test
Condition specific	Assess specific conditions to the shoulder	Most sensitive to small changes in the condition being evaluated	Limited usefulness in comparing outcomes across different disorders, anatomic sites, and population Need several outcome measures to assess all conditions affecting the shoulder	Western Ontario Rotator Cuff Index (WORC) Western Ontario Shoulder Instability Index (WOSI) Western Ontario Osteoarthritis of the Shoulder Index (WOOS) Rotator Cuff Quality of Life (RCQOL)

Improvements in the development and utilization of these measures have resulted in an increased and improved number of tools. Consequently, outcome measures assessing the shoulder can be further subdivided into limb-specific outcome instruments, joint-specific outcome instruments, and disease-specific outcome instruments (Table 50.1).

General health outcome measures appraise the impact of any medical condition on general health, including physical, mental, and emotional parameters. Its utilization across different medical disciplines allows shoulder pathologies to be compared to other major medical conditions, such as hypertension, congestive heart failure, acute myocardial infarction, diabetes mellitus, and clinical depression [15]. However, its broad multidisciplinary application lacks content specific to the shoulder and upper extremity, so changes to shoulder function may go undetected. Additionally, general health outcome scores tend to be affected more by lower extremity function than upper extremity function [13].

Because the upper extremity operates as part of the kinetic chain, its function is predicated by the concerted effort of the shoulder, elbow, wrist, and hand. Thus, any ailment affecting one of these joints will have an impact on upper extremity function. Limb-specific outcome instruments evaluate the influence of a single or multiple disorders of the upper limb on physical function, symptoms, and psychosocial issues. These types of questionnaires are specially designed to better detect changes in upper extremity function when compared with general health outcome instruments. Additional advantages include its utilization when more than one part of the upper extremity is involved or when the diagnosis is less certain. The evaluation of limb-specific instruments has found a close correlation with general health, joint-specific, and condition-specific outcome measures; however, they are less sensitive to changes in shoulder function compared with joint-specific and condition-specific instruments [4, 12, 16].

Joint-specific and condition-specific outcome measures concentrate on factors directly related to the shoulder or a particular condition of the shoulder, respectively. These specialized questionnaires are best able to detect small changes in shoulder function, with condition-specific outcome measures being the most sensitive to small changes in the condition for which they were designed. However, as these questionnaires become more specific toward a particular joint or pathology, they become less valuable in appraising

overall health and function, especially the more mental and emotional aspects. Also, the evaluation of a narrowed patient population prevents comparisons between different conditions, anatomic sites, and interventions.

A general health outcome measure should always be included as part of the patient evaluation because of its ability to evaluate the impact of a shoulder condition or treatment on the overall health of a patient. In addition, the instrument's ability to compare a shoulder condition to other systemic diseases is an advantage. Because of its limited upper extremity content, the clinical evaluation of a shoulder patient should also include either a limb-specific or joint-specific outcome measure. The addition of a condition-specific outcome measure is usually only needed for research purposes, in which a very specific patient population is being evaluated.

Measurement Properties of Outcome Measures

Once the appropriate categories of shoulder outcome measures have been decided, the next step is determining which specific outcome measure to employ. With over 40 different instruments for functional assessment of the shoulder, several factors are involved in the decision-making process of which outcome measurement tool to use. Each specific measure has specific strengths depending on the population being assessed and the reason for using the instrument. An outcome measurement tool must be context specific, and selection should be based on evidence that the instrument has the necessary measurement properties in the population being sampled for a study or assessment. The quality of an outcome measure can be assessed objectively, and given the plethora of outcome measures that have been developed, it is advisable to use an outcome measure for which there are data on its measurement properties. The measurement properties of an outcome measure that are important to clinicians are reliability, validity, responsiveness, and level of measurement (Table 50.2).

Reliability describes the repeatability of an outcome score taken at different settings from patients with a stable condition. Shoulder outcome instruments should be sufficiently reliable such that the score derived from the instrument does not change if the patient's clinic problem has remained the same, even if the questionnaire is completed on different occasions. Quantifying this measurement property is accomplished through test-retest analysis, in which individuals complete the same questionnaire on more than one occasion and the difference in scores is statistically analyzed [17]. Most problems with test-retest reliability occur because of the wording of the items, and as such there is sometimes difficulty when a questionnaire is developed in one country and used in another [18]. Moreover, some patients with certain conditions have problems with consistency in their questionnaires [19]. Therefore, an outcome measure can only be considered reliable for the specific condition evaluated.

Validity is the degree to which an outcome score accurately reflects or assesses the condition being measured [17, 20]. Validity has several different facets and thus cannot be appraised by a single statistic, instead requiring a body of evidence demonstrating a relationship between the true functional status of the patient and the score obtained from the instrument. *Face validity* is the degree to which a test appears to measure what it is intended to measure. The extent to which an outcome measure covers all the important domains of the condition being measured is referred to as *content validity*. An outcome measure evaluating shoulder instability would lack content validity without questions regarding apprehension and overhead activity. Both face validity and content validity are considered lower levels of validity, as they cannot be examined experimentally and can only be evaluated subjectively. The higher forms of validity, namely, criterion and construct, can be objectively examined with different statistical measures. *Criterion-related validity* compares the accuracy of the instrument to a gold standard or

Table 50.2 Measurement properties of outcome measurement tools

Property	Definition	Examples
Reliability	An outcome instrument generating similar scores for a patient with a stable condition	Test-retest reliability
Validity	The accuracy of an instrument regarding the impairment of a patient's condition	Face validity Content validity Criterion-related validity Construct validity
Responsiveness	The ability of an instrument to detect a change in a patient's condition	Minimal clinically important difference Ceiling effect Floor effect
Level of measurement	The measurement scales used for responses to the questionnaires	Yes-or-no Likert scale Visual analogue scale

another outcome score that has been previously validated for the specific condition. *Construct validity* assesses the instrument's ability to measure the underlying concept of interest [17, 20]. Shoulder outcome measures with construct validity demonstrate scores that compare with patient-derived and physician-derived assessments of the severity of the shoulder impairment, the level of pain, the ability to perform normal activities of daily living, and the responses to other contemporary patient-completed questionnaires [4]. Collectively, evidence demonstrating that an outcome measure has validity for a specific shoulder condition indicates the resultant score will yield an accurate account of its impact on a patient's symptoms and function.

The ability of an outcome measurement tool to detect a true change in a patient's condition defines *responsiveness* [21]. One of the primary roles of an outcome instrument is to evaluate the changes in the condition of patients following different therapy modalities. As such, several authors believe that responsiveness is the most important property of an outcome tool. Responsiveness is calculated by defining a cohort of patients whose health condition has changed between testings.

The *minimal clinically important difference* (MCID) of an outcome measure is the minimum change in a score that indicates a change in disability. An understanding of MCID is critical for an accurate appraisal of orthopedic literature, as well as when selecting appropriate instruments for research. A statistically significant improvement in an outcome score following a certain therapy would not be clinically significant if the improvement is not greater than the MCID. Therefore, when deciding between outcome measures, the smaller the MCID, the more sensitive the instrument in picking up small changes in a patient's outcome. Additionally, outcome measures may have ceiling or floor effects, which would adversely affect the responsiveness of the instrument.

The *ceiling effect* occurs when patients score so high on the scale that improvements in their impairments cannot be detected. The *floor effect* occurs when patients score so low on the scale that declines in their disability cannot be detected. The potential for ceiling or floor effects, together with MCID, are important properties when evaluating outcome instruments as measures of change in a patient's condition.

Outcome measurement tools utilize different levels of measurement, or measurement scales, in their questionnaires. Among shoulder outcome measures, responses to questions can be yes-or-no answers, Likert scales, or visual analogue scales. The more complex the response methodology is, the more difficult the questionnaire is to complete by the patient and to be analyzed by the clinician. However, these more complex modalities allow for an improvement in the responsiveness of the instrument [4]. Yes-or-no answers limit the number of possible responses by the patient, making completing and analyzing the questionnaire relatively easy, but also limiting the instrument's responsiveness (Fig. 50.1a). The Likert scale is an ordinal scale of responses to a question or statement ordered in a hierarchical sequence (Fig. 50.1b). The number of responses can vary from two responses to seven, but typically has four or five responses. The increasing complexity adds to the instrument's responsiveness, but may be more difficult for patients to complete and for the clinician to analyze. Finally, the visual analogue scale displays a scale as a straight line without any discrete choices, differentiating it from the Likert scale (Fig. 50.1c). Patients respond to questions by indicating a position along a continuous line in between two end points. Evidence has shown that a visual analogue scale has superior metrical characteristics than discrete scales; thus, a wider range of statistical methods can be applied to the measurements [22].

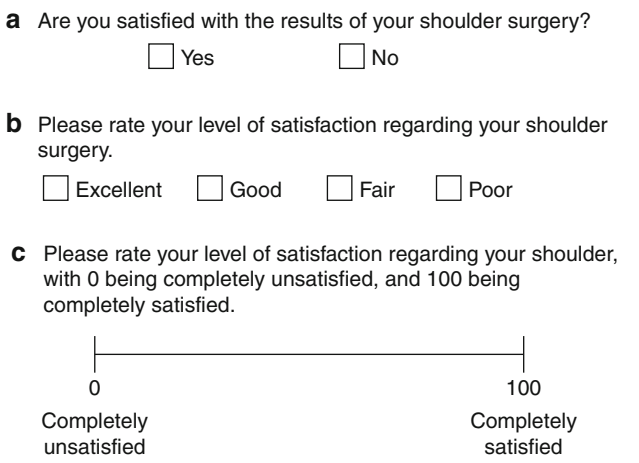

Fig. 50.1 Measurement scales in shoulder outcome measurement tools. (**a**) Yes-or-no scale. (**b**) Likert scale. (**c**) Visual analogue scale. The same question with different responses increases the responsiveness of the item, but also increases the complexity for the patient answering the question and the clinician quantifying the answers

As illustrated with the levels of measurements, increasing the complexity of the questionnaire can increase the level of data and information obtained. However, it also increases the difficulty from both the physician and patient perspective. The evaluation process for outcome measurement instruments needs to be based on both the measurement properties of the outcome score and the practicality of the questionnaire. Given the increasing paperwork demands being placed on both patients and physicians, the administration of these questionnaires needs to be feasible for both parties. From the patient's perspective, the questionnaire needs to be easy to understand, simple to answer, and not too time-consuming. Different factors the physician must account for include the time required to administer and score the questionnaire, the potential training or staff required to manage the outcome scores, and the availability of normative data. These factors

can establish whether an outcome measurement tool is seamlessly integrated into a clinical practice, or inundates it with increasing workload and more paperwork.

General Health Outcome Measurement Tools

Medical Outcomes Study 36-Item Short Form (SF-36)

The medical outcomes study 36-item short form (SF-36) is the most popular general health outcome measure [23]. It is a shortened version of a 149 validated health-related questionnaire originally reported as part of a medical outcomes study of more than 22,000 patients [24]. The questionnaire consists of 35 items in eight health domains and one general overall health status question that assesses the patient's perception of changes in health (Table 50.3). The SF-36 is designed to be a self-administered paper or computer questionnaire, taking an average of 5–10 min to complete. Each health domain is scored from 0 (worst possible health, severe disability) to 100 (best health, no disability), with normative population data available for comparison [25] An alternative scoring system consolidates these eight health domains into two components, a physical component summary (PCS) and a mental component summary (MCS) score. No single, total score is quantified for the SF-36.

A scoring manual is available for the SF-36 that provides information regarding its measurement properties and provides normative data for the eight health domains and two component summary subscales [25]. A multitude of studies have demonstrated a high level of reliability among patients with similar medical conditions [26, 27], as well as validity across a wide number of medical disciplines [13]. Validation studies have found the SF-36 able to differentiate psychiatric and physical illnesses, as well as discriminate several major medical illnesses from moderately ill or healthy individuals [25, 28]. However, the SF-36 is limited in its responsiveness, with limited data describing the instrument's ability to detect changes in clinical status on all eight subscales of the questionnaire [29].

The SF-36 has been employed in a number of different ways for the functional assessment of the shoulder. One main advantage of the SF-36 is its ability to determine the impact of a condition on a patient's quality of health. Gartsman et al. [15] demonstrated that 544 patients with five common shoulder conditions compared closely in quality of health with five major medical conditions: hypertension, congestive heart failure, acute myocardial infarction, diabetes mellitus, and clinical depression. The SF-36 can also be utilized to evaluate the outcomes of different treatment options. McKee et al. [30] exhibited improvements in SF-36 scores for pain, physical role functioning, and vitality following an open acromioplasty and subacromial bursectomy in 71 patients. A prospective study evaluating rotator cuff repair for chronic rotator cuff tears found a significant difference in SF-36 scores between workers' compensation patients and nonworkers' compensation patients [31]. Overall, SF-36 scores have been found to correlate well with shoulder-specific scores, despite being less responsive and reliable [32–34]. Because of its lack of shoulder function content, most authors would agree that an SF-36 questionnaire should be used in combination with another more shoulder-specific questionnaire for a more complete assessment of the shoulder patient.

Limb-Specific Outcome Measurement Tools

Disabilities of the Arm, Shoulder, and Hand (DASH) Score

The Disabilities of the Arm, Shoulder, and Hand (DASH) score was developed for the evaluation of single or multiple disorders of the entire upper extremity. The DASH evaluates the physical function (21 items), symptoms (6 items), and social or role functions (3 items) of the upper extremity in this self-administered, 30-item questionnaire. Two optional sections are also included that assess work activities (4 items) and sports and/or performing arts activities (4 items). Interestingly, the questionnaire does not differentiate which arm, the injured or uninjured, is needed to perform the activity. Rather, the questionnaire produces a score of patient function employing both upper extremities. This feature is both an advantage and disadvantage of this instrument. The DASH score ranges from 0 to 100, with a higher score reflecting greater disability [35] and normative data available for comparison [36].

The DASH questionnaire is intended to be used for any upper extremity condition, including all shoulder conditions. However, this outcome instrument has been specifically validated for glenohumeral arthritis, rotator cuff tendonitis, total shoulder arthroplasty, rotator cuff repair, and psoriatic arthritis [37–41]. Although no specific age limit has been set for this instrument, it is recommended in patients between the ages of 18 and 65 years old. The minimal clinically important difference was calculated to be 10 for the shoulder and 17 for more distal joints of the upper extremity [42].

As a limb-specific outcome measurement tool, the DASH questionnaire has demonstrated improved responsiveness with fewer ceiling or floor effects than the SF-36 [16]. These results are not surprising given the increased emphasis on upper extremity function rather than general health. However, the broad scope of the DASH encompassing upper extremity function as a whole limits its usefulness to shoulder-specific

Table 50.3 Outcome measurement instruments for functional assessment of the shoulder

Outcome instrument	Category	Target population	Items/time to administer	Health domains	Measurement scale	Score (worst to better)	Validated parameters	Minimal clinically important difference
SF-36	General health	All medical disciplines	31	Physical functioning General health Emotional well-being Vitality Physical role Emotional role Pain Social functioning	Primarily Likert scale, also yes-or-no scale	0 to 100	Yes	Not defined
DASH	Limb specific	Upper extremity disorders	30 (+ 8 optional)	Physical function Symptoms Social/role function Work activities (optional) Sport/music activities (optional)	Likert scale	100 to 0	18–65 years Glenohumeral osteoarthritis Rotator cuff tendonitis Total shoulder arthroplasty Rotator cuff repair Psoriatic arthritis	10.2
UCLA	Joint specific	Shoulder disorders	5 items (3 patient based, 2 clinician based)	Pain Function Forward flexion motion Forward flexion strength Patient satisfaction	Likert scale	00 to 35	Not validated	Not determined
Constant	Joint specific	Shoulder disorders	8 items (6 clinician based, 2 patient based)	Pain Activities of daily living Range of motion Strength	Likert scale	0 to 100	14–85 years General shoulder patient RCR, adhesive capsulitis, prox humerus fx	Not determined
ASES	Joint specific	Shoulder disorders	10	Pain Instability Activities of daily living	Likert scale	0 to 100	20–81 years General shoulder patient Rotator cuff disease Glenohumeral osteoarthritis Shoulder instability Shoulder arthroplasty	6.4

SPADI	Joint specific	Shoulder disorders	13	Pain Activities of daily living	Visual analogue scale	100 to 0	General shoulder patient Rotator cuff disease Glenohumeral osteoarthritis Glenohumeral rheumatoid arthritis Adhesive capsulitis Total joint arthroplasty	8–13
SST	Joint specific	Shoulder disorders	12	Pain Function Range of motion	Yes-or-no scale	0 to 100	General shoulder patient Glenohumeral osteoarthritis Rotator cuff disease	17.1–25.0
SANE	Joint specific	Shoulder disorders	1	Shoulder percentage of normal		0 to 100	All shoulder diagnoses	Not determined
Shoulder activity level	Joint specific	Shoulder disorders	7	Five different functional tasks	Likert scale	0 to 20	RCR, OA, instability	Not determined
WOOS	Condition specific	Osteoarthritis	19	Pain and physical symptoms Sports, recreation, and work Lifestyle function Emotional function	Visual analogue scale	1,900 to 0	OA, TSA	Not determined
WOSI	Condition specific	Shoulder instability	21	Physical symptoms Sports, recreation, and work Lifestyle Emotions	Visual analogue scale	2,100 to 0	Shoulder instability	220
WORC	Condition specific	Rotator cuff tears	21	Pain and physical symptoms Sports and recreation Work function Social function Emotional function	Visual analogue scale	2,100 to 0	Rotator cuff disease	245.26
RCQOL	Condition specific	Rotator cuff tears	34	Symptoms and physical complaints Sports and recreation Work Lifestyle Social issues Emotional issues	Visual analogue scale	0 to 100	25–83 years Rotator cuff disease	Not determined

conditions. In fact, Dowrick et al. demonstrated the DASH score does not exclusively assess disability in the upper extremity and is affected in patients with lower extremity injuries [43]. Furthermore, many of the items within the questionnaire are irrelevant to patients with shoulder complaints, limiting the test's usefulness for shoulder-specific patients. The validity of the DASH score in intercollegiate athletes may be limited by a ceiling effect because of the high overall function of this population [35]. Beaton et al. also showed a general lack of validity and responsiveness of the DASH score when compared with disease-specific outcome measures of the shoulder. In general, the DASH questionnaire is a useful outcome measure for patients presenting with general upper extremity complaints without a specific diagnosis, rather than patients with specific shoulder complaints in which a shoulder-specific or condition-specific outcome instrument has demonstrated improvement measurement properties.

Joint-Specific Outcome Measurement Tools

UCLA Shoulder Score

One of the first outcome measurement tools created primarily for the shoulder is the UCLA shoulder score [44]. This outcome instrument evaluates shoulder pain, function, forward flexion motion, forward flexion strength, and overall satisfaction with the shoulder. Since its inception in 1981, this instrument has been utilized for nearly every shoulder condition [45–49] and continues to be popular given its historic standing. However, it has never been formally validated because there were no comparable tests at the time of its development. Also, the combination of self-administered, subjective evaluations with "objective" physical examination findings increases the inaccuracies and biases that can adversely affect the score results. As such, recent comparison studies have identified poor reliability, validity, and responsiveness associated with this score [50, 51].

Constant Score

Originally published as a master's thesis, the Constant score is the most commonly used shoulder evaluation instrument in Europe [50] and has been recommended by the European Society for Surgery of the Shoulder and Elbow (SECEC) and the *Journal of Shoulder and Elbow Surgery* to be a mandatory evaluation tool for all shoulder publications and presentations [52]. The Constant score comprises a patient self-assessment portion evaluating pain and activities of daily living, as well as a physical examination component that attempts to assess active range of motion and strength of the shoulder [53]. This measure has a 100-point scale, with 100 points being the best score; 35 points are derived from the patient's self-assessment, while 65 are generated from the physical examination. Similar to the UCLA score, the combination of a physician-based and physical examination domain increases the risk of bias and measurement error, generating instrument imprecision and inaccuracy.

An original validation study was performed for 25 patients with a wide variety of shoulder conditions including arthritis, instability, and impingement [54]. The score was easy to use, but was not very reliable and demonstrated a ceiling effect in instability patients. Further studies evaluating the Constant score raised several concerns regarding its reliability, correlation with other shoulder scores, scoring methods that increase the risk for bias, and variability in objective testing measures [52, 54–57]. In addition, age-related declines in scores and strength occur for both sexes [57–59]. Despite these limitations, the Constant score has been validated for total shoulder arthroplasty [37], rotator cuff repair, adhesive capsulitis [60], and proximal humerus fractures [61]. To improve upon the inherent weakness associated with objective testing measures, the Constant score has attempted to better define the methodology and measurements of strength and range of motion to produce more consistent results [52].

American Shoulder and Elbow Surgeons (ASES) Outcome Score

The American Shoulder and Elbow Surgeons (ASES) society developed their own questionnaire in 1994 with the goals of creating a scoring system that would allow consistent communication between physicians, stimulate the undertaking of multicenter trials, encourage validity testing of shoulder outcome measures, and create a scoring system that could be utilized in all patients with any shoulder condition [51, 62]. The ASES outcome score consists of a 10-item patient self-assessment containing domains in pain, instability, and activities of daily living, as well as a physician-directed domain which is rarely used. The questionnaire takes about 3–5 min to complete and about 2 min to score [63, 64]. The raw score is converted to a 100-point scale, with 100 points being the best score, and normative data is available to compare [65].

The ASES outcome score has demonstrated reasonable reliability, responsiveness, and validity for patients aged 20–81 years old with a wide variety of shoulder diagnoses managed both operatively and nonoperatively [64]. More specifically, the ASES outcome score has been validated for rotator cuff disease, glenohumeral osteoarthritis, shoulder instability, and shoulder arthroplasty [37, 66]. The MCID has been quantified to 6.4 for general shoulder problems [64]

and has also been estimated to be 12 for rotator cuff disease [67]. The ASES correlated well with other shoulder-specific outcome measures, but often times did not correlated with the SF-36 [68–70].

Shoulder Pain and Disability Index (SPADI)

The Shoulder Pain and Disability Index (SPADI) was developed by a group of rheumatologists to measure the pain and disability associated with "the clinical syndrome of the painful shoulder" of musculoskeletal, neurogenic, or undetermined origin [71]. This questionnaire consists of five items related to pain symptoms and eight items related to physical function and disability, and takes between 2 and 5 min to complete. The score is converted to a 100-point scale, with 0 points being the best score and 100 being the worst score.

The SPADI, the DASH, and the ASES are the three most studied shoulder outcome measurement tools in terms of their measurement properties [72]. The reliability and responsiveness have been found to be acceptable in a variety of patient populations and in patients with improving and deteriorating conditions [69, 72, 73]. Early validation studies focused in the primary care settings on heterogeneous populations with a wide variety of shoulder conditions [69, 71]. More recently, the SPADI has been validated for rotator cuff disease [74], osteoarthritis, rheumatoid arthritis [75], adhesive capsulitis [76, 77], and joint replacement surgery [78]. The MCID has been reported to be between 8 and 13 [64, 79]. Overall, the SPADI was found to be more responsive and reliable than general health outcome measures, and correlate well with other shoulder-specific outcome tools [72, 80].

Simple Shoulder Test (SST)

The Simple Shoulder Test (SST) was designed to be a simple and efficient instrument to characterize the severity of a condition and functional improvement seen after a surgical procedure. The questionnaire focuses on 12 different functional tasks that focus on pain, function/strength, and range of motion [81]. Response items employ the yes-or-no scale, allowing the questionnaire to be completed in less than 3 min. This dichotomous response option allows for excellent reliability [82, 83]; however, its simplicity does impact the validity and responsiveness. Criterion-related validity is lacking for this instrument, as there is no gold standard for comparison and most other shoulder outcome instruments use different scales for their responses. Several evaluations of the SST have found the questionnaire to have a high degree of reliability, as well as acceptable content and construct validity [72, 83, 84] for a wide variety of shoulder conditions treated both nonoperatively and operatively. In general, the responsiveness of the SST is weaker compared to other shoulder outcome measures because of its binary response choices. Several authors illustrated that the SST cannot be used to differentiate patients with varying degrees of the same condition [50, 85]. Godfrey et al. [83] found decreasing responsiveness in younger patients and patients with instability. Limited data is available regarding MCIDs for different conditions, but recent studies have calculated the MCID to be 17.1 for rotator cuff disease [67] and 25.0 for shoulder arthroplasty [86], which are substantially higher than other shoulder outcome tools. Therefore, despite being highly reliable and valid for most nonoperative and operative shoulder conditions, most authors are cautioning the use of the SST for clinical use given its decreased responsiveness.

Single Assessment Numeric Evaluation (SANE)

The simplest outcome measure, the Single Assessment Numeric Evaluation (SANE), attempts to quantify the shoulder function into one question: "How would you rate your shoulder today as a percentage of normal (0–100 %, with 100 % being normal)?" [87]. This outcome measure is inherently used in most clinical offices in evaluating patient progress. Several studies have reported its use for a variety of shoulder conditions [87–90], but it has yet to be validated and no other measurement properties are known.

Shoulder Activity Level

Most shoulder outcome measurement tools evaluate some aspect of pain and function in their questionnaire. Patients can artificially elevate their scores by decreasing their activity level, which decreases their pain while still allowing them to accomplish most activities of daily living. This is especially evident in outcome measures susceptible to the ceiling effect, in which high-level athletes are able to score high on questionnaires despite having significant disability in their sport or activity. Thus, the shoulder activity level was developed as a supplementary tool to evaluate their current activity level associated with their other outcome measures [91]. The test evaluates five different activities: carrying weights ≥8 lb by hand, handling objects overhead, weight lifting or weight training with the arms, executing a swinging motion (e.g., golf, baseball), and lifting objects weighing ≥25 lb. Each task is graded from 0 to 4 in terms of frequency performed per month: never or once per month (0 points), once per month (1 point), once per week (2 points), more than once per week (3 points), or daily (4 points). Validation studies have been performed in patients with rotator cuff dis-

ease, glenohumeral osteoarthritis, and glenohumeral instability [92], but an MCID is not known.

Condition-Specific Outcome Measurement Tools

Western Ontario Shoulder Outcome Instruments

Three condition-specific outcome measurement tools for the shoulder were developed at the University of Western Ontario from 1998 to 2003. Outcome measures for shoulder instability (Western Ontario Shoulder Instability Index, WOSI), glenohumeral osteoarthritis (Western Ontario Osteoarthritis of the Shoulder Index, WOOS), and rotator cuff pathology (Western Ontario Rotator Cuff Index, WORC) were established by reviewing the literature and preexisting scoring systems, interviewing orthopedic surgeons and physical therapists, and interviewing patients with the specific condition [51]. The goal for these disease-specific instruments was utilization as a primary outcome measure in clinical trials evaluating treatments.

The WOSI and WORC are 21-item questionnaires, while the WOOS is a 19-item questionnaire. All outcome instruments are subdivided into health domains concerning physical symptoms, sport/recreation/work function, lifestyle function, and emotional function. The response items used a 100-mm visual analogue scale, with the score summations from the items ranging from 0 to 2,100 for WOSI and WORC and 0 to 1,900 for WOOS, with higher raw scores representing worse function. Each test takes about 10 min to complete.

The measurement properties of the WOSI, WOOS, and WORC have been evaluated by the developers of the instrument. As characteristic of condition-specific outcome measures, all three instruments demonstrated the most responsiveness to small changes in their respective condition compared to all other outcome measurement tools [50]. The estimated MCID for WOSI is 220 and for WORC is 245 [26, 50]. No MCID has been established for the WOOS. The developers also concluded that their outcome instruments were reliable and valid for their specific condition [93–95]. Overall, the homogeneity of the population studies and the use of the visual analogue scale help to make these measurement properties the strongest of any outcome tool. However, other than the developers' data, there is not much evidence evaluating the reliability, validity, and responsiveness of these tools. Due to this lack of testing data, caution is necessary for measurement at an individual patient level.

Rotator Cuff Quality of Life (RCQOL)

The Rotator Cuff Quality of Life (RCQOL) score was developed for the evaluation of large and massive rotator cuff tears [96]. It consists of a 34-item questionnaire with health domains evaluating symptoms and physical complaints, sports and recreation, work function, lifestyle, social issues, and emotional issues. The responses are recorded using the visual analogue scale, with scores ranging from a low, worst outcome of 0 and a high, best outcome of 100. It has been validated for patients aged 25–83 years old with different forms of rotator cuff pathology [97]. The MCID has not been determined.

Summary

Patient-based outcome measurement tools are becoming increasingly popular as the modality of choice for patient evaluation. Utilization of these tools allows for an unbiased and accurate account of a condition's impact on a patient's health, including pain and physical symptoms, sports and recreational function, occupational function, mental health, social issues, emotional issues, and impact on general health. The functional assessment of the shoulder typically involves a general health outcome measurement tool along with a shoulder-specific or disease-specific outcome measurement tool. Scientific evidence of an instrument's measurement properties, including reliability, validity, and responsiveness for the condition of interest, is appraised when deciding on which specific instrument to utilize. The goal is choosing an outcome measurement tool that is able to accurately appraise the disease state, as well as recognize any change in that state. Ultimately, these quantifiable values can be utilized as critical feedback for treating physicians, provide transparency of different treatment options to patients, and give values to surgical procedures for third-party payers.

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Self-Assessment of Treatment Outcomes in Shoulder Arthroscopy

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Nicholas G.H. Mohtadi

Introduction

The assessment of outcomes after any type of surgery can be categorized in a variety of different ways. Simply put, the outcome of a procedure can be anything that is measured or observed. It can range from something as simple as measuring shoulder range of motion using a goniometer in a single plane, to a complex, multifaceted, disease-specific, health-related quality-of-life outcome questionnaire.

Similarly, measuring the overall outcome of a particular arthroscopic procedure can be considered in many ways and from differing perspectives. The healthcare system has a different perspective than the surgeon and most importantly so does the patient. The healthcare system whether it is represented by a government payer or private insurer should be primarily interested in the "value" of the arthroscopic treatment. Therefore, the system needs to consider the resources and costs in addition to the overall outcome of the patient. Simply put this would be a measure of cost-effectiveness. The system would require the ability to measure cost-effectiveness across several disease states, between hospital and clinics, between surgical procedures, and surgeons and also consider patient-related outcomes. In this way, the system could compare the value of treating a patient with a shoulder problem to one with a knee problem or a completely different disease entity. Therefore, from the perspective of self-assessment, the outcome would need to be generic in order to apply to patients with a variety of diseases and problems. An example of this type of self-assessment outcome would be the SF-36 [1–3]. The problem with a generic self-reported outcome measure is that it is unlikely to evaluate patients with shoulder problems to the same extent as a shoulder-specific outcome measure [4].

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It should be made perfectly clear at the outset that no one outcome measure will be able to serve all purposes all of the time. It is also very necessary to recognize that each perspective has its own bias. From the healthcare system perspective, the financial impact of the treatment may be the most important characteristic to measure. From the patient's perspective, the negative impact of their problem on their quality of life and their improvement with treatment is paramount. From a surgeon's perspective, the correction of the pathology, determination of healing, and type of surgical procedure may be critical issues with respect to outcome assessment.

Furthermore, outcomes can be subjective or objective and assessed generically, or in joint-specific or disease-specific terms, and can be discriminative, evaluative, or predictive. Outcomes can be considered objective; this means that they are undistorted by emotion or personal bias and based on observable phenomena. These observable phenomena are reproducible and quantifiable. Outcomes can also be described as subjective, which means that the effect takes place within the mind and is modified by individual bias. The irony of this categorization when it comes to measuring outcomes in medicine or surgery is that we consider something like an x-ray to demonstrate objective data but visual analogue pain assessments to represent subjective information. However, the fact is that the interpretation of an x-ray image is open to observer bias and therefore has a component of subjectivity. By contrast, a patient's response to a visual analogue pain scale can be reproduced and assessed for error, measured for reliability, and therefore quantified: these are the essential properties of an objective measurement. Whether the outcome is objective or subjective is not at all critical. It is far more important to understand the measurement properties of the tool or instrument of measurement. For example, we can appreciate that using a goniometer to assess the amount of external rotation in the shoulder has a certain amount of error. A long-arm goniometer will likely have less inherent error compared to a shorter instrument. If we are treating a stiff shoulder with physiotherapy, serial range-of-motion measurements are critical to determine the

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response of treatment. If the goniometer has an error of ±5°, then an improvement from 25° to 30° may be irrelevant and demonstrate that the treatment is ineffective. If we then include 100 patients and the average gain in external rotation is the same value, and it is statistically significant, this improvement is still irrelevant and of no clinical importance. The same concepts apply to patient-reported outcome measures. These outcomes can be patient derived, determined by a consensus of experts, administered in a variety of different ways, and utilize various response formats [e.g., Likert scale, visual analogue scale (VAS), ordinal scales, nominal scales]. It is critical to understand the measurement properties of these tools just as clearly as we appreciate the inaccuracies of a goniometer.

The reason we measure outcomes is to gain a clear understanding of how our patients are doing with respect to a particular procedure. This understanding must be representative of the truth; otherwise, we will make mistakes along the way.

This chapter will attempt to provide a brief but general overview of outcome assessment and a focused review on the self-assessment of treatment outcomes in shoulder arthroscopy.

Overview of Outcome Assessment

In order to measure the outcome of surgical treatment in a patient with a shoulder disorder, we must understand that the outcome is absolutely contingent on three independent constituents or variables.

Firstly, we need to consider the *patient*. Patient demographics, the specific shoulder disorder, the natural history of this disorder, the extent of the disease, the impact of this problem on the patient, and any associated characteristics or comorbidities are likely to influence the outcome of surgical treatment. To better understand the results of surgical treatment between studies, it is necessary to understand the patient population, the sampling frame, and how the patients were selected into the study. We can account for many of these patient characteristics and match as many characteristics as possible. However, patients also have inherent biases. These biases are not likely to be known, predictable, or anticipated. Therefore, the only way to account for patient-related bias is to randomize patients to one treatment or the other [5, 6]. The randomized clinical trial (RCT) study design creates the best opportunity to address differences between patients, in order to measure the outcome of a particular treatment. One would anticipate that any known or anticipated biases would be equally distributed between groups. This can be illustrated by using an example from the literature. Haahr et al. published an RCT comparing physiotherapy to arthroscopic subacromial decompression [7]. The randomization resulted in very comparable patient groups at baseline with almost identical age, work characteristics, previous treatment, clinical findings, duration of symptoms, and Constant scores. To create such consistency with any other study design would be lucky or would require matching on every one of these characteristics. Therefore, from the patient's perspective this trial had excellent internal validity. The authors did not comment on who refused to be in the trial, withdrew consent, or were excluded. If the characteristics of those not included were distinctly different and were biased in one way or another, then it would be impossible to generalize the information from this study to our own population of similar shoulder pain patients. The RCT minimizes the effect of bias between patients by randomly allocating patients to each treatment group, but it is necessary to understand the sampling frame from the population of patients with shoulder pain in order to fully understand the outcome assessment.

The second independent component is that which is attributed to the *surgeon/surgical procedure*. Surgeons are in a relatively unique position in medicine because patient outcome is based to a greater or lesser extent on the surgical procedure performed, the surgeon's approach to the patient, patient selection, and the skill of the surgeon. It is not necessarily appropriate for an individual surgeon to simply quote results from the literature and apply them to his/her patients. The administration of a medication would be expected to have the same effect and therefore patient outcome anywhere in the world, assuming that patients were similar in their disease state and demographic characteristics. The equivalent cannot be said for surgical treatment on similar patients. Therefore, surgeons should be obligated to measure outcomes in some meaningful way. The impact of surgical experience is well understood. This has been addressed in the context of performing clinical trials by using an expertise-based design [8–10]. In this design patients are randomized or matched to surgeons who perform a particular procedure. The surgeon is comfortable and experienced in the particular procedure. Therefore, the outcome of the study is more likely to pertain to the procedure, rather the surgeon. An example where this may be important is the study by Bottoni et al. [11]. This randomized trial showed that there was no difference in clinical outcomes comparing arthroscopic stabilization to traditional open stabilization. However, one criticism of this trial is that the arthroscopic procedure took on average 59 min compared to 149 min for the open procedure. Therefore, one may interpret that the surgeons were more skilled in the arthroscopic procedure. An expertise-based design would presumably compare surgeons who are able to perform the open procedure in a similar length of time [10].

The surgical procedure can also be measured by considering whether or not a complication occurred, pre- and post-op x-ray findings, as well as many other technical details of the

surgical procedure. Assessing the surgical procedure may also be necessary to provide outcome information for third parties such as insurance companies, government organizations, or Workers Compensation Boards. In these cases, reporting information on Key Performance Indicators (KPIs), such as measures of safety, use of antibiotic and DVT prophylaxis, hospital stay, and costs, would be necessary. These types of outcomes are typically objective measures, which are observed, counted, and described in a reliable and valid way. Assuming that an unbiased observer documents this information independently, this can be trusted and represent the truth.

The third independent variable or component (the most important within the context of this chapter) is *the outcome measure* itself.

Outcome measures can be called instruments, tools, scales, scores, indices, measures, outcomes, or questionnaires. These terms are used interchangeably for the purpose of this chapter. Outcome measures can be classified in many ways. The purpose of outcome measures can be classified as either disease specific, such as those tools created to assess osteoarthritis, or joint specific, such as those created to assess the outcome of any pathology of the shoulder. These measures can also be classified according to the person who completes the assessment. Traditionally, outcomes have been assessed by clinicians and include objective measures such as radiographic assessments. The clinician also asks the patient about pain and other subjective measures. These "clinician-based" or "clinician-administered" tools may introduce bias due to the way they are administered but more importantly may not capture the patient's perceived outcomes [12]. The Constant score represents a shoulder-specific outcome that includes both a clinician and patient-based components [13, 14]. More recently, patient-based and administered tools have been created [15–25]. These patient-reported outcomes (PROs) are typically self-administered and can be completed in a nonthreatening environment to the patient. Patient-reported outcomes are considered to be the reference standard for reporting clinical trials. It is necessary to distinguish between self-administered (i.e., by the patient) outcomes from those which are not only self-administered but are also patient-derived or determined outcomes. There is some debate regarding what constitutes a patient-reported outcome [26–30]. The commonly accepted definition is: "any report coming directly from patients, without interpretation by physicians or others, about how they function or feel in relation to a health condition and its therapy" [28]. This definition works very well for simple outcomes such as measuring pain intensity over time using a visual analogue scale. The PRO takes on a different context when one is attempting to measure more complicated concepts such as quality of life (QoL). A more complete definition of a PRO is that it is collected from a patient but more importantly the "information gained is necessarily of direct concern to the patient" [26]. It is well recognized that the patient perspective is different from that of the clinician and most importantly the surgeon [12]. Therefore, if we accept the first and simple definition of a patient-reported outcome, i.e., the patient is the source of the information, it becomes critical to define and/or label the content, construct, or concept of the specific PRO [31]. Typically, this content has included measures "that includes direct subjective assessment by the patient of elements of their health including: symptoms, function, well-being, health-related quality-of-life (HRQoL), perceptions about treatment, satisfaction with care received, and satisfaction with professional communication. The patient is asked to summarize his or her evaluation of the disease, treatment, or healthcare system interactions through various modes, providing perceptions related to the condition, its impact, and its functional implications" [29]. It is evident from the literature that there is discussion and debate regarding the definition of a PRO, what context it is measuring, and the importance of patient input, not to mention how it is analyzed and reported [26–32].

The objective of the tool must also be considered. If the goal is to follow patients over time and to assess changes, an evaluative index is necessary, because it can measure the magnitude of longitudinal change in an individual or a group of individuals [33]. If the objective is to differentiate among patients to determine treatment, a discriminative index should be used, because it distinguishes between individuals or groups [33]. It is very important to understand that the properties of each outcome measure change depending on the objective of the tool. One of the key properties of an evaluative index is the demonstration of responsiveness [33]. Responsiveness refers to the ability of the outcome measure or instrument *to detect within patient change over time* [34]. A discriminative index needs to *differentiate*, *between patients*, *at a particular point in time*, in other words being able to distinguish patients with more or less severe "disease" states. Guyatt has explained the differences between these two types of instruments by using the statistical concept of quantifying the signal-to-noise ratio [34]. The better the signal-to-noise ratio, the better the instrument. "If the variability between patients (the signal) is much greater than the variability within patients (the noise), an instrument will be deemed reliable" [34]. Discriminative instruments need to be highly reliable, and the questions included in these instruments must enhance the ability to measure variability. Evaluative instruments are subtly different in that they need to detect change over time and responsiveness is a reflection of that change. Responsiveness is "directly related to the magnitude of the difference in score in patients who have improved or deteriorated (the signal) and the extent to which patients who have not changed provide more or less the same

scores (the noise)" [34]. If the change over time is clinically meaningful, then a responsive instrument will be able to measure whether or not a specific treatment (i.e., surgery) has improved a patient's outcome. Finally, it is very important to understand how each item in a PRO is determined. It is this initial item pool through the process of item generation that is critical [34, 35]. Once a comprehensive item pool is identified, then the final set of items is reduced and formulated into the questionnaire [35]. "The procedure for achieving comprehensiveness is different when selecting an item pool for an evaluative instrument than for either a discriminative or predictive tool" [33]. In a discriminative index, it would be important to have the majority of the respondents to answer the questions, whereas in an evaluative index, all relevant and important aspects should be included in order to measure clinically important outcomes [33].

Several book chapters and review articles have been written on the topic of outcomes with respect to shoulder problems and treatment [36–48]. Rather than providing a comparative list of the available outcomes, this chapter will take a different approach. The chapter will use common hypothetical patient presentations in order to describe the ways to assess the outcome of shoulder arthroscopic treatment from the self-assessment, patient-reported perspective. In other words we should….

Base the Outcome on the Patient!

In order to understand the outcome of an arthroscopic procedure, we must start with the patient. The assumption is that we know that the patient has a shoulder problem. Depending on the particular setting, the patient may present to the surgeon with an undisclosed, undiagnosed shoulder-related problem. Let us call this patient presentation the generic shoulder patient, i.e., *Patient #1*. Since the diagnosis is unknown, asking the patient to fill in a complex shoulder outcome score would be unnecessary at this stage of presentation. A simple screening assessment may be very appropriate since there is no expectation of doing arthroscopic surgery at this point. The simplest scenario would be to globally screen the patient on arrival or during the history taking by asking them to rate their shoulder problem using the subjective shoulder value (SSV) [49] or the single assessment numeric evaluation (SANE) [24]. The SSV asks the patient, "What is the overall percent value of your shoulder if a completely normal shoulder represents 100 %?" [49]. Similarly, the SANE asks the question, "How would you rate your shoulder today as a percentage of normal (0–100 % scale with 100 % being normal)?" [24]. Yet another approach would be to use the classic example of the visual analogue scale (VAS) response format to report the amount of pain that the patient is experiencing on a scale of 0 to 100 where "0" represents no pain at all and "100" represents pain as bad as it could be [50]. The surgeon then completes the history, physical examination, and investigations; confirms the diagnosis; and determines the treatment course. Depending on the severity of the problem, the surgeon may be inclined to consider nonsurgical treatment or opt for surgery. The patient's score before and after treatment represents the treatment effect and therefore the benefit or not of the treatment. The advantage of this outcome assessment is the ease of use and simplicity with respect to analysis, and generally speaking these simple tools correlate with more complex outcome measures [24, 49]. The major criticism of these outcome measures is the fact that everything is taken into account with a "global" rating. It is not possible to glean from this "global" rating, which aspects of the patient-reported outcome are driving the score. For example, if the patient has no pain, then they might consider their shoulder to be 80 % even if they cannot move their arm, play sports, or work at their usual job. Similarly, it may be possible for a patient to score 80 % and still have pain if they are able to work and play sports. Nevertheless, a surgeon could legitimately use this approach to assess the outcome of his/her patients with particular surgical procedures. For example, the average improvement in VAS pain scores in patients undergoing arthroscopic rotator cuff repairs before and after surgery could be used as a measure of patient-reported outcome. This would be very simple; would be easy to administer, record, and analyze; and would be reliable, responsive to change, and valid in the context that pain relief is likely the most important patient-reported symptom.

The next "level" of patient outcome assessment would be to evaluate *Patient #1* with a shoulder-specific outcome tool. This would include outcomes such as the Simple Shoulder Test (SST) [51], the Shoulder Rating Questionnaire [52], the American Shoulder and Elbow Surgeons Evaluation (Subjective) Form (ASES) [53], the Oxford Shoulder Score (OSS) [15], the Shoulder Pain and Disability Index (SPADI) [54], and the Disabilities of the Arm, Shoulder, and Hand (DASH) [55, 56].

These outcome questionnaires are self-administered by the patients and could be used as a baseline/screening tool prior to the clinical assessment or after the history and physical examination prior to confirmation of the diagnosis. These tools are more sophisticated than the global assessment tools, are shoulder specific, and therefore provide the clinician with more information regarding the impact of the patient's problem. Kirkley et al. have critically evaluated these instruments [40]. The most essential characteristic of any patient outcome questionnaire is whether or not patients were fully represented at the item generation phase of development. If not, the tool may very well miss key items or characteristics that have a direct bearing on the patient's outcome. It is evident that what is important to a patient may not be important

to a surgeon and vice versa [12, 32]. Therefore, if we look at each of these shoulder-specific outcomes from the perspective of whether the questions represent patient-based complaints, it is possible to decide which questionnaire may be best suited.

The Simple Shoulder Test was based on previous tools and represents common patient complaints [42, 51]. This very practical tool is said to be reproducible, sensitive to shoulder problems, and able to quantify change over time [42, 51]. The 12 questions were assumed to be patient derived but were not truly patient generated.

The Shoulder Rating Questionnaire was developed from a preliminary questionnaire and subsequent patient input. It has six domains (global assessment, pain, daily activities, recreational and athletic activities, work and satisfaction) with differing response scales [52]. Formal assessment of reliability and construct validity was reported and responsiveness was suggested, but this has been questioned in subsequent publications [38, 40]. Patient input was utilized during the questionnaire development, but it is not clear if items were generated comprehensively.

The ASES subjective form was developed from existing questionnaires and a great deal of input from the research committee of the American Shoulder and Elbow Surgeons [53]. No direct patient input was included. There is an associated physician assessment component, but this part is not scored and therefore not assessed in any quantitative way. The ASES subjective self-evaluation includes 11 separate questions: One item assesses pain on a visual analogue scale, and the other ten items are function related [53].

The Oxford Shoulder Score (OSS) is a shoulder-specific outcome measure designed for the evaluation of shoulder conditions exclusive of patients with instability [57, 58]. Four separate groups of patients with painful shoulders related to a degenerative or inflammatory condition were interviewed and assessed to develop this 12-item questionnaire. A separate instability questionnaire was developed because of observations that this patient group had decidedly different symptoms related to the anticipation of subluxation or dislocations associated with specific activities [16, 57, 58]. The OSS is divided into 8 questions related to the activities of daily living and 4 related to pain. The original description used response scales from 1 to 5 points with a higher score representing more difficulty and pain [57]. More recently the authors have revised the scoring to have a response scale of 0–4 with a higher score representing a better outcome [58]. Although there was direct patient input, this questionnaire is focused on pain and activities of daily living only.

The Shoulder Pain and Disability Index (SPADI) was developed in an outpatient setting based on 37 male patients with shoulder pain primarily of musculoskeletal origin [54]. Originally, 20 items were identified based on the assessment of three rheumatologists and one physical therapist, and these were reduced to 13 questions in order to improve the ease of administration, reliability, and whether the item correlated with shoulder range of motion [54]. The SPADI consists of two subscales, pain (4 questions) and disability (8 questions) using a horizontal line visual analogue response format with 12 equally spaced segments with assigned values of 0–11. A higher score represents more pain or disability. Since patient input was limited to male outpatients, the SPADI would not be considered to be fully representative of patients with shoulder problems.

The Disabilities of the Arm Shoulder and Hand (DASH) is not strictly speaking a shoulder-specific questionnaire. However, it has been evaluated in the context of assessing shoulder patients and has been shown to be as good as or better than other shoulder-specific instruments [43, 59]. The DASH was not initially based on patient input but utilized 13 outcome scales and an initial list of 821 items [56]. Further development involved 200 patients from two centers with wrist/hand or shoulder problems [55]. The DASH is more comprehensive than any of the other shoulder-specific outcomes since it includes functional activities, pain assessment, social and emotional items, as well as optional sport and work-related sections [55].

It is not possible to determine exactly which is the best outcome for use in this scenario [38, 45, 59]. The SST is highly reliable due to its yes/no response format, but there is no measure of responsiveness. The OSS, SPADI, and ASES are similar in the sense that they have two components, pain and "physical function." The ASES is easier to score and the DASH is most comprehensive. The key issue with respect to choosing one of these outcomes in a surgeon's practice would be the type of patient that they see. If ones patient population is slightly older with more patients with rotator cuff disease, arthritis, and stiff shoulders, then any of these outcomes will do. If ones practice is upper extremity disorders, then use the DASH. If the patients are younger athletic and have instability, then only the optional component of the DASH would apply.

There are three practical considerations with respect to choosing an outcome. Firstly, the length of the questionnaire is always considered to be an issue for surgeons. "A shorter questionnaire is better." However, if you have a great deal of experience with patient-based questionnaires, it is evident that the length of the questionnaire is not an issue to the great majority of patients. The issue to the patient is whether they understand the questions and are the questions important to them or relevant to their situation. However, asking patients to fill in multiple questionnaires will result in responder burden and lead to poor compliance. The second concern is how the questionnaire is administered. Examples would be a paper form, desktop computer in the office or clinic, from a home computer using a secure

web-based application, or more recently using a handheld device. Over the years we have compared paper to computer-based format and found no difference between the two, with better acceptance of the computer format. However, there are patient populations that may not have the necessary computer savvy to feel comfortable enough and a paper-based back up is sometimes necessary. Finally the scoring of the questionnaire is also important from the perspective of ease of use and understanding the meaning of the scores. It is necessary to understand why there are so many differences in the way questionnaires are scored. The most basic issue is whether a higher score represents a better outcome or not. Typically when measuring pain we think that more pain equates to a higher score such as with the VAS pain scale [50]. We have created patient-reported quality-of-life questionnaires where a higher score equates to a better quality of life [19, 22, 25]. The Oxford Shoulder Score was originally described with a lower score representing a better outcome, and more recently the authors have suggested the opposite [57, 58]. So there seems to be some consistency in representing better outcomes with a higher score. The next issue is how the actual questions are scaled. The SST uses a yes/no response, which is simple but does not lend itself to the use of parametric statistical analysis. In fact, it is statistically incorrect to use parametric analyses for questionnaires that use a nominal (yes/no) scale or an ordinal scale format such as 0–4 or a Likert scale format even if the overall score is converted to 1 out of a 100 points. Visual analogue response formats, which use a 100 mm horizontal line representing a continuous scale from 0 to 100, can be considered for parametric analyses [19–22, 25, 60]. The SPADI uses a VAS format but then applies a scale with 11 separate categories for each question [54]. The next issue is whether or not the measured score needs to be converted. The DASH takes the sum of the circled responses (i.e., 1–5) subtracts 30 points, and divides by 1.2 to get a score out of 100 where a higher score represents greater disability [36, 37]. Complex scoring and conversions require the surgeon to have assistants who can perform these tasks leading to a practical barrier to use a particular outcome measure.

The ideal questionnaire would be computer or electronically administered with a built-in program to automatically convert the score and provide the clinician with information on the individual questions, domains if applicable, and the overall score. This is what we currently use in our office setting. Patients can use a web-based program or desktop computers in our setting. There are three ways to use electronic outcome assessment. There is commercially available software that is designed for research. These programs can be easily customized to collect outcome data. It is also possible to use commercially available outcome vendors to track your patients using their software and data collection servers. These companies can also store and analyze the information. Finally, it is possible though somewhat costly to develop the software, store the data, and analyze it on your own.

Patient #2 represents a patient presenting with pain and stiffness. After conducting the history and physical examination, the differential diagnosis is between arthritis and a frozen shoulder/adhesive capsulitis. Radiological assessment confirms that the shoulder does not have arthritis. Therefore, a very useful way to assess outcome would combine a visual analogue scale (VAS) pain assessment (i.e., 100 mm line with "0" representing no pain at all and "100" representing pain as bad as it could be) [50, 61], with the physical examination of the shoulder range of motion (ROM) compared to the unaffected shoulder. These two measurements, VAS pain and shoulder ROM, could be easily followed over time to determine the benefits of nonsurgical or arthroscopic intervention. This combination of a self-assessment tool and a "medical metric" [42], as long as the physical examination is done consistently and in a reproducible fashion, would be very simple and effective in measuring outcome. Another option would be to use a validated clinician and patient-based outcome such as the Constant score [14]. The Constant score is comprised of a subjective assessment of pain (0 = severe, 5 = moderate, 10 = mild, and 15 = none) and activities of daily living (on a scale of 0–20 points) [14]. It also comprises a physical examination with assessment of the active shoulder ROM in forward and lateral elevation and external and internal rotation (10 points each) and finally an assessment of power using a spring balance to test the power of shoulder abduction at 90° or at the maximum level of active abduction [14]. This is measured in pound force with 25 lb being considered normal for the maximum 25 points. The Constant score is calculated out of a maximum of 100 points and has more recently been revised to reduce the vagary of the original description [13]. Therefore, *Patient #2* would be well assessed by addressing the four parts of the Constant score. However, the issue with this outcome measure despite its adoption in Europe as the standard shoulder assessment tool is that it is not patient self-reported and the measurement of strength is not reliable [13].

Patient #3 would be a patient presenting with a known rotator cuff tear. Whereas any of the shoulder-specific self-assessment tools would be helpful to evaluate this patient, it would make more sense to use a disease-specific outcome tool. There are two published rotator cuff disease-specific outcome tools, the Western Ontario Rotator Cuff Index (WORC) and the Rotator Cuff Quality-of-Life Questionnaire (RC-QOL) [19, 20]. The WORC has been demonstrated to have all appropriate psychometric properties compared to the RC-QOL [40]. The WORC is comprised of 21 questions and was originally scored out of 2,100 points with a higher score meaning poorer quality of life. The score can be easily transformed to a score out of 100 with a higher score meaning a better outcome. Recently the responsiveness of the WORC has come into

question compared to shoulder-specific outcomes [62]. The RC-QOL is comprised of 34 questions in five domains (16 in symptoms and physical complaints, 4 in sport/recreation, 4 in work-related concerns, 5 in lifestyle issues, and 5 in social and emotional issues) [19]. The RC-QOL has been used in a randomized clinical trial demonstrating that it can be used effectively as an outcome measure with excellent statistical properties [63]. There are significant benefits to using a disease-specific outcome like the RC-QOL with 34 questions. It can be used as a surrogate for the history taking on an individual patient. There are many different questions that are important to the patients that typically would not be asked in a standard history. Also an overall score out of a 100, with separate domain scores generated automatically when the questionnaire is computerized. Finally, the RC-QOL has been used in a predictive situation to determine who will be successful with a nonsurgical treatment protocol [64].

Patient #4 is a patient with shoulder instability. It is clear that this patient population is not well evaluated by the shoulder-specific instruments. In fact, Dawson et al. determined this very early on, in the process of developing the OSS, and stated that:"It became clear that there was a distinct group of patients with a tendency towards recurrent dislocation or subluxation of the shoulder" [57]. They then created the Shoulder Instability Questionnaire (SIQ) [16]. The typical patient with traumatic recurrent anterior instability of the shoulder will have an intermittent problem characterized by minor or severe episodic symptoms and may have to avoid certain activities but typically does not have the same persistence or level of pain compared to other shoulder conditions. The exception to this presentation would be the patient with multidirectional instability [65]. There are several questionnaires designed for this population. Originally the Rowe Score was used to measure the outcome of the Bankart procedure for anterior instability [66]. This was modified by Jobe to include the assessment of pain and to increase the weighting of the functional assessment [67]. However, these outcomes are both clinician and patient-based measures with no described development methodology [40]. The same criticism can be applied to the UCLA score also used in the evaluation of shoulder instability [40]. Romeo compared four scoring systems in the evaluation of 39 patients who had shoulder stabilization (Rowe, Modified Rowe, UCLA, and ASES) [68]. They concluded that there was too much variability in the outcomes and that a better scoring system is necessary [68]. Two shoulder instability disease-specific instruments are currently available: the Western Ontario Shoulder Instability Index (WOSI) and the Shoulder Instability Questionnaire (SIQ) [16, 60]. There has been no head-to-head comparison in shoulder instability patients, of these questionnaires to date. However, if one compares each instrument question to question, then the WOSI covers a greater breadth of items. The WOSI has been shown to distinguish between different types of shoulder instability and specifically patients who need surgery from ones who do not [69]. Therefore, the WOSI would likely be the preferred outcome instrument for the assessment of *Patient #4*.

Patient #5 would be a patient presenting with shoulder arthritis. The arthritic shoulder would have pain and stiffness as significant symptoms, and the shoulder-specific instruments would likely be able to evaluate these patients relatively well. There is only one disease-specific outcome measure, the Western Ontario Osteoarthritis of the Shoulder Index (WOOS) [21]. This 19-item questionnaire has been demonstrated to be highly reliable, responsive, and valid in evaluating patients with osteoarthritis [21]. Therefore, to follow patients over time with osteoarthritis of the shoulder, the WOOS would be the instrument of choice.

Patient #6 could be considered the generic research patient. If a surgeon intends to evaluate the arthroscopic treatment of his or her patients from a research perspective, then the recommendation would be to use a validated shoulder-specific outcome measure alongside a disease-specific outcome measure. The disease-specific measure would be the primary outcome in order to determine the appropriate sample size and the generic outcome to aid in comparisons with other shoulder problems and studies. It is usually the case that disease-specific outcomes are more responsive to change and have a more favorable minimal clinically important difference [40, 70]. This lends itself to the need to recruit fewer patients into randomized clinical trials. Since patient recruitment is the most common barrier to successful randomized trials in surgery, disease-specific outcomes are preferred.

Patient #7 is the athletic patient or laborer. Not only is it necessary to assess their shoulder complaints but also their activity level since this may be critical in order to compare groups of patients with respect to the results of arthroscopic treatment. The Shoulder Activity Scale (SAS) was developed for this purpose [71]. This simple self-reported scale asks the patients to rate themselves at their highest activity in the past year. The maximum score of points is also characterized by two questions regarding the type of sports played, i.e., contact or overhead-type sports [71]. This questionnaire was intended to be a discriminative instrument rather than an evaluative one in contrast to the previously described tools. The SAS should be used alongside an appropriate evaluative tool in order to characterize groups or individual patients into high, average, and low activity levels [71].

Summary

This chapter has been written in a different way compared to other similar chapters. Rather than providing the reader with a list of outcomes and their relative psychometric properties,

the chapter has attempted to start with the patient in mind and then apply the logical and most appropriate outcome measure to that patient. We must continually remind ourselves that there is not one best outcome or only one way to measure a patient's outcome. However, patient self-assessment of outcome is the current standard for reporting research and or evaluating ones practice. The goal of assessing outcome is to determine the truth with respect to how an individual or group of patients has fared with respect to a particular treatment. In the world of shoulder surgery, it was E.A. Codman who was the first to admonish surgeons and hospitals to evaluate their patients to determine the "end result idea" [72]. This is as apropos today as it was then in 1914. What has changed most in terms of outcome assessment is the unquestioned concept that the patients' concerns may not be ours. Any outcome that is used must have its genesis in the patients' concerns. An individual patient will fill in a self-assessment questionnaire if the questions are meaningful to them. The concept that a shorter questionnaire is better is not necessarily true. The best questionnaire is the one that reflects the patients' circumstances better, while demonstrating the properties of reliability, responsiveness, and validity.

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