Sports Injuries to the Shoulder and Elbow

Jin-Young PARK
Editor

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Foreword

Sports are an important aspect of human lives. With our increasing average life expectancy, the number of people who participate in sports is growing rapidly. Consequently, treating sports injuries is emerging as an important concern for professional and amateur athletes.

There are numerous books on sports injuries. This book not only presents shoulder and elbow sports injuries but also outlines their treatment and rehabilitation protocols. There are many specialists who deal with sports injuries; the best experts worldwide have worked together to help professional and amateur athletes to improve their performance and understand the pathophysiology and treatment of the various sports injuries.

This book is not only for physicians and athletes but will also be useful for athletic and physical trainers, physical therapists, sports-related nursing experts, and other associated sports specialists. This book introduces the pathoanatomy (biomechanics), clinical presentation, essential physical examination, disease-specific clinical and arthroscopic pathology, treatment options, and rehabilitation for each injury or disease. Later in the book, rehabilitation methods for each muscle are introduced, which will enable the medical team and sports trainers to help athletes continue to participate in sports after injuries. To simplify the rehabilitation process, videos of each muscle and rehabilitation exercise are provided online in the Springer website.

I want to thank all of the physicians who contributed to this book. Their dedication will enhance the sports activities participated in by athletes and increase the understanding of their injuries by the medical team and trainers. Special thanks go to Dr. Jin-Young PARK for gathering and revising all of the texts.

It is my hope that this book makes an important contribution to sports clinic personnel, professional and amateur athletes, and athletic trainers.

Gilles Walch, MD

Foreword

The diagnosis and treatment of sports injuries of the shoulder and elbow continue to be very challenging. The injuries span a broad age spectrum from adolescence to the aging athlete. The complex biomechanics and pathophysiology of the shoulder girdle and elbow must be understood so that an accurate diagnosis can be made and the proper treatment, conservative or operative, can occur for the patient.

This superb text achieves the goal of providing current and insightful information concerning the etiology, diagnosis and treatment of the more common upper extremity sports injuries. The authors are international experts in sports medicine and share their knowledge to provide a wide spectrum of useful information to deal with these complex problems. This text is essential for trainers, therapists and physicians dealing with shoulder and elbow sports medicine injuries.

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Preface

Athletes should always consult sports medicine experts during rehabilitation and competition. However, players not affiliated with professional teams tend to compete while tolerating pain due to a lack of access to medical facilities or rehabilitation centers. Concerned that athletes need more help from sports medicine, I was inspired by a rehabilitation booklet written by Dr. Lennard Funk of Wrightington Hospital. If surgeons, rehabilitation doctors, physiotherapists, other medical practitioners, and trainers can better understand a disease by reading this book about shoulder, elbow, and sports medicine, I believe that they will return athletes to sports in better condition. Many experts concurred with my thoughts, and this book was written. I thank all of the authors who collaborated in the writing of this book, inspired by the regard in which they hold athletes.

Various factors should be considered when an athlete suffers from pain while performing or in a clinic. Some joint pain originates from the joint itself, but in many cases the joint pain is caused by cumulative effect of problems in other parts of the body. This book considers the many problems that can explain a single disease or injury. For each disease or injury, we gathered various expert opinions to show readers that many sports physicians think differently. Nevertheless, in most diseases and injuries, the treatments are similar and overlap. I am convinced that the readers will obtain better outcomes for the athletes they treat if they apply the overlapping treatments of the many experts introduced here.

Finally, I thank Professor Louis U. Bigliani who taught me about shoulders and still guides me as a shoulder surgeon. I also thank Associate Professor Kyung-Soo Oh, Assistant Professor Suk-Won Chung, Assistant Professor Young-Min Roh, and Jae-Hyung Lee, Deputy Director of the NEON Orthopaedic Clinic, for their dedication in preparing this book.

Seoul, Republic of Korea — Jin-Young PARK, MD, PhD

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1 Biomechanics of the Shoulder: Stability and Kinematics of Shoulder Motion, Throwing Kinematics

Gregory J. Adamson, Timothy J. Jackson, Michelle H. McGarry, and Thay Q. Lee

1.1 Newton's Laws of Motion in Orthopedics

Sir Isaac Newton is the author of a frequently used quote in shoulder surgery: "If I have seen further it is by standing on the shoulders of giants"-Sir Isaac Newton, *letter to Robert Hooke*, 5 February 1676 (Fig. 1.1). Newton's laws are applied to objects which are idealized as single point masses and are excellent for describing structure and function of the musculoskeletal system at the scales and speeds of everyday life. This requires that the size and shape of the object's body are neglected [1]. Therefore, Newton's Laws in orthopedics are predominantly used for free body diagrams of joints or in approximation of dominant force components in the musculoskeletal system. It is also important to note that Newton's laws of motion are not appropriate for use in ultra-small scales, very high speeds, or very strong gravitational fields.

Fig. 1.1 A portrait of Sir Isaac Newton during his time as President of the Royal Society (Ref: www.parrswood.manchester.sch.uk)

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From the Original Latin of Newton's *Principia*

Lex I: *Corpus omneperseverare in statusuoquiescendivelmovendiuniformiter in directum*, *nisi quatenus a viribusimpressiscogiturstatumillummutare*.

Lex II: *Mutationemmotusproportionalemesse vi motriciimpressae*, *et fierisecundumlineamrectam qua vis illaimprimitur*.

Lex III: *Actionicontrariam semper et æqualemessereactionem*: *sivecorporumduorumactiones in se mutuo semper esseæquales et in partescontrariasdirigi*.

Translated to English

Law I: Every body persists in its state of being at rest or of moving uniformly straight forward, except insofar as it is compelled to change its state by force impressed.

Law II: The change of momentum of a body is proportional to the impulse impressed on the body and happens along the straight line on which that impulse is impressed.

Law III: To every action, there is always opposed an equal reaction, or the mutual actions of two bodies upon each other are always equal and directed to contrary parts.

In developing his laws of motion, Sir Isaac Newton credits Galileo Galilei for the first law. Initially, a Greek philosopher Aristotle observed and believed that for the body at rest to move in a straight line at a constant speed, an external force was necessary to maintain the constant speed. Centuries later, Galileo Galilei realized that an external force is necessary to change the velocity of a body, which is defined as acceleration, but no force was required to maintain its velocity. This tendency of objects to resist changes in motion was what Galileo called "inertia." This was then refined by Newton who made it into "Newton's first law," also known as the "law of inertia." Therefore, Newton's first law is a restatement of the "law of inertia" which Galileo described. Although not widely publicized, Newton appropriately gave credit to Galileo for the first law.

For shoulder biomechanics, it is very important to have a good understanding of Newton's laws of motion to understand shoulder function and the intricate biomechanical characteristics of each tissue structure and their complex interplay that occurs to provide stability and function.

1.2 Shoulder Biomechanics

1.2.1 Stability and Kinematics of Shoulder Motion

The shoulder provides the greatest range of motion of any joint in the body, but the trade-off is the propensity for instability. This unique function is essential at the shoulder to enable the hand to be placed in the multitude of positions required in everyday life. This is provided by the intricate and complex coordinated interplay of the active and passive stabilizers of the shoulder. Shoulder motion is comprised of many joints, primarily the glenohumeral and scapulothoracic joints. Abduction in the scapular plane is created by both joints in a movement termed "scapulohumeral rhythm." Shoulder abduction in the scapular plane is described in a 2:1 ratio between glenohumeral and scapulothoracic motion [2, 3], with some variation in the first 30° of abduction (Fig. 1.2) [2, 4, 5]. The sternoclavicular and acromioclavicular joints move at the extremes of motion. Shoulder motion can be broken down into three planes of motion: abduction and adduction in the coronal plane, flexion and extension in the sagittal plane, and rotation about the long axis of the humerus. Arm abduction has an arc of motion of approximately 0–180°, flexion and extension is approximately 180°, and internal and external rotation is approximately 150°. In the glenohumeral joint, as in all diarthrodial joints, six degrees of freedom are present, three translational and three rotational. The three motions that best describe shoulder function are spinning, sliding, and rolling. Spinning occurs when the contact point on the glenoid remains the same while the humeral head contact point is changing. Sliding is pure translation of the humeral head on the articular surface of the glenoid. At the extremes of motion, and certainly in unstable joints, glenohumeral translations occur. In this

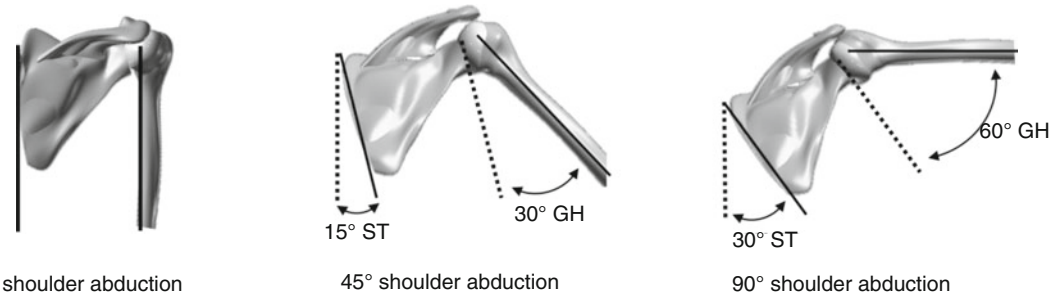

Fig. 1.2 Shoulder motion is comprised of many joints, primarily the glenohumeral (*GH*) and scapulothoracic joints (*ST*). Abduction in the scapular plane is created by both joints in a movement termed "scapulohumeral rhythm"

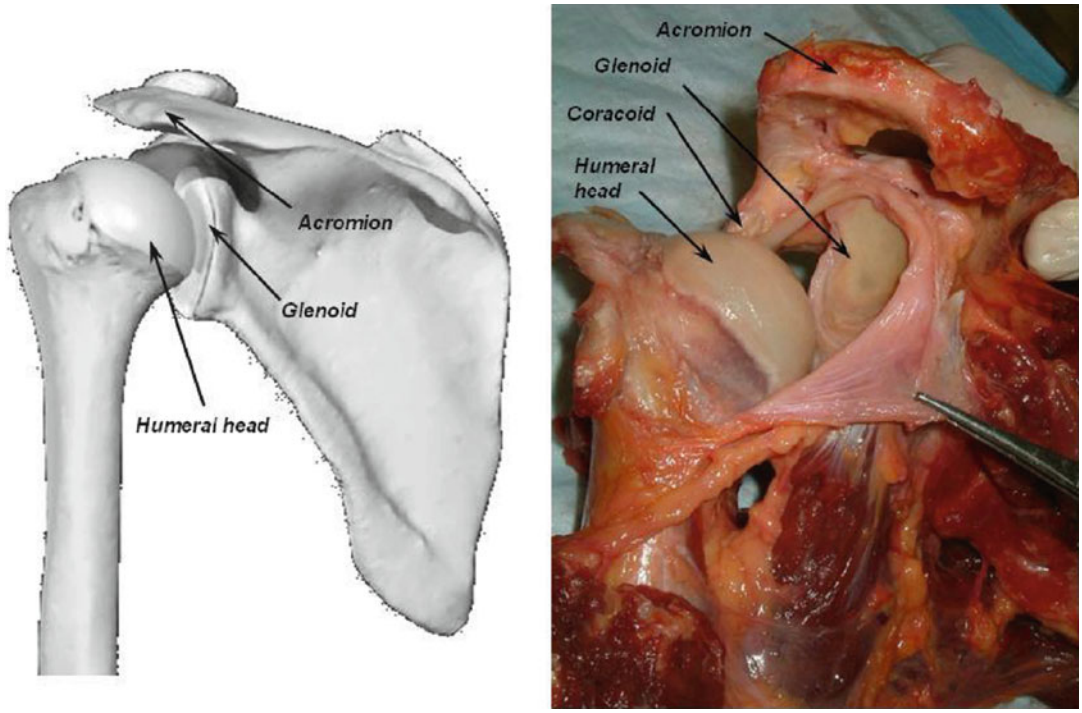

Fig. 1.3 Images showing the bony stability of the glenohumeral joint

circumstance, the contact point on the glenoid is moving, while that for the humerus remains the same. The third type of action, rolling, may also occur at the glenohumeral joint. Rolling is a combination of humeral head translation and spinning with respect to the glenoid, and the contact point changes on both the glenoid and the humeral head [6]. All three rolling motions may take place at the glenohumeral joint about all three orthogonal axes of the glenohumeral joint.

1.2.2 Passive Bony Stabilizers

The anatomic relationship between the humeral head and glenoid can be thought of in a relative ratio of the diameter of each, known as the glenohumeral index (Fig. 1.3). This glenohumeral index is calculated by the maximum diameter of the glenoid divided by the maximum diameter of the humeral head. It is reported to be approximately 0.75 in the sagittal plane and 0.6 in the transverse

plane [7]. Glenohumeral stability is often characterized by the stability ratio, which is the force necessary to dislocate the humeral head from the glenoid divided by the compressive load [8, 9]. This stability ratio is dependent on the depth of the glenoid and increases with greater glenoid depth. The labrum contributes to this by deepening the concavity of the glenoid. It has been shown that this ratio decreases approximately 20 % if the labrum is removed and even further with chondrolabral defects [10]. Stability ratios have been shown to be higher in the superior-inferior versus anterior-posterior plane and with humeral adduction compared to abduction. In this same report, the labrum was noted to contribute only 10 % to the stability [11].

1.2.2.1 Humeral Head

Only 25–30 % of the humeral head is covered by the glenoid at any given anatomic position. Humeral position has been shown to affect contact area between the humeral head and glenoid. With increasing abduction, the contact area as well as the congruity between the glenoid and humeral head improves [12]. This is due to an articular surface mismatch in adduction. Rotation affects contact area with the contact point of the humeral articular surface moving forward and inferior during internal rotation and posterior and inferior in external rotation [7]. With elevation, the contact moves superiorly on the humeral surface [13] and contact area of the glenoid shifts posteriorly. The humeral contact in elevation moves from inferior to superocentral-posterior region with the maximum contact areas occurring at 120° of elevation [14]. Ultimately the stability provided by the humeral head lies in its ability to achieve congruence with the glenoid which is maximized in elevation, a common functional position, and a position that occurs in the act of throwing.

1.2.2.2 Glenoid

In the coronal plane, the glenoid is inclined superiorly with a mean inclination reported to be 4.2° (range −7 to 15.8°). In the sagittal plane, the glenoid is retroverted 1.2 ± 3.5° (range 9.5° anteversion to 10.5° retroversion is measured from the axial plane) [15]. The little stability the glenoid provides comes from the depth, version, and inclination. Glenoid version varies greatly with excessive retroversion causing posterior instability [16]. Shoulder arthroplasty studies have shown that when the glenoid component is retroverted, a common occurrence in osteoarthritis, significant eccentric forces are placed on the glenoid component that could lead to loosening [17]. The exact effect and amount of excessive retroversion or anteversion of the glenoid on native shoulder stability are unclear.

The slight superior tilt of the glenoid articular surface is considered a factor in preventing inferior subluxation of the humerus when combined with the superior capsule and superior glenohumeral ligament [18]. A downward-facing glenoid is related to multidirectional instability. A biomechanical study by Itoi et al. demonstrated the relationship between scapular inclination and inferior stability [19]. In this study, as the scapulae were adducted to create a downward-facing glenoid, the shoulders dislocated inferiorly. As the scapulae were abducted, creating a more inclined glenoid, the humeri reduced. Combined with the superior capsuloligamentous structures, the mechanism of scapular inclination is that of a cam effect determined by the geometry of the glenoid and humerus. This was confirmed in a biomechanical study on posteroinferior glenoplasty where the glenoplasty procedure increased the slope from 0.55 ± 0.07 to 0.83 ± 0.12 and increased the posteroinferior stability ratio from 0.47 ± 0.10 to 0.81 ± 0.17 [20].

1.2.3 Passive Soft Tissue Stabilizers

The passive soft tissue stabilizers include the glenoid labrum and the glenohumeral ligaments (Fig. 1.4). These help limit glenohumeral joint rotation and translation, often in a position-dependent manner. The role of the soft tissue passive stabilizers and particularly the glenohumeral ligaments on shoulder function has been extensively studied. Of the soft tissue stabilizers, the middle glenohumeral ligament and the anterior fibers of the inferior glenohumeral ligament

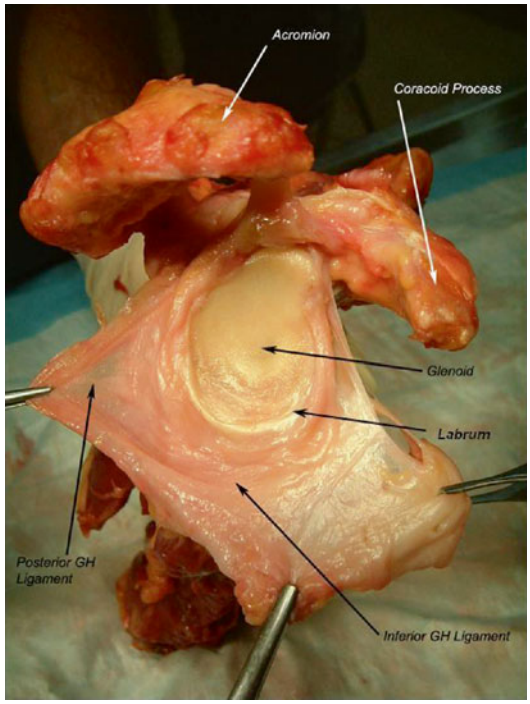

Fig. 1.4 Passive soft tissue stabilizers include the glenoid labrum and the glenohumeral ligaments. The middle glenohumeral ligament is not shown

(IGHL) work together as barriers to dislocation at 45° glenohumeral abduction. The IGHL alone prevents anterior dislocation of the joint at 90° of abduction [21]. For anterior stability, the anterior-superior portion of the IGHL has been shown to be the primary capsular restraint [22]. In this section, we will discuss the anatomy and biomechanics of the soft tissue passive stabilizers.

1.2.3.1 Labrum

The labrum is a triangular rim of fibrocartilaginous tissue that surrounds the glenoid. It deepens the socket by an average of 9 mm in the superior-inferior plane and 5 mm in the anterior-posterior plane. This accounts for up to 50 % of the total glenoid socket depth. Removal of the labrum, as in a Bankart lesion, reduces the depth from 5.0 to 2.4 mm [23]. A cadaveric study in which the labrum was removed but the capsule remained intact demonstrated an increased laxity with labral resection in the adducted position [24]. The stability ratio decreases 20 % with resection of the labrum and further decreases with chondrolabral defects [10].

The superior labrum has a distinct anatomy from the remainder of the labrum, due to the long head of the biceps tendon insertion. The superior labrum functions as a passive stabilizer of the humeral head. In a cadaveric study, surgically created Type II SLAP tears resulted in a significant increase in total range of motion, external rotation, internal rotation, anterior-posterior translation, and inferior translation. After arthroscopic repair, these values were restored [25]. However, the glenohumeral kinematics were not significantly altered by a surgically created Type II SLAP tear [26].

1.2.3.2 Inferior Glenohumeral Ligament Complex (IGHLC)

The anterior IGHL is the most important stabilizer in 90° of abduction and external rotation [21]. Strain measurements confirmed this by showing the anterior band to be tight in abduction and external rotation with the posterior band tight in abduction and internal rotation [27]. The axillary pouch (6 o'clock position) was also found to be an important anterior stabilizer with the arm in abduction and external rotation [28]. Further, the anterior band of the IGHL has been shown to be the primary anterior stabilizer with the arm in abduction in the scapular plane [29]. It is important to note that IGHL plastic deformations exist in shoulders with pathologic recurrent instability. This has been shown to occur in a cyclic overloading study whereby repeated subfailure strain resulted in an overuse injury to the IGHL [30]. However, the permanent stretching in the IGHL has been shown to be quite small (less than 1 mm), suggesting that only a slight plication is necessary to restore capsular anatomy after a primary instability injury [31, 32]. In another cadaveric study, a positive linear correlation between the length of the anterior band of the inferior glenohumeral ligament, external rotation of the humerus, and anterior translation was demonstrated [33]. For anterior instability resulting from capsular laxity, arthroscopic anterior-inferior plication of 10 mm has been found to be effective in reducing anterior

translation and external rotation but altered the glenohumeral center of rotation posteriorly and inferiorly [34, 35].

The labrum-IGHL complex does not have any significant role in the joint reactive forces of the glenohumeral joint. In a cadaveric study, incision and resection of the anteroinferior capsule and labrum did not significantly affect the joint reactive forces demonstrating that the concavity compression effect seen in the shoulder is greatly affected by the dynamic stabilizers [36]. However, the IGHLC is a major contributor to the passive stabilizers of the glenohumeral joint, with its function dependent on arm position. Based on cadaveric studies, arthroscopic techniques have the potential to restore the normal passive stabilizing function of the capsule and labrum but significantly alter the path of glenohumeral joint articulation and potentially initiate subsequent glenohumeral osteoarthritis.

1.2.3.3 Superior and Middle Glenohumeral Ligaments

In conjunction with superior glenoid tilt, the SGHL acts to provide passive resistance to inferior subluxation [18, 37, 38]. Strain in the SGHL is maximal with the arm in adduction and external rotation. The MGHL is a major constraint to anterior humeral displacement and becomes taut in the abducted, externally rotated position [21]. With the arm in external rotation, strain in the MGHL was found to be highest at 0 and 45° of abduction but decreased in 90° of abduction [39]. Sectioning of the MGHL does allow increased excursion of the humeral head but does not typically result in instability, making this a contributor to anterior stability, but not the essential ligament that is damaged in anterior instability [37].

1.2.3.4 Rotator Interval

Shoulders with rotator interval lesions demonstrate inferior instability with the arm in internal rotation [40]. Sectioning of the rotator interval has been shown to create inferior and posterior instability while imbrication provides resistance to inferior and posterior translation [41]. In a more specific study, the coracohumeral ligament (CHL) has been shown to be a restraint to inferior translation but only in the position of external rotation. In internal and neutral rotation, the negative intra-articular pressure provided by an intact rotator interval capsule has been shown to provide superior-inferior stability [42]. Rotator interval closure involves imbrication of the capsule from the supraspinatus to the subscapularis and has been shown to improve translation in all directions for multidirectional instability [43]. More specifically, in 60° of glenohumeral abduction, a medial-lateral rotator interval closure restored range of motion to the intact state and was more effective in reducing posterior translation than a superior-inferior closure [44]. Additional studies have shown that a rotator interval closure reduced translation as well as abduction, flexion, and external rotation [45, 46]. However, when combining rotator interval closure with capsule plication in the treatment of multidirectional instability, a surgeon should evaluate patients individually, so as to avoid over-tightening when performing both of these procedures [47].

1.2.3.5 Coracoacromial Ligament

The coracoacromial (CA) ligament spans from the anterolateral acromion to the coracoid. It consists of an anterolateral band and a posteromedial band with the anterolateral band covering the entire anterior acromial undersurface. This ligament has been shown to act as a humeral head stabilizer and suggests that it plays a role in the static restraint of the glenohumeral joint. It provides a suspensory function and may restrain anterior and inferior translations through an interaction with the coracohumeral ligament [48]. Its function in rotator cuff pathology however is unclear. Cyclic loading of the CA ligament demonstrated a greater drop in peak stress in rotator cuff tear shoulders than in normal shoulders, whereas the stress relaxation response was not different. This was attributed to ultrastructural changes within the ligament as a result of an altered loading environment. However, it remains unknown whether this occurs as a result of a rotator cuff tear or if it contributes to the pathogenesis of cuff disease [49]. Currently there is debate regarding management of the CA ligament in the setting of rotator cuff pathology and further studies are warranted.

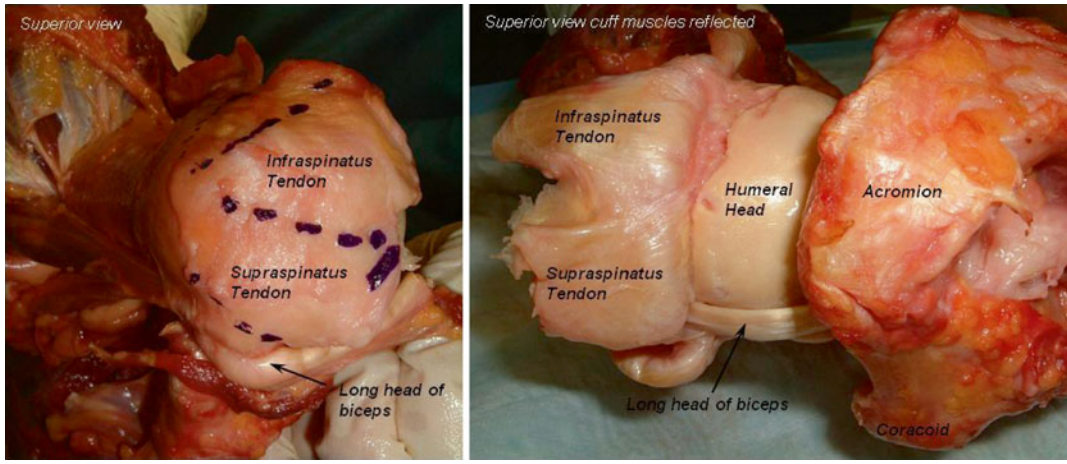

Fig. 1.5 The rotator cuff muscles provide significant stability to the shoulder joint, almost hugging the joint to the glenoid

1.2.4 Active Stabilizers

The active stabilizers of the shoulder are muscle-tendon complexes that provide stability and function to the shoulder. These include the rotator cuff, biceps, deltoid, pectoralis major, and latissimus dorsi. The effect of the shoulder muscles on shoulder stability has been recognized since 1884 [50]. These shoulder muscles are very important to normal shoulder function [51, 52]. These muscles generate a joint compressive force, which in combination with the passive restraints maintain joint stability [8, 51]. Muscle forces are probably most important in the midranges of shoulder motion when the capsule and glenohumeral ligaments are thought to be lax. However, shoulder muscles are also active when the shoulder is abducted and fully externally rotated [53, 54]. This interplay of active and passive stabilizers is critical for glenohumeral joint stability. Strengthening of the shoulder muscles enhances joint stability and function where large forces are generated in the shoulder for movement [55]. In the normal shoulder, this contributes meaningfully to joint stability through the application of a compression force. This is the component of the glenohumeral joint force that acts perpendicular to the glenoid fossa such that the concave humeral head is compressed into the glenoid fossa. Coined "concavity compression," this action was initially reported as being important in maintaining joint stability at the midranges of shoulder elevation when the passive restraints are lax [8]. The shoulder muscles that are active in elevation of the arm include the rotator cuff muscles. In addition to the rotator cuff muscles, any muscle that crosses the glenohumeral joint can contribute to concavity compression [56–60]. When this complex and intricate interplay between the shoulder muscles is altered, the force environment in the shoulder will also be altered, according to functional demands. As a result, the abnormal force environment in the shoulder may also initiate a series of subsequent shoulder pathologies. In this section, we will discuss the stability provided by the active stabilizers and the functions of the rotator cuff and surrounding musculature (Fig. 1.5).

1.2.4.1 Rotator Cuff

The rotator cuff provides stability through concavity compression and barrier effects [61–63]. The contribution of the active stabilizers was thought to contribute more than the passive stabilizers [64]. However, one study has shown an equal contribution to anterior stability with the cuff playing a more important role posteriorly [65]. The subscapularis functions to adduct and internally rotate the humerus. The supraspinatus functions as a shoulder abductor, initiating the first 30° of forward elevation and assisting the deltoid in the first 90° of abduction. The supra-

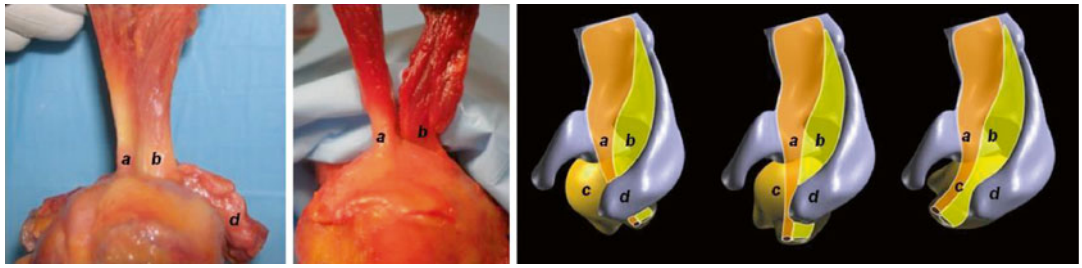

Fig. 1.6 Photographs and schematic drawing demonstrating the supraspinatus subregional footprint orientation at different humeral rotation positions (*a* anterior supraspinatus, *b* posterior supraspinatus, *c* humeral head, *d* acromion) [70]

spinatus is not a single fusiform muscle and tendon, but rather a more complex structure with distinct anterior and posterior subregions [66–69]. The tendon associated with the anterior subregion is thicker and more tubular, while that of the posterior subregion is thinner and flat (Fig. 1.6). The anterior tendon also extends further medially from its insertion on the greater tuberosity and branches into a fibrous framework, while the posterior tendon does not have this fibrous organization. The anterior and posterior subregions also have different muscle physiological cross-section areas (PCSAs) despite having similar tendon cross-sectional areas [67]. The function of the supraspinatus is complex. Besides initiating shoulder abduction and stabilizing the glenohumeral joint, it contributes to humeral rotation. Its role in humeral rotation, however, is dependent on the initial position of the humerus [70]. In the scapular plane, the anterior subregion of the supraspinatus acts as both an internal and external rotator, depending on the initial humeral position, while the posterior subregion acts only as an external rotator. The infraspinatus and teres minor work as humeral external rotators and are most active with the humerus adducted [70].

1.2.4.2 Long Head of Biceps Tendon (LHBT)

The long head of the biceps tendon's function has been difficult to establish, mostly because it spans the glenohumeral and ulnohumeral joints. It has been shown to be a depressor of the humeral head as well as aid in rotational and translational stability [71, 72]. This was demonstrated by sequential loading of the LHBT in a human cadaveric model where significant decreases in external rotation, internal rotation, and total range of motion were observed. Glenohumeral translation in all planes was significantly decreased as well. The humeral head shifted posteriorly with biceps loading at maximum internal rotation, 30° and 60° of external rotation. At maximum external rotation, the biceps loading shifted the humeral head anterior. These findings support the belief that the LHBT may act to center the humeral head on the glenoid during extremes of motion [73]. The biceps has also been shown to be as efficient as the supraspinatus, infraspinatus, and teres minor as a stabilizer, because the biceps becomes more important than the cuff muscles in an unstable shoulder [74]. In the setting of rotator cuff tears, active contraction of the biceps prevents superior migration of the humeral head, almost normalizing the kinematics of the glenohumeral joint [75]. In a cadaveric study, cuff-deficient shoulders had more hypertrophy of the long head of the biceps tendon, while muscle hypertrophy did not occur. This hypertrophy of the tendon indicates a local pathology such as impingement or secondary stabilization, rather than compensatory overuse and hypertrophy of the muscle [76].

1.2.4.3 Deltoid

The deltoid functions primarily as a shoulder abductor and forward flexor. An EMG study showed that the action of the deltoid was highly differentiated in its five different regions with a component of dynamic stability in the scapular plane [77, 78]. Anterior stabilization by the deltoid has been shown in the abducted, externally

rotated position [79]. The large and powerful nature of the deltoid has a strong influence on stability and normal shoulder motion.

1.2.4.4 Force Couples

To understand the combined function of the rotator cuff, one must understand the concept of force couples. A force couple is defined as two forces that act on an object to cause rotation. In order to reach a state of equilibrium, the sum of forces on an object must be equal in magnitude and opposite in direction. Two major force couples act synergistically on the glenohumeral joint: one in the coronal plane and the other in the transverse plane.

The force couple acting in the coronal plane is comprised of the moments produced by the deltoid and inferior rotator cuff (infraspinatus, teres minor, and subscapularis) [2]. The deltoid moment lies above the center of rotation, while the inferior cuff moment acts below the center of rotation, parallel to the lateral border of the scapula. This force couple is important in producing stable glenohumeral abduction. Also important contributors, the pectoralis major and latissimus dorsi are included in this force couple. The depressor moment produced by the inferior rotator cuff may be too weak to counterbalance the strong deltoid moment. However, the pectoralis major and latissimus dorsi have similar depressor moments as the deltoid [80] and therefore are thought to work along with the inferior rotator cuff in the coronal plane force couple [81]. Recent biomechanical studies have replicated the moments produced by the pectoralis major and latissimus dorsi in order to create a more anatomic shoulder construct [80–86].

The transverse force couple is comprised of moments produced by the subscapularis anteriorly and infraspinatus and teres minor posteriorly. Inability to maintain a balanced transverse force couple can lead to anterior or posterior translation of the humeral head. In the setting of a massive rotator cuff tear involving the infraspinatus and teres minor (or posterior moment), the larger moment produced by the subscapularis can lead to anterior translation of the humeral head. This uncoupling between forces leads to an unstable fulcrum for glenohumeral motion.

When the force couples are disrupted, force imbalance occurs in the shoulder affecting the stability and the kinematics of the shoulder. This was demonstrated in a cadaveric study by simulating a weakened subscapularis, as seen in overhand throwers, on glenohumeral joint kinematics and contact pressures [87]. In this study, the authors replicated multiple lines of pull for the rotator cuff, deltoid, pectoralis major, and latissimus dorsi, using all muscles in the transverse and coronal force couples. They concluded that less force on the subscapularis lead to a significant increase in external rotation and posterosuperior glenohumeral contact pressure. This can be attributed to the disrupted transverse force couple leading to an imbalance between anterior and posterior forces as the strong moment arm of the infraspinatus, usually restrained by the subscapularis, now has a relatively stronger moment arm due to the weakened subscapularis moment arm, leading to posterosuperior translation.

1.3 Kinematics of Throwing

The kinematics of throwing is complex with tremendous forces acting on the shoulder. The throwing motion requires a rapid transmission of force from the lower extremity and core to the upper extremity. These immense forces and velocities place the shoulder at great risk of injury, especially in the setting of improper mechanics. An intricate and complex interplay between the scapula, humerus, and scapulohumeral and scapulothoracic muscles is required to maintain glenohumeral stability throughout all phases of throwing. Overhead throwing can be divided into five stages: windup, early cocking, late cocking, acceleration, and deceleration with follow-through (Fig. 1.7).

1.3.1 Windup

Windup begins with the stride foot stepping backward, away from home plate, and the arms

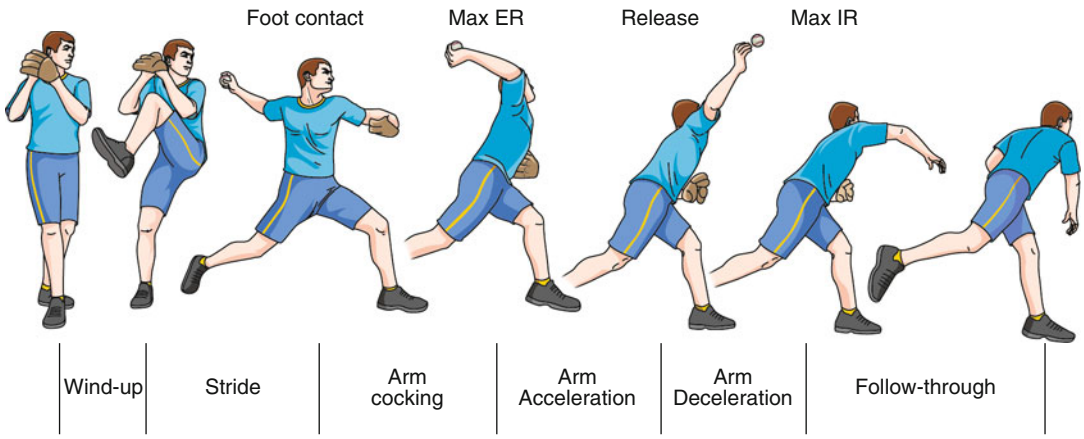

Fig. 1.7 Schematic of the phases of throwing motion from windup to follow-through. *ER* external rotation, *IR* internal rotation (Fleisig et al. [111])

are lifted upward, often overhead. The pivot foot rotates to be parallel on the rubber as weight is transferred to it. This stage ends with the ball leaving the glove hand and the body balanced on the pivot foot. During the windup, EMG activity of the shoulder girdle and upper extremities is low. Instead, there is significant activity in the trunk and lower extremities as energy is stored for transfer to the throwing arm [88]. In the last moments of this phase, stance limb stability is provided by the gluteus medius. This is important for all pitchers, as it provides a stable base to initiate the pitch and minimize anterior-to-posterior sway of the body.

1.3.2 Early Cocking

During early cocking, the hip of the pivot leg slightly flexes in preparation for extension during late cocking and acceleration phase. The gluteus maximus is important in providing this propulsion. The pivot leg propels forward the stride leg, the nondominant upper extremity, and trunk. It is important that the pitcher stride rather than rotate too early. This "opening" of the pelvis and trunk pivots the body instead of propelling forward. While this force transmission and propulsion is occurring in the legs and torso, the trapezius and the serratus anterior muscles form a force couple to upwardly rotate and protract the scapula. This scapula motion is essential to place the glenoid in a stable position for the abducting and rotating humeral head. If the scapula is not positioned correctly, impingement can occur [89]. The deltoid and supraspinatus muscles act in synergy to abduct the humerus. The deltoid provides much of the abduction force with the supraspinatus fine tuning the position of the humeral head in the glenoid [90]. The remainder of the rotator cuff muscles have less activity during this phase, due to the lack of rotational forces applied to the humerus. As the stride foot strikes the ground during late cocking, the biceps becomes mildly active as the elbow is flexed. The hand should be on top of the ball, preventing early external rotation and supination which can decrease velocity.

1.3.3 Late Cocking

Late cocking begins when the stride leg makes contact with the ground with rapid forward motion of the trunk [88]. The nondominant lead shoulder rotates forward and horizontal abduction of this shoulder is minimized by keeping the lead arm closed in front of the body. This optimizes the centripetal forces by keeping more mass close to the center of rotation of the trunk. Abduction of the humerus is maintained and external rotation increases up to 170° [91, 92]. Just before the arm reaches maximum external rotation, an internal rotation torque measuring 67 Nm, shoulder compression force measuring

1,090 N and anterior force measuring 380 N occur. Static and dynamic restraints combine to stabilize against these forces. In this position, the primary static anterior stabilizer of the glenohumeral joint is the anterior band of the inferior glenohumeral ligament complex [21]. While the supraspinatus and the deltoid activity diminish as the humerus ceases to abduct, the subscapularis increases activity to act as a dynamic stabilizer to help center the humeral head [30]. The subscapularis acts as a barrier to anterior translation, together with the pectoralis major and the latissimus dorsi. These muscles act as a dynamic sling to augment the anterior-inferior glenohumeral ligament. The latissimus and teres major act eccentrically as they are internal rotators of the humerus. The infraspinatus and teres minor show increased EMG activity as they act to externally rotate the humerus. Additionally, the posterior rotator cuff functions as a checkrein by preventing excessive anterior subluxation. The scapulothoracic muscles continue to be active to produce a stable platform for the humeral head and to enhance maximal humeral external rotation [89]. The middle portion of the trapezius, the rhomboids, and the levator scapulae are all important in providing this scapular stabilization. The serratus anterior is also important in opposing retraction of the scapula.

1.3.4 Acceleration

The acceleration phase begins when the humerus has reached maximum external rotation and ends with ball release. The angular velocity of the humerus reaches 7,000°/s with internal rotation torque measuring 14,000 in.-lb [91]. This phase lasts only 50 ms [91, 92]. The huge torque and rapid angular velocity during acceleration is transferred from the trunk, with augmentation by the latissimus dorsi and the pectoralis major. The latissimus dorsi has even higher activity than the pectoralis major. These two muscles are important in actively contributing to ball velocity. It has been shown in a clinical study that there is a positive correlation between pitching velocity shoulder adductor peak torque testing [93]. The subscapularis, especially the upper portion, also has very high activity during the acceleration stage and functions with the pectoralis major and latissimus dorsi [53]. Whereas the pectoralis major and the latissimus dorsi are the primary internal rotators of the humerus, the subscapularis functions as a steering muscle to position the humeral head precisely in the glenoid. The teres minor activity is also high, with the muscle acting as a checkrein to anterior instability. During the acceleration phase, the biceps becomes less active and the triceps becomes more active as the elbow extends.

1.3.5 Deceleration and Follow-Through

Follow-through occurs after the ball is released and can be divided into early and late stages. Follow-through consists of eccentric contractions with muscle activity acting to decelerate the upper extremity complex. The deceleration phase has the highest forces of simultaneous muscular firing [89]. It is in this phase that the rotator cuff is acting eccentrically [53, 54]. This is the time when the rotator cuff is at most risk of injury as the kinetic energy not transferred to the ball must be absorbed by the decelerating arm and body. Initially, the trunk and dominant lower extremity rotate forward, while the shoulder continues to adduct and internally rotate to 30°. Deceleration is estimated to be 500,000°/s^2 at the shoulder, with an external rotation torque of approximately 15,000 in.-lb at the humerus [88]. The trapezius, serratus anterior, and rhomboids all demonstrate high or very high activity. The deltoid is active, especially the posterior and middle portions, which are positioned to oppose the motion of the upper extremity. The teres minor has the highest activity of all the glenohumeral muscles, continuing to provide a posterior stabilizing checkrein. Injury to the posterior glenohumeral joint stabilizers will commonly become apparent during this stage. Late follow-through is a noncritical stage, with all of the shoulder muscles exhibiting decreasing activity [89].

1.4 Thrower's Shoulder Adaptations

The rotational arc in the thrower's shoulder is shifted in the direction of external rotation, secondary to developmental humeral retroversion and soft tissue adaptation (Fig. 1.8).

1.4.1 Soft Tissue Adaptations

There is a developmental shift towards greater external rotation in overhead athletes. This accommodates their throwing arc. Along with this increase in external rotation, commonly there is also a decrease in internal rotation that occurs. It has been suggested that these changes occur by a posteroinferior contracture, causing decreased internal rotation, and, when combined with anterior capsule laxity in throwers, cause labral and rotator cuff pathology. This was tested in a cadaver model to study the effects of such posterior contracture and anterior laxity on glenohumeral translation and motion (Fig. 1.9) [94].Ten fresh frozen human cadaver shoulders were tested before and after stretching the anterior capsule by 30 % beyond the normal range of external rotation and a simulated posterior capsule contracture which was simulated by performing a 1 cm posterior capsule plication. For biomechanical testing, glenohumeral positions and translations were measured with 15 and 20 N of loads applied in anterior, posterior, superior, and inferior directions at 90° of external rotation. Range of motion in external and internal rotation was also measured. External rotation of the joints increased significantly (18.2°, $p<0.001$) after stretching 30 % beyond the normal external rotation and internal rotation decreased after posterior capsule plication (8.8°,

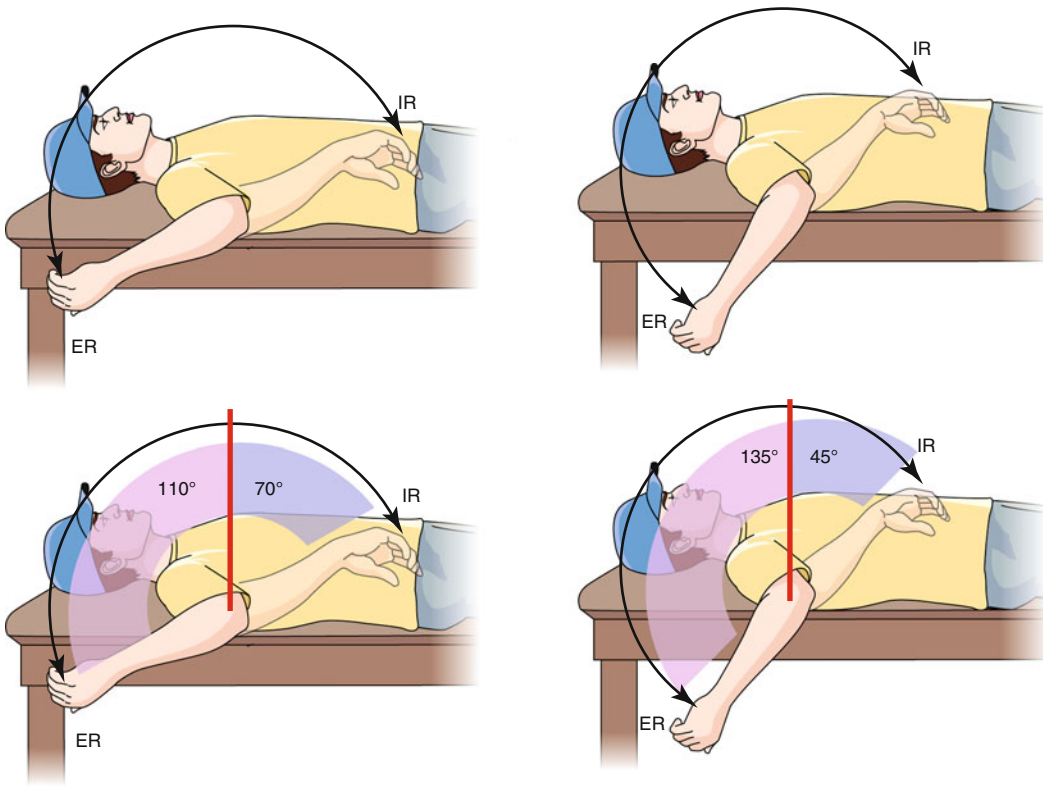

Fig. 1.8 Schematic showing the adaptation of the rotational arc in a thrower's shoulder. *ER* external rotation, *IR* internal rotation (The thrower's ref: Limpisvasti et al. [112])

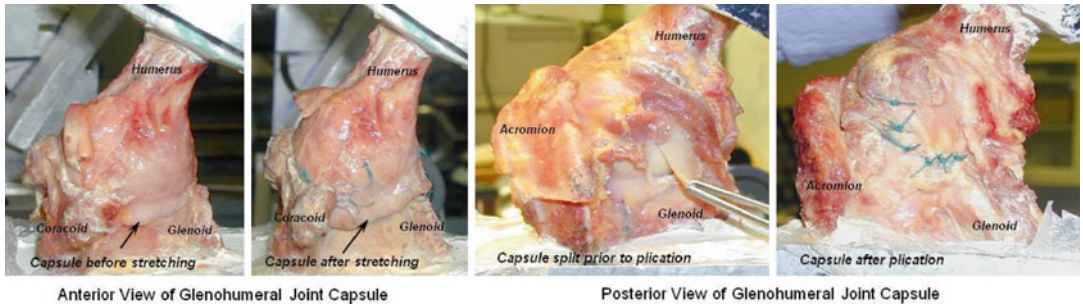

Fig. 1.9 Photographs showing cadaveric glenohumeral joint with simulated anterior laxity and the simulated posterior capsular contracture to create a thrower's shoulder model. Rotator cuff muscles have been removed during dissection

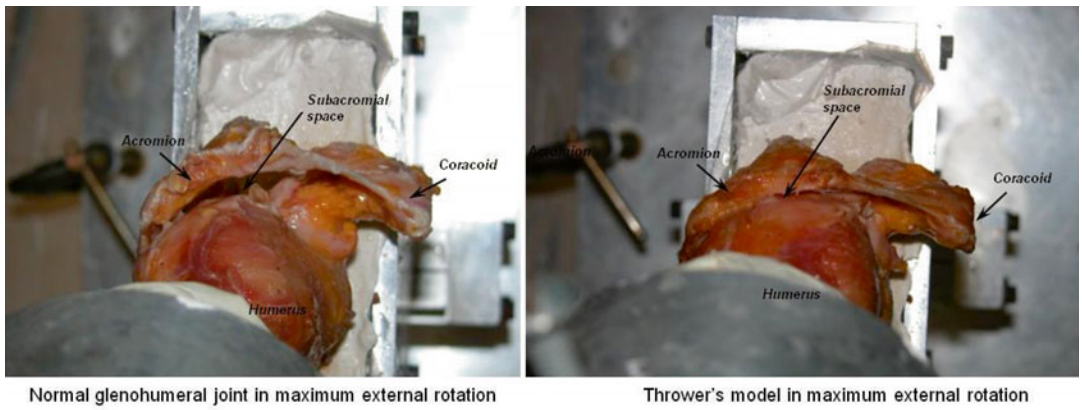

Fig. 1.10 Photographs showing superior shift of the humeral head and decrease in subacromial space with simulated anterior laxity and the simulated posterior capsular contracture at maximum external rotation of the humerus

$p = 0.005$). With the humerus in maximum external rotation, there was a normal posteroinferior shift in the humeral head in the normal capsule condition, but with capsule contracture, there was a superior shift in the position of the humeral head (1.95 mm, $p = 0.013$) (Figs. 1.10 and 1.11). These authors concluded that capsular changes simulating those in throwing athletes do not allow the humerus to externally rotate into its proper posteroinferior position in the late cocking phase of throwing. Instead the humeral head assumes a posterosuperior position, possibly accounting for the etiology of Type II SLAP lesions and rotator cuff dysfunction seen in throwing athletes. Similar results were reported where "GIRD" (glenohumeral internal rotation deficiency) of as low as 5 % resulted in abnormal superior translation, which worsened at even higher percentages [95].

1.4.2 Humeral Adaptations in Throwers

The increased external rotation and decreased internal rotation seen in throwers have been attributed to humeral retroversion differences as well as soft tissue changes. In a study of collegiate baseball pitchers, the retroversion of 19 male college baseball pitchers was evaluated by CT scan and range of motion measurements of passive glenohumeral external rotation at 0° and 90° of abduction and internal rotation at 90° of abduction were performed [96]. The dominant shoulder had significantly greater retroversion of the humerus compared with nondominant shoulders. A significant difference was found between dominant and nondominant external rotation at 0° and 90° of abduction and internal rotation at 90° of abduction. There was a significant correlation

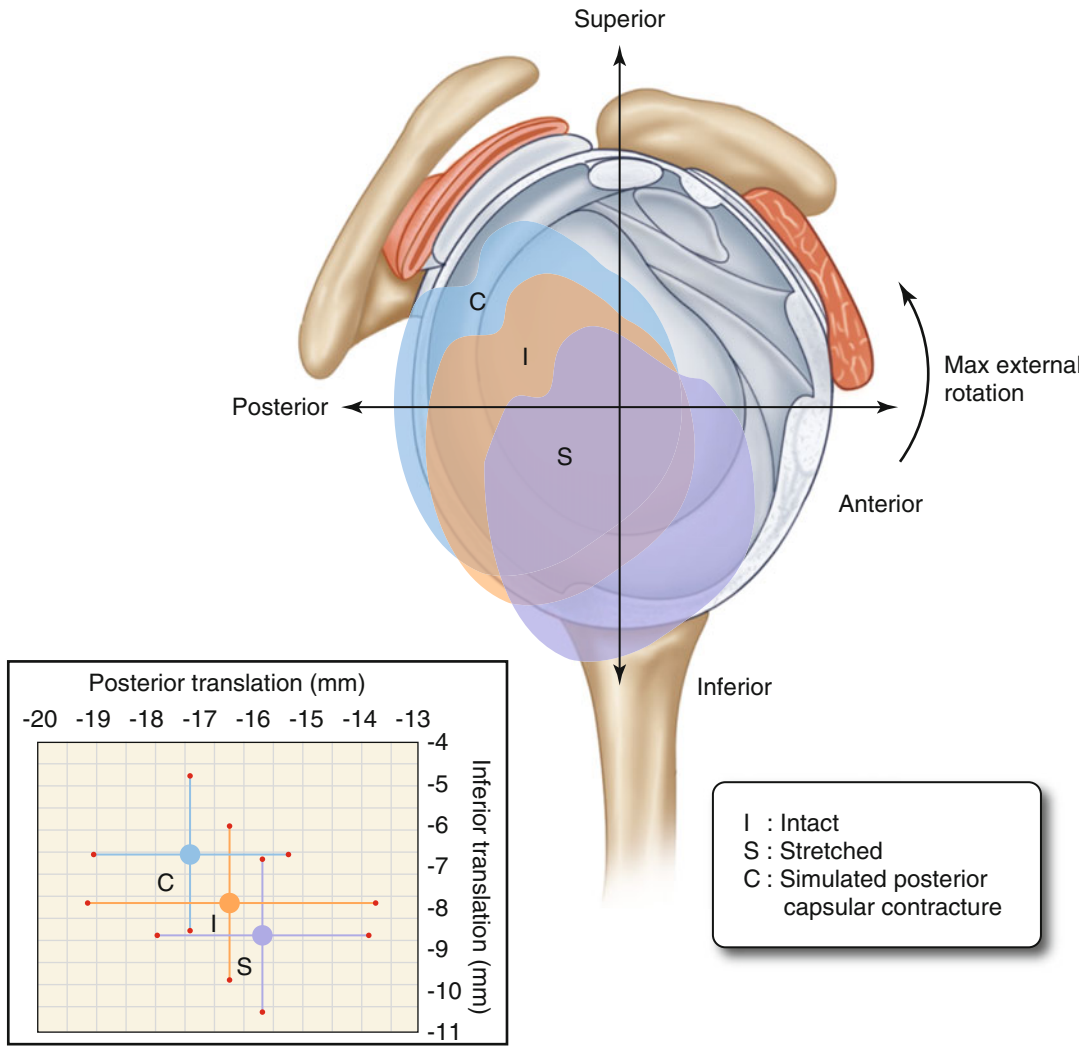

Fig. 1.11 Schematic and graphical representations of the humeral shift from neutral to maximum external rotation, showing the superior shift of the humeral head following simulated anterior laxity and the simulated posterior capsular contracture [94]

between retroversion of the humerus and external rotation at 0° and 90° of abduction in the dominant arm. These authors noted a significant correlation between the side-to-side differences in retroversion of the humerus compared with the side-to-side difference in external rotation at 90° of abduction leading the authors to conclude that rotational changes in the throwing shoulder are due to bony as well as soft tissue adaptations.

This is thought to occur from a slower retroversion derotation during development rather than an active increase in retroversion. In an ultrasonography study, rotation angle of the proximal humerus relative to the elbow (bicipital-forearm angle) was measured to determine the relationship between humeral retroversion and growth in dominant and nondominant shoulders in elementary and junior high school baseball players [97]. The bicipital-forearm angle was significantly smaller in dominant shoulders than in nondominant shoulders, indicating the retroversion angle was greater in dominant shoulders than in nondominant shoulders. There was a moderately positive correlation between age and the bicipital-forearm angle in both dominant and nondominant shoulders, demonstrating that the humeral retroversion angle decreases with age and the decrease is smaller in dominant shoul-

ders. These authors concluded that repetitive throwing motion during development does not increase the retroversion of the humeral head but rather restricts the physiologic derotation process of the humeral head during growth.

1.5 Patterns of Shoulder Injury

1.5.1 Pathomechanics of the Overhead Athlete

Shoulder impingement syndrome, which is the most common shoulder pathology in throwers, is likely initiated by shoulder muscle imbalance, from relative weakness of select shoulder muscles [98]. The impingement syndrome was originally described as compromise of the space between the humeral head and the coracoacromial arch [99]. In the classic case, the coracoacromial ligament and the anterior-inferior aspect of the acromion are compressed against the bursal side of the RC during forward flexion of the shoulder. Importantly, bursitis is often associated with other shoulder problems. In those under 35 years of age, impingement syndrome is almost exclusively associated with anterior glenohumeral instability [100].

Internal impingement was confirmed in a cadaver study simulating a posterior capsule contracture and measuring humeral head shift, maximum humeral rotation angle, and glenohumeral contact pressure and contact pressure in the area of the posterior/superior cuff and labrum [101]. These authors concluded that in the setting of posterior capsule contraction, significant internal impingement occurs in the position of maximum external rotation. This leads to posterior rotator cuff tendon dysfunction and SLAP tears from internal impingement.

1.5.2 The Role of Arm Position

The effects of arm position on internal impingement are very important [102]. Internal impingement was assessed by mapping the location of the supraspinatus and infraspinatus articular insertions relative to the glenoid and measuring the contact pressure in the posterosuperior quadrant of the glenoid. These authors concluded that horizontal abduction beyond the coronal plane increased the amount of overlap and contact pressure between the supraspinatus and infraspinatus tendons and glenoid. This suggests that the mechanics of throwing, specifically excessive humeral horizontal abduction, can lead to internal impingement, causing SLAP lesions and rotator cuff tears.

Scapula position, and in turn glenoid position, also has a very strong influence on shoulder stability [103]. In this study, the authors reported that with increasing amounts of scapula protraction, there was increasing strain in the anterior band of the IGHL. These results suggest that repetitive protraction of the scapula may result in excessive strain and insufficiency in the anterior band of the inferior glenohumeral ligament. The implications of this are important when considering rehabilitation for anterior instability associated with overhead throwing. Scapula position and its relationship to internal impingement were determined in a cadaveric study quantifying the glenohumeral contact pressure and the area of impingement [104]. These authors reported that with increasing internal scapular rotation, the glenohumeral contact pressure and internal impingement increased. The authors concluded that in a simulated throwing motion, increasing internal scapular rotation and decreasing upward scapular rotation significantly increase the area of impingement of the rotator cuff tendon between the greater tuberosity and glenoid. The implications of this are important in preventing and treating internal impingement in overhead throwers.

1.6 Common Pathology in the Overhead Athlete

1.6.1 SLAP lesions

Lesions of the biceps origin and the superior glenoid labrum are termed SLAP (superior labral anterior to posterior) lesions and may occur as a result of superior subluxation of the humeral head [105]. These authors reported five types of SLAP lesions: Type I lesion is simple degenerative fraying of the superior labrum, and the peripheral

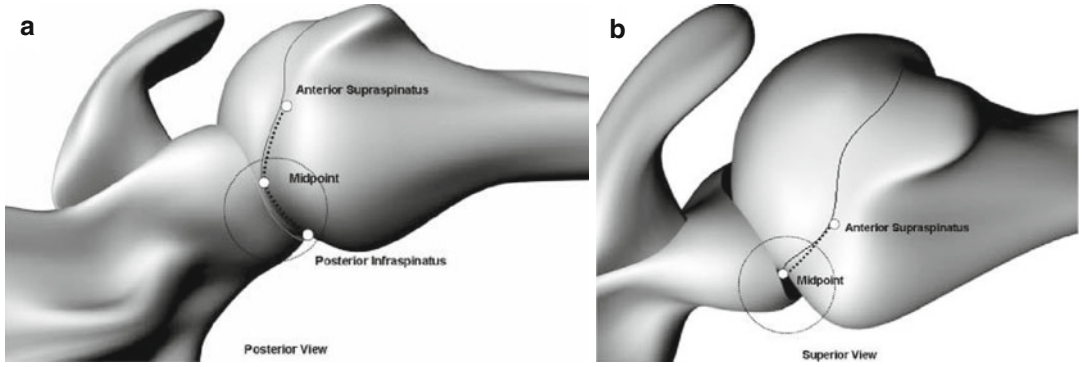

Fig. 1.12 Schematics representing the overlap of the articular insertion points and the potential for internal impingement, located within *circle*. (**a**) Posterior view. (**b**) Superior view. The posterior infraspinatus is not visible in this view [102]

edge of the labrum remains firmly attached to the glenoid as well as the biceps tendon attachment on the supraglenoid tubercle. Type II lesion is a tear of the superior labrum where the labrum and the biceps tendon attachment is avulsed from the glenoid; this is the most common type. Type III lesion is a bucket-handle tear of the labrum with preservation of the biceps anchor. Type IV lesion occurs when the biceps tendon is split with a portion remaining attached to the supraglenoid tubercle; this is similar to the Type III lesion except that a portion of the biceps tendon attachment is also involved. Type V lesion is any combination of these. As we have discussed previously, the long head of the biceps tendon has a role in preventing superior translation of the humeral head, with the humerus loaded in the anterior to posterior direction [106] and the superior to inferior direction [107]. It has been shown that both the long head and the short head of the biceps contribute to anterior stability, especially when there is joint instability from a Bankart lesion [52].

The superior labrum was shown to have significant strain in the throwing motion. Pradhan et al. found that strain was greatest at the posterosuperior portion of the labrum when the arm was in abduction and external rotation [108]. In the setting of SLAP tears and anterior instability in the thrower, the repair of the SLAP tear alone did not restore anterior translation and that anterior capsule laxity that is seen in throwers may continue alter translation despite SLAP repair.

1.6.2 Anterior Instability and GIRD

The soft tissue adaptations seen in throwers were first replicated in a cadaveric study [94]. In this study, the anterior laxity and increased external rotation of the humerus were created by nondestructive stretching of the anterior capsule [33], and posterior capsule contracture was simulated by capsular plication. This cadaveric model mimicked the externally shifted rotation arc seen in throwers resulting from increased external rotation and decreased internal rotation. This anterior instability contributes to the pathology found in the shoulder in relation to internal impingement.

1.6.3 Internal Glenoid Impingement

Internal glenoid impingement is abnormal abutment of the rotator cuff against the glenoid (Fig. 1.12). This occurs in the setting of altered biomechanics seen with GIRD, with anterior laxity and posterior capsule contracture. Throwers with internal impingement complain of posterosuperior shoulder pain that is associated with throwing. This pain usually resolves with rest but returns when throwing resumes. It has been suggested that a subtle increase in anterior glenohumeral translation can cause internal impingement [109]. A cadaveric model of a thrower's shoulder demonstrated that the humerus assumes a posterosuperior position in late cocking phase, possibly

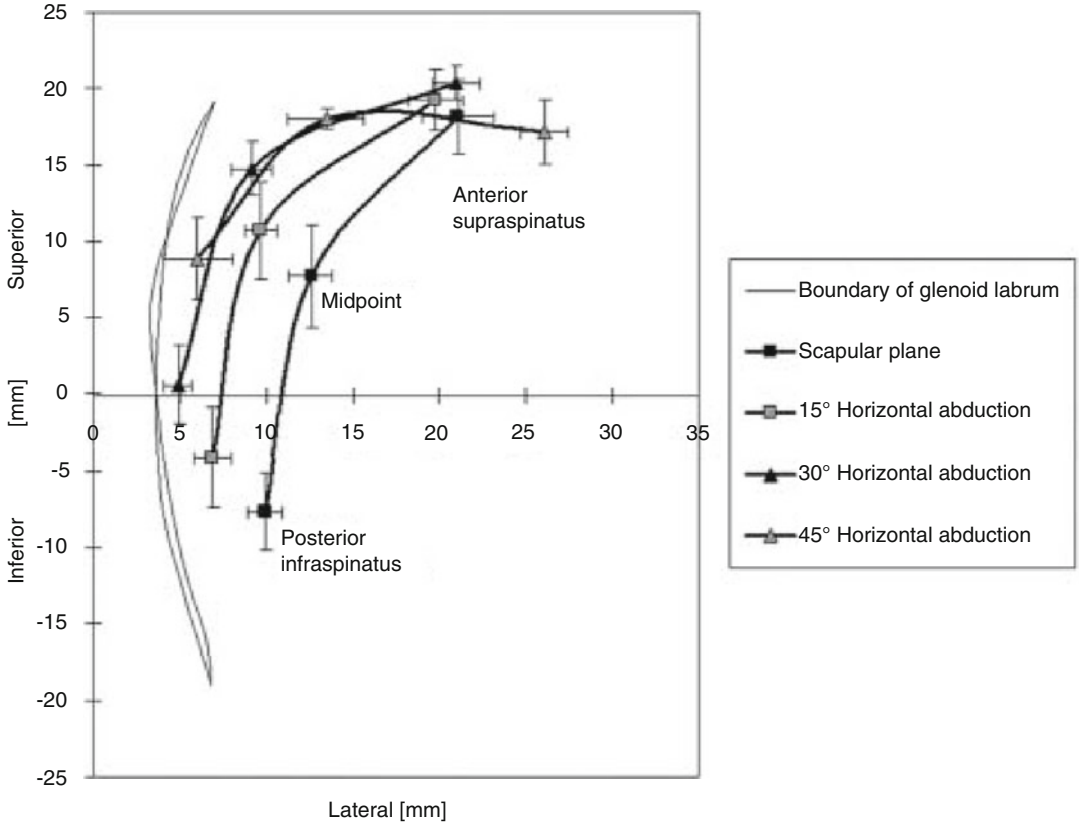

Fig. 1.13 The medial-lateral position of the rotator cuff insertion points relative to the geometric center of the glenoid for each horizontal abduction position [102]

accounting for the etiology of Type II SLAP lesions and rotator cuff dysfunction in throwing athletes [94]. In addition, it has been demonstrated that with excessive humeral horizontal abduction, the rotator cuff can be impinged against the posterosuperior glenoid rim (Fig. 1.13) [102].

1.6.4 Rotator Cuff Injuries

Injury to the rotator cuff has a high prevalence in overhead athletes with the supraspinatus being the most commonly injured of the rotator cuff tendons. This is often in the setting of GIRD due to the pathomechanics involved causing internal impingement and compression of the rotator cuff against the glenoid. Recently, the biomechanical effects of anterior laxity and supraspinatus tears were investigated [110]. These authors concluded that with both lesions, there was a significantly increased total rotational range of motion and significantly decreased force required for dislocation compared with intact shoulders. Bankart repair combined with supraspinatus repair restored range of motion and the force required for dislocation; however, it shifted the humeral head posteriorly at the midrange of rotation.

1.7 Summary

In summary, studies demonstrate the pathomechanics of GIRD and how these altered mechanics may cause pathology in the shoulder. Normally, both static and dynamic stabilizers act to prevent anterior instability, but in the abducted, externally rotated position of throwing, the distance from the rotator cuff to the posterosuperior rim of the

glenoid is small and little tolerance to altered mechanics exists. During the throwing motion, an increased humeral abduction may occur in the late cocking and acceleration stage, resulting in internal impingement. Increased anterior translation combined with posterior tightness then aggravates the internal impingement with more rotator cuff abutment against the glenoid rim.

When conservative measures fail, arthroscopic evaluation of the glenohumeral joint reveals the characteristic lesions of fraying of the supraspinatus tendon in a location slightly more posterior to that seen with classic impingement. Fraying of the posterosuperior glenoid rim is also seen. During arthroscopy, with the scope in the posterior portal, a dynamic exam can be performed with the arm in maximum abduction and external rotation. The internal impingement can be seen as the rotator cuff abuts the posterosuperior glenoid rim.

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Epidemiology of Shoulder Injuries in Overhead Athletes

2

Michelle A. Noreski and Steven B. Cohen

2.1 Introduction

Shoulder injuries, including both acute and chronic, are a frequent occurrence among overhead athletes [2]. Baseball pitchers, football quarterbacks, swimmers, volleyball players, and tennis players are included in this distinct group of athletes who consistently place an exquisite amount of stress on their shoulders. It is important to note that the overhead throw may produce a velocity greater than 7,000°/s and is the fastest movement recorded in humans [2, 3]. Because of this, the throwing athlete can be quite a challenging patient in sports medicine. To effectively perform a throw, there must be a delicate balance between shoulder laxity to accomplish extreme ranges of motion, so that velocity can be generated to the hand or racket and ultimately to the ball, and adequate stability to prevent subluxation or instability [8]. This is defined as "the thrower's paradox." Inherent stress is put on the stabilizing structures including the glenohumeral joint and the scapula and also soft tissue stabilizers such as the rotator cuff, scapulothoracic musculature, fibrocartilaginous labrum, and the joint capsule [2]. Awareness of the forces to which these structures are subjected to in the throwing motion is essential to the diagnosis and treatment of injuries [8]. It is also crucial for the overhead athlete to participate in a preventative stretching and strengthening program during the season and in the off-season to maximize performance and avoid injuries [7].

Unfortunately, not all shoulder injuries can be avoided and may occur due to muscle fatigue or weakness, poor mechanics and conditioning, and altered stability [4–6]. Also, although many shoulder injuries in the elite overhead athlete are common and predictable, there still may be some controversies as to the precise mechanisms by which these injuries occur [1]. In this chapter, we will review the biomechanics of a throw and the adaptive changes that occur in the dominant throwing shoulder. We will also discuss the evaluation and treatment of the many common pathologies observed in the shoulders of overhead athletes.

2.2 Shoulder Biomechanics and Pathoanatomy

2.2.1 Phases/Mechanics of a Throw

Overhead athletes most frequently subject their shoulders to the unstable position of maximal abduction and external rotation during a throw. It is important to obtain a thorough understanding

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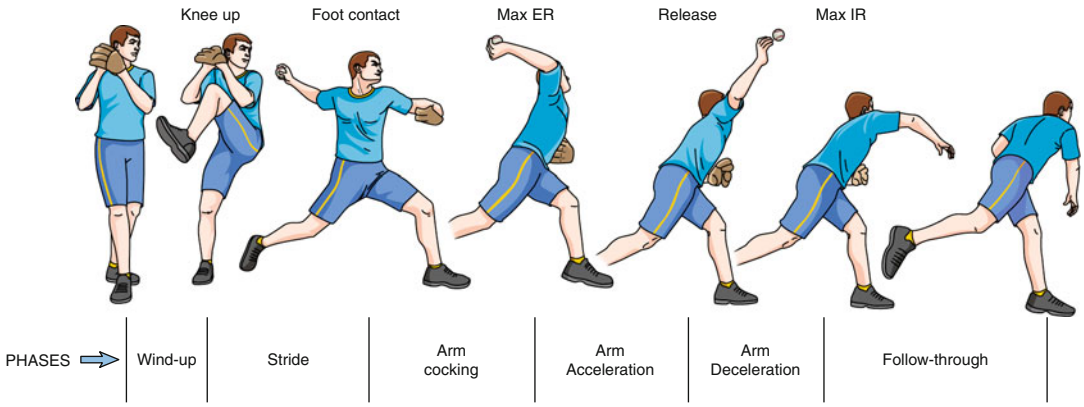

Fig. 2.1 Elbow medial collateral ligament injuries (Ra'Kerry K. Rahman1, William N. Levine1 and Christopher S. Ahmad. Department of Orthopaedic Surgery, Center for Shoulder, Elbow and Sports Medicine, Columbia University Medical Center, 622 West 168th Street, PH 11th Floor, New York, NY 10032, USA)

of the biomechanical stresses placed on specific shoulder structures as they correlate to distinct phases of the throwing motion [8]. It is also necessary to take into account that the legs and trunk (core) play a significant role as force generators in the transfer of energy to the ball. The scapula aids in allowing for extreme ranges of motion by providing a stable platform for the humeral head to rotate. This is part of the "kinetic chain concept." There are six phases included in the description of a throw, which are achieved in approximately 2 s, and have been described in Chap. 1 but are reviewed briefly in this chapter (Fig. 2.1).

Phase I is the *wind-up* or *readying phase*, in which there is the least amount of stress placed on the shoulder. Toward the end of this phase, the dominant shoulder is in slight internal rotation and abduction.

Phase II is the *early cocking phase*, which involves recruitment of the deltoid muscle early on and the supraspinatus, infraspinatus, and teres minor muscles later. The shoulder is moved into 90° of abduction with the elbow positioned slightly posterior to the plane of the body.

Phase III is the *late cocking phase* and involves planting of the lead striding leg with the shoulder culminating in its maximal external rotation to approximately 170–180°. The scapula retracts and aids in stabilization of the humeral head. Shoulder abduction remains at 90–100° and combined with the maximum external rotation, the humeral head, then translates anteriorly on the glenoid. The deltoid muscle firing decreases, and recruitment of the supraspinatus, infraspinatus, and teres minor muscles reaches its peak. Later in this phase, the subscapularis is fired as the torso rotates forward. The biceps, pectoralis major, latissimus dorsi, and serratus anterior muscles begin to fire at the end of this phase allowing maximum horizontal adduction.

Phase IV, *acceleration*, involves internal rotation of the shoulder 90° to the ball release point. In this phase, the scapula protracts and continues to provide a stable base for the humeral head as the body begins to move forward. This allows for a conversion of eccentric to concentric muscle function anteriorly and concentric to eccentric posteriorly. The triceps muscle is recruited early on in this phase, followed by the pectoralis major, latissimus dorsi, and serratus anterior later.

Phase V is known as *deceleration* and is the most violent phase of the throwing cycle due to the dissipation of the remaining kinetic energy to the ball. It is a reversal of the first three phases. Ball release occurs and the humeral rotation returns to 0°. Shoulder abduction is at 100° and horizontal adduction increases to 35°. Eccentric contraction of all of the above muscle groups occurs in order to slow down arm rotation.

Phase VI is last and is known as the *follow-through*. During this phase, the body moves forward along with the arm until motion stops.

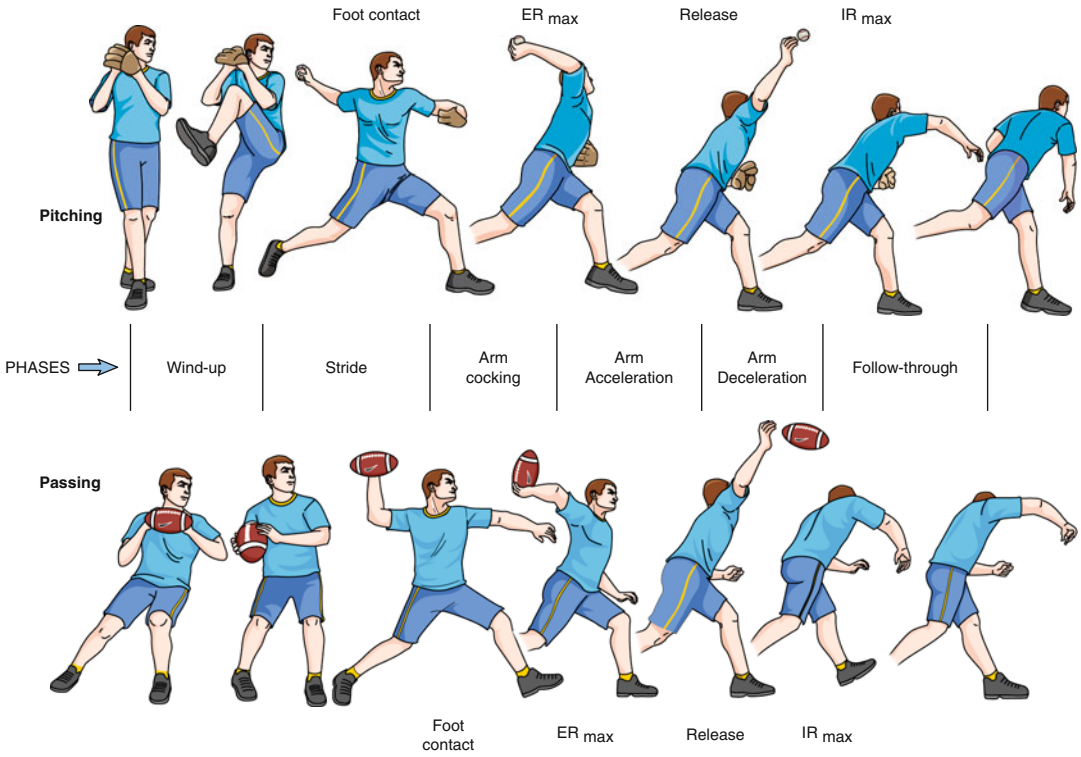

Fig. 2.2 Throwing Phases for baseball and football. From Meister [3]

Muscle firing ceases to resting levels and the loads on the joint decrease.

The phases of a throw described above are more descriptive of a baseball pitch. Although similar, there are some subtle differences when comparing the pitch with a throw in different sports. In football, the increased weight of the ball provides changes in mechanics (Fig. 2.2). It has been shown that quarterbacks rotate the shoulder sooner and assume maximal external rotation of the shoulder earlier on in the throw. This allows for more time in acceleration during internal rotation. Also, the shoulder horizontal adduction is increased and greater elbow flexion is achieved in the late cocking phase. This decreases the potential load on the shoulder. Arm velocity is decreased in the football throw because the quarterback must have a more erect position and a complete follow-through that is observed in the baseball pitch does not occur in football. Due to the decreased force generated when throwing the ball, shoulder injuries are not as common.

2.2.2 Adaptations of the Dominant Throwing Shoulder

Due to the repetitive motion of the dominant upper extremity and the extreme stresses of the throw, adaptive changes occur that affect the stabilizing structures. In overhead athletes, the ability to externally rotate the humerus is very important to generate high velocities to the ball. This leads to an increase in humeral retroversion and a relative capsular laxity. It has been shown in many high-level throwing athletes that the dominant shoulder exhibits increased external rotation and decreased internal rotation in abduction. These changes occur due to laxity within the anterior inferior glenohumeral ligament (AIGHL), which normally functions as a restraint to anterior translation of the arm in abduction and external rotation. The coracohumeral ligament (CHL) is also a restraint to external rotation at the side and may develop laxity, especially in baseball pitchers, and as a result, an increased sulcus sign may be seen. Retroversion of the humeral

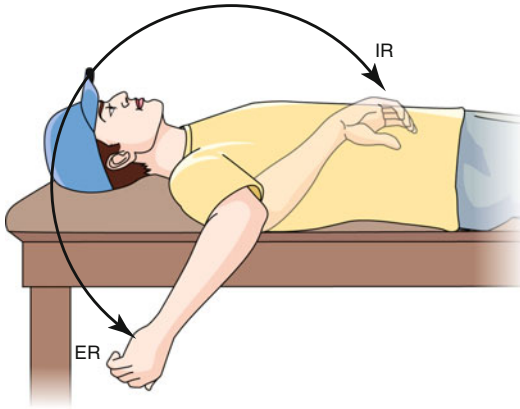

Fig. 2.3 Total arc of rotation. http://www.orlandparkorthopedics.com/patient-education/glenohumeral-internal-rotation-deficit

head (an average of 10–20°) is also a finding in overhead athletes that may lead to increased external rotation of the dominant shoulder. The increase in external rotation can range from 9° to 16°. However, there are instances when the increase in external rotation does not make up for the loss of internal rotation. This typically occurs later in the athlete's career and attributes to a loss of overall rotational motion of the dominant shoulder (Fig. 2.3). This is the general process and evolution of glenohumeral internal rotation deficit (GIRD).

Muscular changes are also seen in the dominant arm of throwing athletes. Hypertrophy of the shoulder girdle muscles is common, while atrophy of the infraspinatus can be seen as well. External rotation strength, exhibited by the infraspinatus and teres minor muscles, has been found to be weaker in the dominant as compared to the non-dominant shoulder. Conversely, muscles used in internal rotation, such as the subscapularis, latissimus dorsi, pectoralis major, and teres major, are typically stronger in the dominant throwing arm.

2.3 Clinical Presentation and Essential P/E

2.3.1 History

A very detailed history must be obtained for accurate diagnosis and treatment of shoulder injuries. Obtaining the mechanism of injury, duration, location, and timing of symptoms will give clues to the diagnosis. The age of the patient will also aid in possible differential diagnoses. For instance, young athletes will frequently have physeal injuries, labral pathology, or instability. On the other hand, older patients will more likely have pathology of the rotator cuff. Overhead athletes in the middle of their career may acquire laxity and rotator cuff pathology.

It is also important to inquire about specific history unique to overhead athletes, such as the phase of the throwing cycle that symptoms usually occur. Pain that occurs during the cocking phase may suggest labral pathology, internal impingement, or instability. During the late cocking or acceleration phases, anterior instability may be seen. In regard to posterior labral pathology or instability, this typically occurs with pain at the follow-through phase. Pain during deceleration or ball release may point toward a diagnosis of rotator cuff pathology. Timing of symptoms during the game, complaints of loss of throwing velocity, lack of command while throwing, and recent decrease in the number of pitch counts during games can also provide clues as to the possible diagnosis. It is also important to ask at what age the athlete began throwing and amount of rest in between seasons. Symptoms pertaining to the experience of numbness, tingling, or discoloration should raise the possible concern of neurologic or vascular pathology.

2.3.2 Physical Examination

2.3.2.1 Observation

Examination of the shoulder should begin with observation of positioning and symmetry, particularly of the scapula, as well as for any obvious gross deformities. Overdevelopment of the musculature of the dominant shoulder is common as discussed previously in this chapter. Atrophy of the supraspinatus and/or infraspinatus within their respective fossa, although usually subtle, may also be detected with inspection. It is also important to assess scapular positioning,

which is usually in a protracted and depressed position when a shoulder injury is present.

2.3.2.2 Palpation and Range of Motion

All bony prominences and joints should be palpated for tenderness, including the glenohumeral joint, acromioclavicular joint, coracoid process, bicipital groove, acromion, scapular, cervical spine, and along the clavicle. Range of motion should be assessed both at the glenohumeral and scapulothoracic joints. The examiner should look for asymmetry or winging of the scapula (scapular dyskinesis). This may be secondary to weakness of the periscapular musculature or, although rare, possible nerve injury. Throwers should be assessed particularly in external and internal rotation in both the seated and supine positions. In the abducted shoulder, decreased motion in internal rotation and increased in external rotation may be due to posterior capsular tightness, and this may lead to an overall loss of motion. Discrepancies in passive and active range of motion may suggest muscle dysfunction or restriction secondary to pain.

2.3.2.3 Strength Testing

It is also important to test the strength of all rotator cuff muscles, scapular muscles, and the deltoid during physical examination. Testing of the subscapularis includes the lift off and the belly press. During the *lift-off test*, the patient places the dorsum of the hand on his or her buttock. With a subscapularis dysfunction, the patient will not be able to lift their hand off the buttock in this position. The *belly press test* will also elicit a limitation in internal rotation strength. As the patient firmly presses his or her hand into the lower abdomen with the elbow kept forward, they will not be able to maintain this position with a subscapularis dysfunction. The infraspinatus and teres minor can be evaluated for their strength in external rotation by having the patient resists external rotation with his or her arm by the side and abducted to 90°. It is common for an overhead athlete to have an increased strength in internal rotation in the dominant arm as compared to the nondominant arm. This may also coincide with a slight decrease in strength in external rotation and abduction.

The supraspinatus is assessed with the arm in 90° of flexion and full pronation with the thumbs pointing inferiorly. The examiner applies a downward force, with pain and weakness indicating a dysfunction of the supraspinatus/inflammation or impingement. The *drop arm test* can also be used to assess the supraspinatus. The patient's arm is brought to 90° of flexion and full pronation, while the examiner lifts the arm and lets it drop. A deficiency in strength is noted when the patient is unable to maintain the arm in the testing position.

The biceps muscle should also be assessed for its strength. This can be performed with *Speed's test*, in which the patient elevates the arm to 90° of forward flexion. Pain with resisted force will indicate a likely diagnosis of biceps tendinitis. *Yergason's test* is also used to evaluate for biceps tendinosis. This is performed with the patient's elbow in flexion and forearm pronation. The examiner has the patient supinate the forearm against resistance. Pain with this test localized to the area of the bicipital groove may suggest pathology within the long head of the biceps.

Scapular retraction strength can be tested with the *pinch test*. If a patient is unable to hold an isometric pinch of the scapula for greater than 15 s without pain or a burning sensation, this may denote a weakness of the periscapular musculature. Often, the scapula is more protracted and anteriorly rotated in the dominant shoulder as compared to the nondominant.

2.3.2.4 Special Tests

Specific tests have also been developed to assess for certain pathologies of the shoulder and may aid in determining a more accurate diagnosis. Stability testing should be evaluated at the glenohumeral joint in the anterior, posterior, and inferior directions. It can be performed in the seated, standing, and supine positions. The *sulcus test* is performed with the patient seated and the arm adducted. An inferior translation is applied and excessive mobility is noted as generalized laxity. The *clunk test* evaluates for instability with the shoulder elevated and circumduction of the humeral head applied. A positive test occurs when a symptomatic clunk is felt during testing.

The *apprehension test* is performed with the shoulder in maximum external rotation with the arm abducted. This can reproduce the pain symptoms felt with throwing. A positive *relocation test* occurs when this unstable sensation ceases as a posterior force is applied to the abducted and externally rotated shoulder.

The *O'Brien's test* is used to evaluate for labral pathology. The arm is forward flexed to 90° and adducted 10°. A positive test is demonstrated when pain is felt with a resisted downward pressure as the forearm is fully pronated. When the forearm is then supinated and the same test performed, the pain is reduced.

Provocative tests are also used to assess for rotator cuff impingement. *Neer's impingement sign* is positive when there is pain elicited with the arm moved into forward flexion and internal rotation. A positive *Hawkin's impingement sign* occurs when pain is exhibited with the shoulder in 90° of flexion, elbow in 90° of flexion, and an internally rotated forced applied.

It is important to note that the cervical spine should also be evaluated in every shoulder examination. There is the possibility that pain in the shoulder is referred from pathology of the cervical spine and/or surrounding structures. A thorough neurovascular exam should also be assessed to evaluate for other more significant pathologies.

2.3.3 Imaging Studies

2.3.3.1 Radiography

Radiographs of the shoulder should, at minimum, include anteroposterior, axillary, and outlet views. Other views to evaluate for more specific pathology can be added to these basic orders. The *Stryker Notch view* is obtained to evaluate for lesions of the posterior humerus, such as a *Bennett lesion* (posterior glenoid exostosis) and a *Hill-Sachs lesion*. To identify *Bankart lesions*, a *West Point view* can be used. A Zanca view can be used to evaluate the acromioclavicular joint as well.

2.3.3.2 Computed Tomography

CT scans are not typically used for diagnostic measures in the shoulder of the overhead athlete. It is, however, used at times to further evaluate for bony Bankart or Hill-Sachs lesions and may also demonstrate labral tears when evaluated using a contrast arthrogram study.

2.3.3.3 Magnetic Resonance Imaging

MRI is the most common imaging study to evaluate pathologies of the thrower's shoulder. It enables visualization of rotator cuff and labral injuries, as well as muscular degeneration. Gadolinium-enhanced arthrography is a useful adjunct to further visualize intra-articular injuries, especially labral tears and partial-thickness rotator cuff tears. It is important to note that many throwers will have abnormalities of the shoulder seen on MRI that may not be symptomatic, so clinical correlation is always necessary.

2.4 Specific Shoulder Injuries/ Pathology in Overhead Athletes

2.4.1 Rotator Cuff Disorders and Impingement

A properly functioning rotator cuff that is well conditioned is essential when exposed to the extreme forces that occur with the normal throwing motion. Rotator cuff injuries can include impingement, tendinitis, and tearing. Rotator cuff tears can include partial thickness, intratendinous, or full thickness. Rotator cuff injuries that occur in throwers usually include partial-thickness, articular-sided tears. This is due to the repetitive microtrauma that occurs with regular throwing. Rotator cuff tears in this athletic population can be secondary to instability, whereas in the older population, they occur more typically in the setting of chronic degeneration or impingement (Gomoll). The overhead athlete may complain of diffuse pain in the shoulder that is exacerbated with overhead activity. They may also notice weakness and decreased velocity with throwing.

The rotator cuff muscles function highest in the late cocking phase of a throw, aiding in the movement of the shoulder to maximum external

rotation. The rotator cuff muscles are also violently activated in the deceleration phase as significant shear forces occur across the joint. The insertion site of the supraspinatus has been noted as a watershed area with diminished blood flow to it and, due to this, is susceptible to repetitive stresses. These stresses of throwing may add to increased degeneration through time. Despite this, full-thickness tears of the rotator cuff are not common in the overhead athlete. When they occur, unfortunately a low percentage of players are able to return to play, even when repaired. This is why preventative measures, including rotator cuff conditioning and proper mechanics, are so important to the overhead athlete. When injury to the rotator cuff does occur, a dedicated strengthening and rehabilitation program for the shoulder, scapula, and core, as well as initiating an interval throwing program, may prevent the need for surgical intervention and allow for return to play.

Impingement is also a common shoulder pathology in the overhead athlete. Several types of impingement have been described in the literature. Classic *subacromial* or *outlet impingement* was the first classified by Neer. It occurs as a result of rotator cuff compression between the coracoacromial arch and the humeral head. This type of impingement is typically secondary to a hooked or laterally sloped acromion and/or acromioclavicular joint arthritis with subsequent inferior osteophyte extension and occurs more commonly in the older throwing athlete. Subacromial impingement can be further aggravated by rotator cuff weakness or dysfunction and subsequent superior migration of the humeral head. Many of these patients will improve with nonsteroidal anti-inflammatory medicines (NSAIDs), a possible subacromial corticosteroid injection, and physical therapy that focus on rotator cuff, scapular, and core strengthening. Arthroscopy with subacromial decompression and rotator cuff debridement is reserved only for those that fail conservative measures.

Secondary impingement, also known as *non-outlet*, results when the subacromial arch is normal, but the humeral head is within too close proximity of the arch and compressing the bursal side of the rotator cuff. This can occur secondary to scapular dyskinesis, as well as posterior capsular tightness. Scapular weakness causes a lack of proper scapular rotation during humeral elevation. Due to this, the space available for rotator cuff is narrowed, causing symptoms of impingement. Another cause of secondary impingement is malunion of a displaced fracture of the greater tuberosity. Also, large rotator cuff tears can cause superior humeral head migration and subsequently impingement. Treatment of secondary impingement is based on the primary etiology, whether that is rotator cuff tearing, scapular dyskinesis, or capsular in origin.

Coracoid impingement results when the tendon of the subscapularis is compressed within the lesser tuberosity and the tip of the coracoid. This occurs secondary to postoperative changes, trauma, anterior instability, or idiopathic causes. This is usually a diagnosis of exclusion. Patients most often localize pain to the coracoid process with the shoulder in forward flexion, adduction, and internal rotation. Conservative measures often provide relief of coracoid impingement including a diagnostic injection of local anesthetic. However, a coracoidplasty can be corrective if those treatments fail (Gomoll).

Internal impingement occurs when the undersurface or articular side of the rotator cuff comes in contact with the posterior-superior labrum as the arm is maximally externally rotated and abducted. This is most likely due to recurrent microtrauma and may coincide with SLAP lesions (to be discussed later), partial-thickness rotator cuff tears, hyperlaxity of the anterior glenohumeral ligaments, and contracture of the posterior capsule. It accounts for a significant percentage of rotator cuff and labral tears that occur in overhead athletes. Internal impingement may also be due to muscle fatigue of the shoulder girdle as the humerus shifts out of the plane of the scapula. This is termed as hyperangulation. The athlete with internal impingement may often complain of posterior shoulder pain in the late cocking and early acceleration phases. Conservative management of internal impingement should include improving throwing mechanics, core strengthening, scapular kinesis, internal rotation stretching, and rotator cuff strengthening. Surgical

management if those treatments fail is to repair the labrum, debride partial-thickness tears, and/or reduce the laxity of the anterior inferior glenohumeral ligament (Braun).

2.4.2 Glenohumeral Internal Rotation Deficit (GIRD)

The concept of GIRD is a common phenomenon in overhead athletes and is used to describe the loss of internal rotation in the dominant shoulder, which can be greater than 25°. It is based on the increased prevalence of contracture of the posterior capsule and the posterior band of the inferior glenohumeral ligament in throwers. When these contractures develop, the contact point of the humerus on the glenoid shifts posterosuperiorly and causes a relative stretch to anterior aspect of the capsule. This provides more clearance for the greater tuberosity and results in excessive external rotation. The biceps anchor is also "peeled back" with tension causing further laxity of the anterior aspect of the capsule (known as the "*peel-back progression mechanism*"). These changes culminate in torsional failure of the rotator cuff and may cause the articular-sided partial-thickness tears as well as SLAP lesions that are commonly seen in the shoulder of overhead athletes.

The treatment of GIRD in throwers is typically a conservative rehabilitation program that focuses on stretching of the tight posterior capsule (sleeper stretch). Those that are not successful with this may opt to be treated with selective arthroscopic posteroinferior capsulotomy in the region of the posterior band of the inferior glenohumeral ligament.

2.4.3 Laxity and Instability

Glenohumeral instability with its associated internal impingement are well studied but not among the completely understood components of pathology in the shoulder of the overhead athlete. It is important to note that laxity is a separate entity from instability. Laxity is defined as excessive motion in a particular direction at a joint. It may be a normal property of the soft tissues at that joint, or it also may be an adaptation that occurs. Although some laxity is essential for overhead athletes, excessive laxity may lead to the development of pathology, such as labral and/or rotator cuff tears.

Instability occurs when there is translation of the humeral head in the glenoid and is usually associated with pain and discomfort. It can occur as primary, posttraumatic, or microinstability. Primary instability occurs when generalized ligamentous laxity is present. Posttraumatic instability is the result of a distinct traumatic event. Microinstability results from repetitive stresses, especially from the cocking and acceleration phases of a throw. Stretching and failure of the anterior capsule occurs through time, as well as anteroposterior translation of the humeral head. This can eventually lead to fraying of the labrum, subacromial impingement, rotator interval laxity, and tearing of the rotator cuff.

2.4.4 Capsulolabral Pathology

The labrum is a fibrocartilaginous structure that surrounds and deepens the glenoid. It is the site of attachment of the long head of the biceps, as well as the superior and middle glenohumeral ligaments. The superior and inferior portions of the labrum are the most susceptible to injury. Labral tears may affect many overhead athletes and can certainly be debilitating, especially if they involve the superior labrum and biceps anchor. These are termed *superior labrum, anterior-posterior* (*SLAP lesions*).

SLAP lesions are divided into ten types. Type I lesions are very common in throwing athletes due to the extreme forces in external rotation of the shoulder. Increased strain of the biceps anchor also occurs during the late cocking phase, adding to the possible development of SLAP lesions. The long head of the biceps does contribute as a restraint to maximal external rotation in the abducted arm. It has been noted that there is an increased incidence of SLAP lesions especially in throwers that have an overall decreased range

of motion due to the deficits in internal rotation greater than the gain of external rotation (as discussed previously within this chapter). Type II lesions, defined as a true avulsion of the biceps anchor from the superior glenoid, occur less frequently. Type II lesions are further divided into Type IIA (anterior), Type IIB (posterior), and Type IIC (anterior/posterior). Type III lesions are defined as bucket handle tears with an intact biceps and most commonly occur secondary to a fall on an outstretched arm. Type IV SLAP tears are bucket handle with biceps extension. Type V lesion is either as a Bankart lesion with extension superiorly or a SLAP lesion with anteroinferior extension. Type VI represents an anterior or posterior flap tear. Type VII SLAP tears involve a middle glenohumeral ligament extension and most commonly occur from acute trauma or anterior dislocation. Type VIII is similar to type IIB but with more extensive abnormalities and most commonly occur after acute trauma or posterior dislocation. Type IX involves global labral abnormalities and also occurs with trauma. Type X lesions involved rotator interval extension as well as articular-sided abnormalities.

SLAP lesions will present with vague pain in the dominant shoulder, at times at the posterosuperior joint line, that is aggravated by overhead activities. Throwers typically report pain in the late cocking phase of the throw and an overall loss of velocity. Symptoms of locking, snapping, and instability are often described as well. Radiographic workup should include radiographs as well as an MRI arthrogram to better visualize the labrum.

Treatment of SLAP lesions begins with the conservative measures of rest and rehabilitation. If those treatments do not enable the athlete to resume throwing, surgical intervention may be necessary. Surgical treatment of SLAP lesions begins with shoulder arthroscopy. If the biceps-labral anchor is avulsed, it is debrided and sutured back to the glenoid with anchors. If only minor fraying is present and the biceps anchor is attached, simple labral debridement is performed. Following a period of immobilization, a rehabilitation program that focuses on throwing mechanics is important. Return to play for patients with biceps anchor detachment can be 6–9 months; however, return to prior level of throwing may take up to a year. Patients with a stable biceps anchor at the time of surgical intervention who have only undergone simple labral debridement may typically resume play after 8–12 weeks of physical therapy.

2.4.5 Bennett Lesion

A *Bennett lesion* is defined as a mineralization of the posteroinferior glenoid and is seen in roughly 20 % of major league pitchers [2]. It is thought to result from enthesopathic changes among the inferior glenohumeral ligament and the posterior capsule. A Bennett lesion can cause pain for throwing athletes and may occur along with posterior labral and rotator cuff tears. Symptoms include pain in the posterior shoulder especially in the follow-through phase of throwing. Conservative treatments can be initially attempted and arthroscopic debridement for those with recurring pain.

2.4.6 Scapular Dyskinesis

It is important to take scapular dynamics into consideration when evaluating the thrower's shoulder. The scapula provides a stable platform for the humeral head during rotation and elevation. It provides a base for the rotator cuff muscles to exert their forces on the glenohumeral joint and allow for movement of the upper extremity. It also helps transfer kinetic energy from the legs and trunk to the dominant upper extremity.

Scapular dyskinesis is abnormal positioning and scapular motion and occurs when imbalances of the periscapular muscles are present. This may be due to fatigue, direct trauma or, less frequently, nerve injury to the long thoracic nerve. These imbalances can directly impact shoulder function. Scapular hyperangulation and an increase in glenoid anteversion may occur, which places the anterior capsular structures at risk for subsequent injury. Scapular dyskinesis also results in loss of velocity since the scapula transfers energy derived from the trunk during a throw. This places more

stress on the shoulder as it tries to compensate for this loss of power. The majority of scapula-related issues can be treated with rehabilitation focusing on scapular stabilization.

2.4.7 Neurovascular Shoulder Conditions

Vascular injuries are typically rare among athletes and occur more commonly with major trauma. However, when they do occur, they can cause significant morbidity. *Effort thrombosis* is an injury that, although uncommon, can be seen in a wide range of sports, including overhead athletes. It presents with symptoms of tiredness, heaviness, and swelling, particularly in the dominant arm, over the course of several days. Physical signs can include slight discoloration, engorgement of the venous system, and size difference as compared to the opposite arm. Venography or MRI may show thrombosis of the subclavian vein at the first rib. It is shown to be caused by compression of the vasculature between the first rib and the clavicle, especially as the arm is maximally abducted and externally rotated. Treatments usually require intervention with catheter-directed thrombolysis, balloon venoplasty, or resection of the first rib. Results are positive and return to prior level of play may occur within 6–36 months.

Thoracic Outlet Syndrome (TOS) is a compression of the neurovascular structures (i.e., brachial plexus and subclavian artery) that travel through the thoracic outlet, which is comprised of the clavicle, first rib, and anterior scalene muscle. TOS in overhead athletes may be secondary to excessive muscle development or depression of the scapula due to dyskinesis. Presenting symptoms include pain, paresthesias, and weakness in the lower plexus distribution. Vascular symptoms are rare and if occur include activity-related claudication or pulse/blood pressure changes. Diagnostic testing is nonspecific so diagnosis is usually clinical. Holding the arm in maximal abduction and external rotation may recreate the symptoms. Adson's test is used to test for TOS and is performed with the patient standing. The examiner palpates the radial pulse while moving the upper extremity in abduction, extension, and external rotation. The patient then rotates the head toward the involved side and while taking in a deep breath and holding it. A positive test will reveal a diminished or absent radial pulse. Conservative treatment includes activity modification, NSAIDs, and rehabilitation. Surgical decompression with resection of the first rib is performed for those who fail these measures.

Quadrilateral space syndrome is another neurologic condition that may occur in throwing athletes. It is compression of the axillary nerve and posterior humeral circumflex artery as they travel through the quadrilateral space, which is defined laterally by the humerus, medially by the long head of the triceps, teres minor superiorly, and teres major inferiorly. Symptoms are usually dull, aching, and/or burning pain at the posterolateral portion of the shoulder. These symptoms increase with activity, especially as the arm is abducted and externally rotated, such as in the throwing motion and specifically within the late cocking phase. Physical exam findings may include weakness and deformity of the deltoid, pain over the muscle at the quadrilateral space, and reproduction of symptoms when abducted and externally rotated. Angiography may be used to confirm occlusion at the posterior humeral circumflex artery. Electromyography may show denervation within the deltoid and teres minor. Treatments may begin conservatively and surgical intervention with release of the compressed neurovascular structures may be performed for refractory cases.

2.5 The Pediatric Throwing Shoulder

2.5.1 Little Leaguer's Shoulder

Little Leaguer's shoulder is defined as a stress fracture of the proximal humeral physis and is seen most often among 13–16-year-old throwers. The rotator cuff muscles attach proximal to the physis and the pectoralis major, deltoid, and triceps muscles attach distally. Athletes with Little Leaguer's Shoulder present with lateral aspect shoulder pain with hard throwing. Dull,

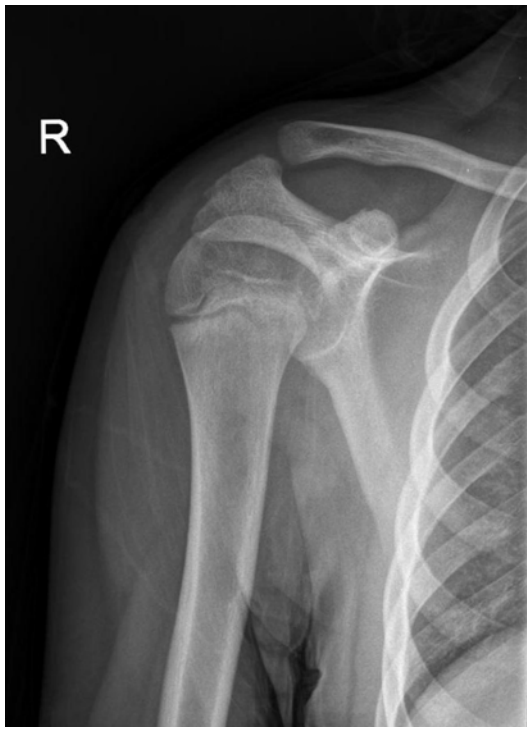

Fig. 2.4 Little league shoulder in a 14-year-old male baseball pitcher (Rothman Institute Philadelphia, PA)

aching pain at rest presents later in the course of the injury. The classic radiographic finding on an AP view of the shoulder is a widening of the lateral aspect of the physis in external rotation (Fig. 2.4). The condition is most often benign and self-limiting. However, it is important to counsel the athlete about a period of rest until symptoms resolve before the return to throwing. This may take up to 3 months. Rehabilitation including strengthening of the rotator cuff may also be implemented and a return to throwing program designed for the skeletally immature thrower.

2.6 Throwing Shoulder Conditioning Programs and Rehabilitation Protocols

From the previous sections of this chapter, one can see the immense complexity of the shoulder pathology in the overhead athlete. The prevention of injuries should be implemented into every throwing program. Core and lower body exercises should be included in training. Any deficits in strength and endurance of the lower body will have a great impact on the forces of the upper extremity during normal pitching mechanics. Core stabilization also takes into account the concept of the kinetic chain. If there is imbalance at any point within the chain, pathology may result.

Off-season preparation is also an important aspect of training. The off-season is a valuable time for rest and recuperation but must also include strength and endurance exercises so that the athlete will be able to return to competition with adequate physical fitness so as not to sustain an injury. The off-season can also be a time to rehabilitate any lingering injury that may have occurred in-season.

2.6.1 Rehabilitation Progression

When injury occurs to the throwing shoulder, it is important to restore motion, muscular strength, stability, proprioception, and endurance in a stepwise process. The *acute phase* of rehabilitation begins following surgery or an injury. Initial goals are to diminish pain and inflammation. Further steps include improving posterior flexibility, strength, and dynamic stability. Modalities may include iontophoresis, phonophoresis, electrical stimulation, and cryotherapy. Exercises will be restricted to closed kinetic chain.

To progress to the next phase, the athlete must have minimal pain and inflammation, normal internal rotation, and adduction, and his or her baseline muscular strength without fatigability. The *intermediate phase* can then progress with strengthening exercises, restoring muscular balance, and maintaining flexibility and mobility. A full rotator cuff and scapular isotonic strengthening program may be implemented. During this phase, the athlete may also begin open chain exercises and two-handed plyometric throws.

For further progression, the athlete must have full, pain-free range of motion of the shoulder and full strength without fatigue. Phase 3 is the *advanced strengthening phase* and includes improving strength, power, neuromuscular control,

and endurance as well as the initiation of light one-handed throwing activities.

The last phase is the *return-to-activity phase* and can begin once adequate dynamic stability and proprioception is achieved. A progression to throwing program may be implemented at this point to allow return to competitive throwing. This includes a full shoulder program and plyometrics. Throwing begins at shorter distances with low intensity and progresses to full distance and maximum effort and velocity.

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3 Physical Examination of the Shoulder and Elbow with a Focus on Orthopedic Special Tests

Eric J. Hegedus

3.1 Introduction to the Physical Examination

Physical examination, whether of the shoulder or the elbow, is a critical skill for any practicing clinician. The physical examination can only be mastered with practice and, especially for the new learner, involves a defined and consistent series of steps that assist in the systematic collection of patient-related data in order to facilitate efficient and effective decision-making.

Physical examination is a manual skill composed of defined steps that require repetition to learn. The examination is also repeated as the patient's status changes over time. Whether for the initial examination or the reexamination, the steps that are required systematically progress from patient history and observation through screening,motion testing, then concluding with palpation, muscle testing, and special tests [1].

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3.2 Patient History

3.2.1 Onset

Assuming relevant past medical history has been gathered and reviewed, the efficient practitioner should focus on the history of the presenting injury. Generally, the patient's history can be divided by onset of injury as either insidious or acute. Insidious injuries are often due to overuse and can indicate pathologies like tendinopathy (e.g., rotator cuff, tennis elbow, golfer's elbow) and instability from repeated microtrauma (e.g., glenohumeral, ulnar collateral ligament). Acute injuries are often traumatic (e.g., fracture, dislocation). Labral tears can either be acute or degenerative in nature which contributes to these injuries being difficult to diagnose with the clinical examination.

3.2.2 Concordant Sign

Whether insidious or acute onset, there is almost always one prevailing symptom that causes the patient to seek care, and this symptom is called the concordant sign. The concordant sign is often pain but could also include numbness or tingling, weakness, or sensations associated with instability like catching and subluxation.

There are many ways to assess pain. The most common are the 11-point (rate your pain on a 0–10 scale) numerical scale; the categorical "mild," "moderate," and "severe" scale; and the

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10 cm visual analogue scale. Likely the best of the pain rating scales is the horizontal scale with five gradations [2]:

- Little
- Mild
- Moderate
- Severe
- Agonizing

For further elucidation of how pain is affecting the patient's life, clinicians often ask for ratings of pain at rest, at different times of day, and with activities. In order to get an idea of the irritability of the pain symptoms, helpful inquiries will address aggravating activities, relieving strategies, and how long the pain stays at maximum level once provoked.

3.2.3 Patient Self-Assessment of Function

In addition to an appreciation of pain and irritability, it is critical for the clinician to understand how their patient is functioning with his or her injury. There are many scales to assess the patient's perception of their function, and these scales can be disease specific (e.g., rotator cuff tear), region specific (e.g., the upper extremity), or joint specific (e.g., the shoulder). For the busy practitioner, generic measures of function are probably best due to their minimal time to complete and ease of use. Two informative generic self-report measures are the single assessment numeric evaluation (SANE) [3] and the Patient-Specific Functional Scale (PSFS) [3]. The SANE asks "How would you rate your shoulder (or elbow) today as a percentage of normal (0–100 % scale with 100 % being normal)?" The PSFS asks the patient to identify up to three important activities that they are unable to do or have trouble doing as a result of their injury and then rate each activity on a 0 (unable to perform the activity) to 10 (able to perform activity at the pre-injury level). Asking detailed pain questions and obtaining the patient's impressions on how pain and injury are affecting their ability to function are two important early steps in the diagnostic process.

3.3 Observation

Observation is most effective when the practitioner begins in a general fashion and proceeds to a more specific and localized inspection. The purpose of proceeding in this fashion is that some pathologies that refer pain and other symptoms to the shoulder and elbow are systemic or regional in nature. An example of systemic disease that causes shoulder pain is rheumatoid arthritis and an example of a regional pathology that may affect the shoulder is cervical radiculopathy, especially in cases where the radiculopathy causes weakness in the arm. Even those pathologies that are local in nature may have regional influences. For example, impingement symptoms in the glenohumeral joint may be made worse in individuals with increased thoracic kyphosis. Also, a localized deep vein thrombosis (DVT) in the axillary-subclavian vein, Paget-Schroetter syndrome, can cause swelling, temperature, and color changes in the arm.

Localized inspection most commonly detects bony asymmetries, soft tissue asymmetries, and ecchymosis. In the shoulder region, one of the most often looked for and, therefore, found asymmetries is scapular dyskinesis. Scapular dyskinesis is defined roughly as a difference in the movement pattern between the scapulae. Dyskinesis is best assessed during repeated elevation of the arm holding a 3–5 lb weight [4] (Fig. 3.1). While dyskinesis is not diagnostic of a particular pathology of the shoulder, it is an

Fig. 3.1 Assessing scapular dyskinesis. The subject performs 3–5 repetitions in each of scaption (pictured) and flexion

impairment often seen in throwing athletes and may be important to address during treatment.

The diagnostic value of observation has only been reported in reference to the shrug sign [5], an elevation of the entire shoulder girdle while attempting elevation of the arm (Fig. 3.2). When the shrug sign is negative, glenohumeral osteoarthritis, adhesive capsulitis, and severe rotator cuff tendinopathy can be ruled out as diagnoses with relative certainty.

Fig. 3.2 Shrug sign. The patient demonstrates the shrug sign in the right shoulder

3.4 Screening

The objective of an effective screen is to raise suspicions of and help rule out non-musculoskeletal sources of pain and also to help focus the physical examination in situations where symptoms encompass more than one joint. The standard screening examination that is inclusive of the shoulder and elbow is the upper quarter screen. The screen combines active motions of the cervical spine and combined motions of the shoulder and elbow (Fig. 3.3a, b) with upper motor neuron testing and testing of dermatomes, myotomes, and reflexes (Table 3.1). Overpressure can be applied by the examiner to further stress the active motions. A more detailed examination can include 2-point discrimination, testing of vibration sense, and vascular testing.

Reproduction of the concordant sign during active and combined motions indicates that a more thorough examination (see following sections of this chapter) of the offending joint should be performed.

Combined testing of dermatomes, myotomes, and reflexes, especially with findings of decreased sensation, weakness, and hyporeflexia when compared to the opposite arm, should alert the

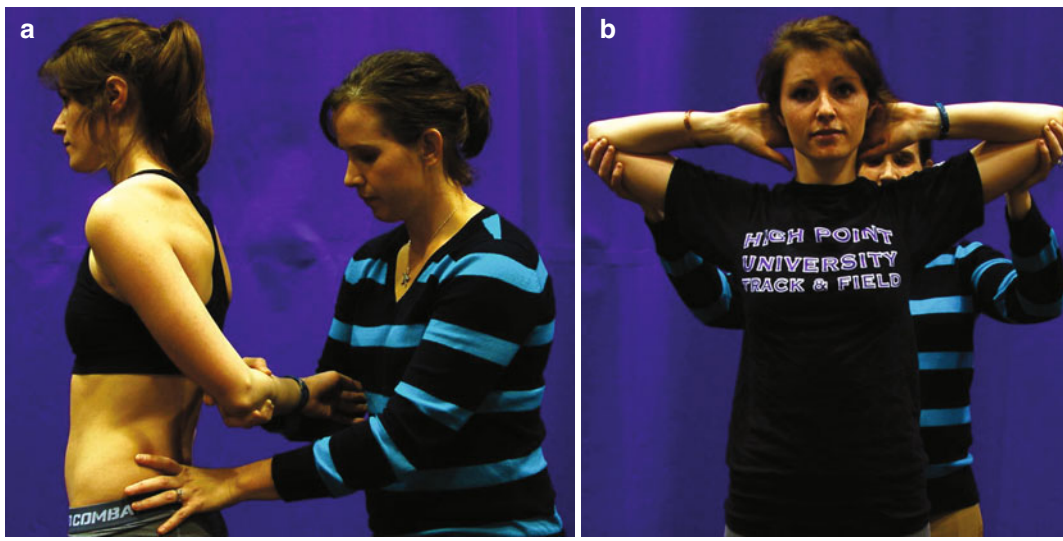

Fig. 3.3 (**a**) Combined motions of the shoulder and elbow with overpressure. Shoulder extension, adduction, and internal rotation. (**b**) Combined motions of the shoulder and elbow with overpressure. Shoulder abduction and external rotation with elbow flexion

Table 3.1 Dermatomes, myotomes, and reflexes of the upper quarter screen

	C1–3	C4	C5	C6	C7	C8	T1
Dermatome	Side of face	Suprascapular region	Deltoid region	Thumb and pointer finger	Middle finger	Ulnar border of hand	Medial border of upper arm
Myotomes	Cervical rotation	Shoulder shrug	Shoulder abduction	Elbow flexion, wrist extension	Elbow extension	Thumb extension	Finger abduction
Reflexes	None	None	Biceps, brachioradialis	Biceps, brachioradialis	Triceps	Triceps	None

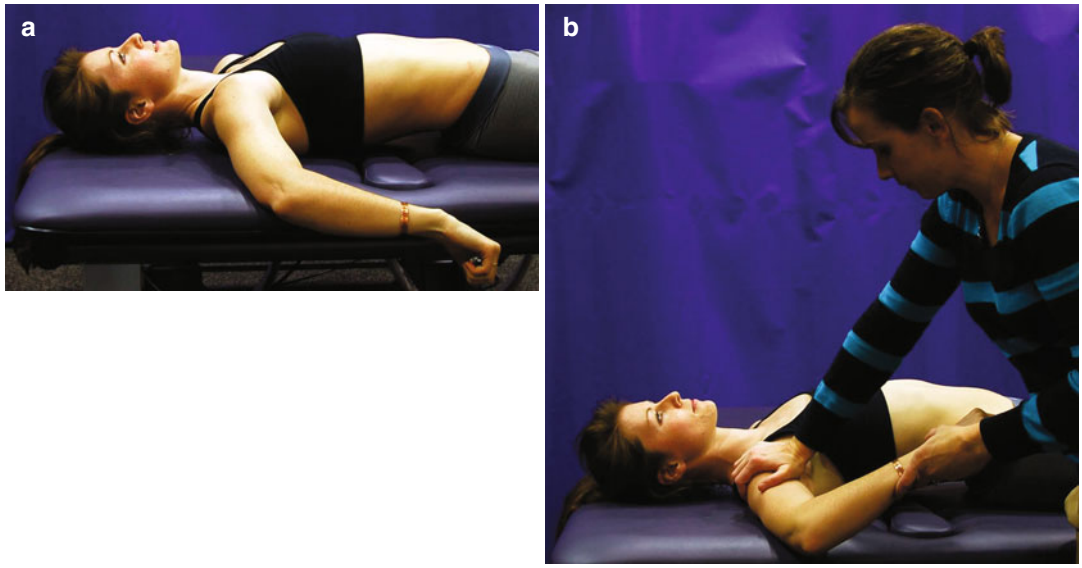

Fig. 3.4 (**a**) Gross internal rotation of the shoulder. Accomplished with anterior tipping of the scapula. (**b**) Isolated glenohumeral motion. Internal rotation is far less when anterior tipping of the scapula is controlled

examiner to the likely presence of cervical radiculopathy.

Testing for an upper motor neuron lesion (spinal cord or brain) in the upper quarter screen is best done with Hoffman's test, which is performed by flexing or flicking the distal phalanx of the middle finger. A positive test is indicated by flexion of the thumb or the distal phalanxes of the other fingers (clawing).

3.5 Motion Testing

3.5.1 Active Motion

Active motions at the shoulder typically performed are flexion, extension, abduction, horizontal adduction, internal rotation, and external rotation. Internal rotation and external rotation are often repeated with the arm in 90° of abduction to more closely mimic the position of the arm during throwing. Active elbow motions include flexion, extension, forearm pronation, and forearm supination. In the presence of trauma, the inability of the patient to fully flex, fully extend, fully pronate, or fully supinate is a highly specific sign for fracture [6]. Whether testing the shoulder, elbow, or both, active motion is performed in comparison to the other arm, and reproduction of the concordant sign is an important finding.

3.5.2 Passive Testing

Passive motions performed at the elbow are the same as active and for the same reasons: to note limitations and excesses (instability) and to look for the concordant sign. A global loss of shoulder motion is highly indicative of adhesive capsulitis [7]. Passive motions at the shoulder are also the same as active with the exception that most clinicians perform isolated glenohumeral flexion, abduction, and internal and external rotation at 90° abduction. The reason that glenohumeral motions are isolated is that gross movements at the shoulder are accomplished with contribution of other joints like the acromioclavicular and sternoclavicular joints as well as adjacent areas of the body like the scapulothoracic pseudo-joint and the thoracic spine. Isolating the glenohumeral joint (Fig. 3.4a, b) gives a better comparison of limitation at the shoulder and is likely to produce the concordant sign in glenohumeral

Fig. 3.5 The hyperabduction test. Greater than 105° of isolated glenohumeral abduction is indicative of inferior laxity

pathology. A specific example can be found in isolated abduction, called the hyperabduction test [8], which can be used to detect inferior laxity or instability (Fig. 3.5). Another example is that many throwing athletes display excessive isolated glenohumeral external rotation with an accompanying loss of isolated glenohumeral internal rotation.

3.5.3 Accessory Motion

The final motions tested are accessory motions, which can be defined as the gliding of one joint surface on another. Sometimes referred to as stability testing, accessory motions are labeled by the direction in which the moving side of the joint is pushed or pulled. For example, a posterior-to-anterior accessory motion of the glenohumeral joint would indicate that the humeral head was mobilized in an anterior direction in reference to the glenoid fossa. Excessive motion with this accessory test, especially when accompanied by patient apprehension, would lead the clinician to suspect anterior instability. Other examples of accessory motion findings and the interpretation of those findings can be found in Table 3.2. In addition to limitations or excessive motion during testing, the clinician also should take note of reproduction of the concordant sign.

Table 3.2 Examples of accessory motions and interpretation of findings

Accessory motion and finding	Interpretation of finding
Glenohumeral anterior to posterior → limited	Tight posterior capsule—often present in patients with impingement pain
Glenohumeral superior to inferior (with arm at side) → excessive or painful	Inferior laxity or instability—injury to the superior labrum, biceps, or coracohumeral ligament
Glenohumeral superior to inferior (with arm at 90° abduction) → excessive or painful	Inferior laxity or instability—injury to the inferior capsuloligamentous structure
Radiohumeral lateral glide → excessive or painful	Radial head laxity or instability—disruption of the annular ligament or fracture of the proximal radius

3.6 Palpation

Palpation is performed next in the systematic examination and the process of palpation should be very directed based on the results of history, observation, screening, and motion testing. While many clinicians palpate in an effort to detect side-to-side differences in bony and soft tissue architecture, there is no research at present to support the value of this approach at either the shoulder or elbow. However, there is value in palpation of the long head of the biceps (tendinopathy) [9], the supraspinatus (tendinopathy) [9], and the acromioclavicular (AC) joint (AC pathology) [10]. Palpation of these structures is a sensitive test and therefore, an absence of pain when palpating contributes to ruling out the associated pathologies.

3.7 Muscle Testing

Traditionally, muscle testing is performed as a manual muscle test in cardinal planes of movement, and comparisons are made between arms with regard to pain and strength. In the shoulder, then, flexion, abduction, internal rotation, and external rotation are most often tested, while at

the elbow, the most common tests are flexion, extension, pronation, and supination.

In cases where the shoulder examination reveals a clinically significant scapular dyskinesis, many clinicians will perform manual muscle testing of the muscles about the scapula, for example, the serratus anterior, the rhomboids, and the trapezius.

In an attempt to have strength testing more closely mimic function, physical performance tests for the upper extremity have been developed. An example of a physical performance test that assesses the coordinated muscle activity of the entire upper extremity is the closed kinetic chain upper extremity stability test (CKCUEST) [11]. However, physical performance tests have not been used for diagnosis but, instead, to quantify recovery after injury or surgery.

3.8 Special Tests

At this late stage in the examination process, the examiner should be discerning between a few closely related, competing diagnoses. Special tests are best used at this stage of the examination process. Some special tests are best at confirming a diagnosis with a positive finding, and some are best at ruling out a diagnosis with a negative finding, but very few tests are effective at both. Therefore, the examiner must remember which tests are valuable when positive and which are valuable when negative. The best tests as determined by the highest level of research and the greatest metrics of diagnostic accuracy are summarized in Tables 3.3 (shoulder) and 3.4 (elbow). Photographs of these tests can be found elsewhere [24]. There is a decided lack of evidence-supported special tests

Table 3.3 The best shoulder special tests and their use

Test name	Brief description of test	Pathology	Rule in when positive	Rule out when negative
Surprise test [12]	The patient is supine with the shoulder in 90° abduction and the elbow in 90° flexion. The patient should report pain or display apprehension followed by the examiner applying an anterior-to-posterior force on the humeral head, relieving pain or apprehension. The examiner then releases the humeral head and the patient again reports pain or registers apprehension	Anterior instability	✓	✓
Jerk test [13]	A force along the long axis of the humerus is maintained as the examiner moves the patient's arm from horizontal abduction to horizontal adduction. A positive test is pain with or without a clunk	Posterior labral tear	✓	
Passive compression test [14]	The patient is in side lying. The examiner externally rotates the shoulder in 30° abduction and pushes the humeral head proximally into the acromial arch. Maintaining this position, the examiner extends the shoulder looking to reproduce pain	Superior labrum anterior-to-posterior (SLAP) tear	✓	✓

(continued)

Table 3.3 (continued)

Test name	Brief description of test	Pathology	Rule in when positive	Rule out when negative
Modified dynamic labral shear [15]	The patient is standing. The examiner places the shoulder into the cocked position for overhead throwing (120° elevation, end range external rotation). The examiner maintains this position while lowering the arm from 120° to 60° attempting to elicit pain	Labral tear	✓	
Lateral Jobe test [16]	The patient is seated. The patient's shoulder is in 90° abduction and internally rotated with the elbow in 90° flexion. The examiner attempts to push the patient's arm in an inferior direction while the patient resists. A positive test is pain, weakness, or inability to perform the test	Rotator cuff tear	✓	✓
Belly-off test [17]	The patient is standing with the palm of the hand on the abdomen. While holding the patient's hand on the abdomen, the examiner moves the patient's arm into maximum internal rotation by raising the elbow. When the hand is released, a positive test is the inability by the patient to maintain the palm on the abdomen	Subscapularis tendinopathy	✓	✓
AC resisted extension test [18]	The patient's shoulder and elbow are in 90° flexion and horizontally adducted. The examiner resists the patient as he or she moves into horizontal abduction. A positive test is pain in the acromioclavicular (AC) joint	Acromioclavicular (AC) joint pathology	✓	
Bony apprehension test [19]	The patient is seated with arm in 45° abduction. With the examiner guiding the arm, the patient will register apprehension or pain as their shoulder nears 45° of external rotation	Bony instability	✓	✓
Olecranon-manubrium percussion test [20]	The patient is seated with arms crossed. The examiner places a stethoscope on the patient's manubrium and taps the patient's elbow. A more muted sound is indicative of a fracture or dislocation somewhere between the elbow and manubrium and indicates the need for an x-ray	Fracture/dislocation	✓	

Table 3.4 The best elbow special tests and their use

Test name	Brief description of test	Pathology	Rule in when positive	Rule out when negative
Elbow flexion test [21]	Patient is seated with both elbows in full flexion with wrists extended. A positive test is reproduction of symptoms along the ulnar nerve	Cubital tunnel syndrome	✓	
Tinel's sign [21]	The examiner taps the patient's ulnar nerve repeatedly just proximal to the cubital tunnel. A positive test is reproduction of symptoms along the ulnar nerve	Cubital tunnel syndrome	✓	
Moving valgus stress test [22]	The patient is seated with the shoulder abducted 90° and in full external rotation and the elbow flexed 120°	Ulnar collateral instability		✓
Biceps squeeze test [23]	The patient rests their arm on their lap with the forearm in slight pronation. The examiner squeezes the biceps and a positive test is failure of the forearm to supinate	Distal biceps tendon rupture	✓	✓

for biceps pathology, although many have been proposed. There is also a surprising lack of tests for medial and lateral epicondylosis.

Conclusion

In summary, diagnosis through clinical examination is an invaluable skill that is important for an effective and efficient clinician. The examination can help make valuable clinical decisions about diagnosis and referral, determine the need for further imaging and lab testing, and guide care. The best diagnosticians are orderly and systematic in their approach and use the best available evidence in informing their decisions.

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Shoulder Arthroscopic Portals: Ordinary Versus Unconventional

4

Yang-Soo Kim and Hyo-Jin Lee

4.1 Introduction

Recent improvement of arthroscopy has replaced most of the conventional procedures of shoulder surgery. Being a primary treatment modality of the shoulder, arthroscopy has many advantages over the open conventional surgery. Minimal invasiveness by small incision has enabled less injury to the deltoid muscle, less postoperative pain, and potentially fast recovery after operation [4, 17]. During the process of operation, thorough visualization of the joint has enabled more precise diagnosis along with discovery of concomitant pathology.

The appropriate position of the portal is essential for the visualization of the intraarticular surgical field and the approach of the surgical instruments to the lesion. It enables to enhance anatomical reconstruction of the injured tissue and finally achieve a successful surgery. We have described the contents into two parts in this chapter: conventional portal placement and unconventional portal placement.

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4.2 Ordinary Portal Placement

4.2.1 Posterior Portal

The posterior portal is the first portal made during shoulder arthroscopy. It is the most convenient portal which can be made safely and allows adequate visualization of the entire glenohumeral joint. It enters the soft spot between the humeral head and the glenoid. A vertical, small incision is made 2–3 cm inferior and 1–2 cm medial to the posterolateral corner of the acromion [1]. Some surgeons prefer further inferior placement of the portal as the portal position may move superior as soft tissue swelling increases during the operation. In this case, close proximity with the posterior acromion may provide a poor angle of approach to the subacromial space. For the access into the glenohumeral joint, the direction of the trocar is recommended to aim toward the coracoid process. After the introduction of the trocar through the capsule, a popping sensation can be felt as the joint is entered. Localizing the joint line by palpating the humeral head and glenoid allows the correct placement of the portal within the glenohumeral joint. After proper placement of the trocar, diagnostic arthroscopy can be performed.

Even though the posterior portal can be established safely, there still lies the risk of injury on the

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nearby nerves or vessels. The axillary nerve and suprascapular nerve are the most common nerves that can be damaged during the portal establishment. The average distance of the posterior soft spot portal insertion site from the axillary nerve is 49 mm, and it can be found as close as 30 mm [12]. Medial placement of the posterior portal may put the suprascapular nerve in danger with the average distance of 29 mm. The nearest anatomic structure at risk is the suprascapular artery with an average distance of 27 mm from the soft spot [12].

The location of the portal can be modified depending on the underlying pathology or the location of the target lesion. Lower placement of the posterior portal is preferred for the approach of labral repair when compared with rotator cuff repair [16]. For the repair of large or massive cuff tear, medial placement of the portal is recommended for an easier approach of the suture device to the torn cuff.

4.2.2 Anterior Portal

The anterior portal can be created either by the outside-in or inside-out technique. The outside-in technique is performed under the direct visualization of the arthroscope. The coracoid process is an important landmark in placing the anterior portal. A spinal needle is introduced 1–2 cm inferomedial to the anterolateral corner of the acromion just lateral to the tip of the coracoid process. Care must be taken not to damage the brachial plexus and the axillary vessels that are located inferomedially [16]. Further inferior placement of the anterior portal may damage the musculocutaneous nerve and cephalic vein. According to Lo et al. [11], the location of the musculocutaneous nerve is on average 33 ± 6.2 mm inferior to the tip of the coracoid.

In case of the inside-out technique, the arthroscope is advance d toward the rotator interval just below the biceps tendon across the glenoid. Holding the cannula firmly, the arthroscope is withdrawn, and a switching stick is inserted through the posterior portal. Advancement of the stick leads to skin tenting, and a small stab incision is made at the tip of the stick which enables the stick to pass through the skin incision. A cannula is inserted over the stick and gently advanced until the capsule is penetrated.

The location of the anterior portal can be modified within the rotator interval according to the surgeon's preference or the pathology being addressed. Superior placement of the portal enables to address the superior labrum anterior posterior (SLAP) lesion as the angle for inserting a superior glenoid anchor is facilitated. Lateral placement is preferred for the anchor in the Bankart lesion repair, whereas medial placement is preferred for the anterior and inferior capsular release. Two separate anterior portals within the rotator interval can be made according to the surgeon's preference.

4.2.3 Lateral Portal

The lateral portal is most commonly used to approach the subacromial space. The anterior placement of the portal is typically for addressing the acromioclavicular joint pathology including distal clavicle resection and acromioplasty (subacromial decompression) (anterolateral portal). If the acromioplasty is unnecessary, posterior placement of the portal is possible depending on the location and the shape of the torn rotator cuff tendon. The skin incision can be placed approximately 2–3 cm lateral to the edge of the acromion. For proper acromioplasty, it is important to make the portal parallel to the undersurface of the anterolateral acromion. Prior insertion of the spinal needle is helpful for the appropriate placement of the portal. Too inferior placement of the portal may damage the axillary nerve, as it lies approximately 3 cm distal to the anterolateral margin of the acromion [5, 15]. Superior placement of the portal makes it harder to access the medial acromion or acromioclavicular joint.

Usually this portal is utilized as the working portal, but it can also be utilized as a viewing portal in case of block-cut acromioplasty.

4.2.4 Posterolateral Portal

The posterolateral portal can be utilized as a viewing portal or a working portal according to

its location. When created 2–3 cm below the posterolateral edge of the acromion, it is used as the viewing portal for acromioplasty, rotator cuff repair.

When the portal is made at the posterior lip of the tear, 1 cm from the lateral margin, it is called the rear viewing portal [9]. The rear viewing portal is capable of providing a good downward en face view of the rotator cuff tear. Cautions should be made during the portal placement. Excessive inferior placement may lead to axillary nerve injury. To avoid overcrowding of the instruments in the subacromial space, at least 2 cm of distance is needed between another lateral working portal [9].

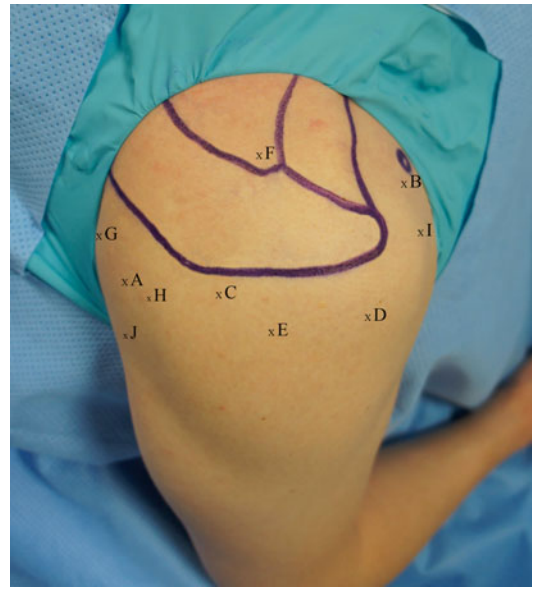

Fig. 4.1 Portal locations marked on the right shoulder. *A* posterior portal, *B* anterior portal, *C* posterolateral portal, *D* anterolateral portal, *E* trans-rotator cuff portal, *F* Neviaser portal, *G* accessory posteromedial portal, *H* axillary portal, *I* 5 o'clock portal, *J* 7 o'clock portal

4.3 Unconventional Portal Placement

4.3.1 Neviaser Portal

This portal is introduced by Neviaser TJ in 1987 and named after the inventor [14]. Also called as the supraclavicular fossa portal, it provides a great view of the anterior glenoid with convenient access of suture anchor for SLAP repair. The location of the portal is surrounded by the clavicle anteriorly and medial acromion and scapular spine posteriorly. The needle is placed through the soft spot laterally. The direction of the portal can be slightly modified by the surgeon's preference. The skin incision is made 1 cm medial to the medial border of the acromion. Suture placement under the biceps anchor is recommended for SLAP repair. The portal also can be utilized for supraspinatus repair by using a curved suture passing device such as Banana SutureLasso (Arthrex, Naples, FL). The structures that should be protected during the insertion are the suprascapular nerve and artery, which are only 3 cm away from the supraglenoid tubercle [3].

4.3.2 5 O'Clock Portal

Established by Davidson et al. [7], the 5 o'clock portal is developed for low anchor placement during the procedure of the Bankart lesion repair. It allows the approach of the glenoid rim at a right angle to the area of the capsulolabral detachment. The portal is made at the 5 o'clock position of the glenoid where the leading edge of the inferior glenohumeral ligament is and usually established by using the inside-out technique. The portal is just inferior to the subscapularis tendon, lateral to the conjoined tendon. The safety of the portal has been controversial due to its proximity to the axillary nerve/artery, musculocutaneous nerve, and cephalic vein [12, 17]. The axillary nerve and artery are placed within 15 mm of the portal, and the cephalic vein can be as close as 2 mm. When the patient is placed in the lateral decubitus position, lateralization of the portal placement is possible, leading to less risk of injury to the adjacent structures (Fig. 4.1).

4.3.3 7 O'Clock Portal

The 7 o'clock portal, also known as posteroinferior portal, is created to retrieve loose body and

for the fixation of the posteroinferior labrum [6]. In order to create the portal in the outside-in manner, a skin incision is made 2–3 cm inferior to the standard posterior portal with the portal positioned just above the posteroinferior corner of the glenoid and labrum. For the inside-out creation of the portal, the switching stick should be inserted through the anterior portal positioned at 7 o'clock and then pushed through the capsule. The structures at risk are the suprascapular nerve and artery, the axillary nerve, and the posterior circumflex humeral artery.

4.3.4 The Axillary Pouch Portal

The axillary pouch portal provides linear access to the entire inferior glenohumeral recess (IGHR) for arthroscopic instrumentation and visualization [2]. In order to make the axillary pouch portal, a spinal needle is introduced from a point 2–3 cm directly inferior to the lower border of the posterolateral acromion angle, approximately 2 cm lateral to the posterior viewing portal, angled approximately 30° medially in the axial plane. There are several advantages of the portal over the 7 o'clock portal and accessory posterior portals. The risk of damaging posterior neurovascular structures is lower than other portal positions due to its higher and lateral placement. Entrance above the posterior band of the inferior glenohumeral ligament prevents direct damage to its innervation or ligamentous fibers. Also, its lateral placement allows improved access to the IGHR and prevents overcrowding with medially placed posterior portal.

4.3.5 Trans-rotator Cuff Portal

The location of the portal varies with the location of the pathology for providing a suitable angle for the placement of the anchors in the posterosuperior glenoid. The port of Wilmington, described by Morgan et al. [13], is the most notable trans-rotator cuff portal introduced for SLAP repair. The portal is established 1 cm anterior and lateral to the posterolateral corner of the acromion. It allows approachment of the suture anchor in the angle of 45° to the posterosuperior glenoid surface. Even though the anteroposterior location may vary according to the surgeon's preference, it is recommended to be medial to the musculotendinous junction as lateral placement may injure the axillary nerve.

4.3.6 Accessory Posteromedial Portal

In case of a retracted tear of the posterior cuff involving the infraspinatus or teres minor, the accessory posteromedial portal is especially useful [8]. It enables an ideal position for suture passage by tendon-penetrating devices instead of using curved or angled suture relay devices, thus simplifying and accelerating the repair of the posterior portion of the rotator cuff tear. The entrance of the portal is made approximately 4–5 cm medial to the posterolateral corner of the acromion and 2 cm inferior to the scapular spine. Extended bursectomy of the medial and posterior subacromial space is recommended for the visualization of the entry point. However, too far medial bursectomy can injure the suprascapular artery.

4.3.7 Suprascapular Nerve Portal (Lafosse)

First introduced by Lafosse et al. [10], this specialized portal is established to cut the superior transverse ligament for the decompression of the suprascapular nerve. The entry of the portal is between the clavicle and the scapular spine, approximately 7 cm medial to the lateral border of the acromion. This is about 2 cm away from the Neviaser portal medially. Under the direct visualization through arthroscope (outside-in manner), a spinal needle is inserted through the trapezius muscle just above the medial aspect of the coracoclavicular ligament aiming toward the anterior border of the supraspinatus muscle. A blunt trocar is recommended to dissect the fatty tissues around the suprascapular nerve and artery and to further clarify the border of the transverse scapular ligament.

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Rotator Cuff Tear in Athletes: Part I. Pathophysiology

5

Robert Z. Tashjian

5.1 Introduction

Rotator cuff disorders are the most common cause of disability related to the shoulder [1]. More than 75,000 rotator cuff repairs are performed in the United States annually. Rotator cuff tearing affects as many as 30 % of individuals between 60 and 80 years of age and up to 50 % of patients older than the age of 80 [2, 3]. Despite its widespread prevalence, the exact etiology of rotator cuff disease is unknown although it is likely multifactorial including various intrinsic and extrinsic factors. Various factors recognized in the development of rotator cuff dysfunction include limited vascularity of the tendon, mechanical impingement on the undersurface of the acromion, and intrinsic degeneration. The etiology of rotator cuff injury in the athlete is similar to the nonathletic population, but there is also a role of tensile overload and internal impingement that is unique to this patient population, specifically the overhead thrower. The purpose of this chapter will be to review the various etiologic mechanisms of rotator cuff injury including genetic contributions, intrinsic factors and extrinsic factors, as well as tensile overload and internal impingement seen in the overhead-throwing athlete.

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5.2 Genetic Factors

Overall, there is only very preliminary evidence suggesting a genetic etiology of rotator cuff disease. Harvie et al. retrospectively evaluated the 129 siblings of a cohort of 205 patients diagnosed with full-thickness rotator cuff tears by ultrasound to identify the prevalence of rotator cuff tearing in this sibling population [4]. Using the 150 spouses of the patients as a control population, the relative risk of full-thickness tears in siblings versus control spouses was 2.42 (95 % CI 1.77–3.31), while the relative risk of symptomatic full-thickness tears in siblings versus control spouses was 4.65 (95 % CI 2.42–8.63). This significantly increased risk for tears in siblings may imply that genetic factors play a major role in the development of full-thickness rotator cuff tears. Drawbacks of Harvie's study included its retrospective nature, limitation to review of close relatives only, and the bias associated with a very high percentage of patients lost to follow-up.

Tashjian et al. investigated the familial predisposition for the development of rotator cuff disease utilizing a unique genealogic database linking patient information with family pedigrees [5]. The authors analyzed 3,091 individuals with a diagnosis or procedure code indicating either rotator cuff surgery or a rotator cuff tear and

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found an increased risk of rotator cuff surgery or tearing in the first- (RR = 2.4, p <0.0001) and second-degree (RR = 1.24, p = 0.018) relatives of individuals with rotator cuff disease compared to controls without evidence of rotator cuff injury. While these results are suggestive of a heritable contribution to the predisposition for rotator cuff injury, they also analyzed a subset of the population at higher risk for a genetic contribution (patients diagnosed before the age of 40; n = 652). In this high-risk population, they observed significantly elevated risks out to third-degree relatives (RR = 1.81, p = 0.0479) strongly supporting a heritable predisposition for rotator cuff tearing. Both the Harvie et al. and Tashjian et al. data are highly suggestive of a genetic predisposition for the development of rotator cuff disease.

5.3 Intrinsic Factors

5.3.1 Hypoperfusion

The region 1–2 cm medial to the insertion of the supraspinatus tendon onto the greater tuberosity has been described as hypovascular [6]. Hypoperfusion in this region has been considered a possible factor in initiation of rotator cuff injury. Despite this presumption, other literature supports that this region is unlikely hypoperfused but rather is a region of vascular anastomosis [7, 8]. Nevertheless, generalized disease leading to global hypoperfusion of tissue may still be a risk factor for the development of rotator cuff tearing which has been shown with smoking, cardiovascular disease, and lung disease [9, 10]. Also, vascularity of the cuff has been shown to decrease with age corresponding to a potential mechanism for age-related rotator cuff degeneration [11].

5.3.2 Age-Related Degeneration

The incidence of rotator cuff tearing increases with age. This has been documented by several authors and supports age-related degeneration as a possible etiology of rotator cuff tearing. Several investigators have evaluated shoulders in asymptomatic individuals with MRI and ultrasound in an attempt to determine the likelihood of rotator cuff tears. Sher et al. found the overall prevalence of tears in asymptomatic individuals to be 34 % by MRI: 15 % full thickness and 20 % partial thickness [3]. In patients older than 60 years of age, full-thickness and partial-thickness tears were found in 28 and 26 % of individuals, respectively. In patients between 40 and 60 years of age, full-thickness and partial-thickness tears were found in 4 and 24 % of individuals, respectively. Finally, no individuals less than 40 years old had full-thickness tears, and 4 % had partial-thickness tears. Tempelhof et al. found an overall prevalence of full-thickness rotator cuff tears in asymptomatic individuals utilizing ultrasound to be 23 %, with 51 % of individuals over the age of 80 having tears [12]. These studies support the theory that rotator cuff tearing occurs, to some extent, as a "normal" degenerative process which increases with aging.

5.3.3 Apoptosis

Several authors have evaluated the role of apoptosis in the development of rotator cuff tearing. Yuan et al. were the first to report the presence of increased apoptosis in torn supraspinatus tendons compared to subscapularis tendons from controls using immunohistochemistry [13]. The percentage of apoptotic cells in the degenerative rotator cuff was significantly higher than controls (34 % vs. 13 %). Since this initial study, numerous investigators have evaluated apoptosis as a potential initiator for rotator cuff tearing. Potential genes associated with increased apoptosis include upregulation of p53 and HIF1alpha [14, 15]. Potential triggers for apoptosis include hypoxic injury. This injury may occur as perfusion of the cuff decreases with aging suggesting a potential mechanism for age-related cuff degeneration.

5.4 Subacromial Impingement

Subacromial impingement of rotator cuff was originally described by Neer and is considered the primary method of extrinsic cuff

degeneration [16]. Neer hypothesized that impingement of the cuff on the undersurface of the acromion was the primary pathologic factor leading to rotator cuff tearing. Initial studies supporting this theory suggested that a more "hooked" acromial shape was associated with a higher incidence of rotator cuff tearing [17]. Recently, clinical studies have shown poor reliability of determining acromial shape and therefore implicating acromial shape as a causative factor for rotator cuff tearing has limited basis [18]. On the other hand, acromial spur formation, which has been shown in cadaver studies to be correlated with aging, can reliably be identified on radiographs and has been shown to correlate with rotator cuff tearing [18, 19]. Consequently, the development of acromial spur formation and the development of rotator cuff tearing are likely coincident but not causative as both are age related. Soslowsky supported this theory further by showing that extrinsic compression alone is insufficient to cause rotator cuff tendinopathy in an animal model [20]. While extrinsic compression likely plays a role in cuff tearing, especially with bursal-sided injuries, it is unlikely to be the primary source of injury but rather one of many causative factors leading to cuff insufficiency. Subacromial impingement can occur in the overhead athlete. Impingement is either due to weakness and dysfunction of the rotator cuff itself or the periscapular musculature. Overhead athletes require maximum abduction and external rotation in order to throw and this repetitive action requires the rotator cuff to pass underneath the coracoacromial arch. Weakness of the rotator cuff can accelerate subacromial impingement through humeral head elevation. With a loss of the normal force couple generated by the cuff, there is decreased resistance to the strong superior pull of the deltoid leading to subacromial impingement.

Scapula dysfunction is the other mechanism of subacromial impingement in the athlete. The SICK scapula syndrome (scapular malposition, inferior medial border prominence, coracoid pain and malposition, and dyskinesis of scapular movement) has been described by Burkhart et al. and is a potential source of rotator cuff injury and impingement [21]. The syndrome presents with a "dropped" scapula statically on examination and is a result of protraction, lateral displacement, and abduction of the scapula. The type III pattern associated with a prominent superior medial border of the scapula is most commonly associated with impingement. As a result of the syndrome, the acromion remains malpositioned with a protracted scapula throughout throwing leading to impingement. Scapula rotation is required to allow the tuberosity to clear under the acromion, and failure to accomplish this due to dyskinesis during throwing will lead to cuff injury. Tightness of the pectoralis minor and weakness and inflexibility of the periscapular muscles and posterior rotator cuff are typically the causative factors. Once dyskinesis has developed with periscapular weakness, poor throwing mechanics typically results with dropping of the elbow during late cocking and early acceleration phases and angulation of the arm posterior to the scapular plane rather than in the plane of the scapula resulting in a further decrease in scapular rotation and elevation worsening the impingement [21]. Correction of both throwing mechanics and rotator cuff and periscapular weakness is required to treat the impingement.

5.5 Tensile Overload

Partial-thickness or intrasubstance rotator cuff tears are very common and can occur in up to 1/3 of throwers diagnosed with superior labral tears. Large eccentric cuff contractions as well as increased tensile loads are considered initiators of rotator cuff injury in the overhead athlete. Large eccentric tensile forces are created in the process of stabilizing the humeral head during the deceleration phase of throwing to slow the arm down [22]. If the forces are not adequate to resist this deceleration force, then abnormal tensile forces within the cuff are generated and can lead to overload and failure [23]. The combination of repetitive eccentric contractions and tensile overload places the overhead athlete at an increased risk for rotator cuff injury.

5.6 Internal Impingement

Unique to the overhead athlete is the pathologic condition of internal impingement characterized by repetitive contact of the greater tuberosity of the humeral head with the posterosuperior glenoid with the arm in the abducted externally rotated position. The condition is a result of rotator cuff and labral impingement by the humerus and glenoid, leading to undersurface rotator cuff tears and posterosuperior labral tears (Fig. 5.1a–c). It has traditionally been described in throwing or overhead athletes who perform repetitive overhead motion with their arm positioned in the abducted and externally rotated position. While controversy exists regarding if this is a normal or pathologic phenomena, repetitive internal impingement can lead to structural damage and clinical impairment in the athletic shoulder.

Walch et al. originally described impingement of the deep surface of the posterior supraspinatus and the anterior infraspinatus on the posterosuperior glenoid in 17 patients undergoing shoulder arthroscopy [24] (Fig. 5.2). Findings of the arthroscopy included articular surface cuff tears and posterior labral tears without any other intraarticular

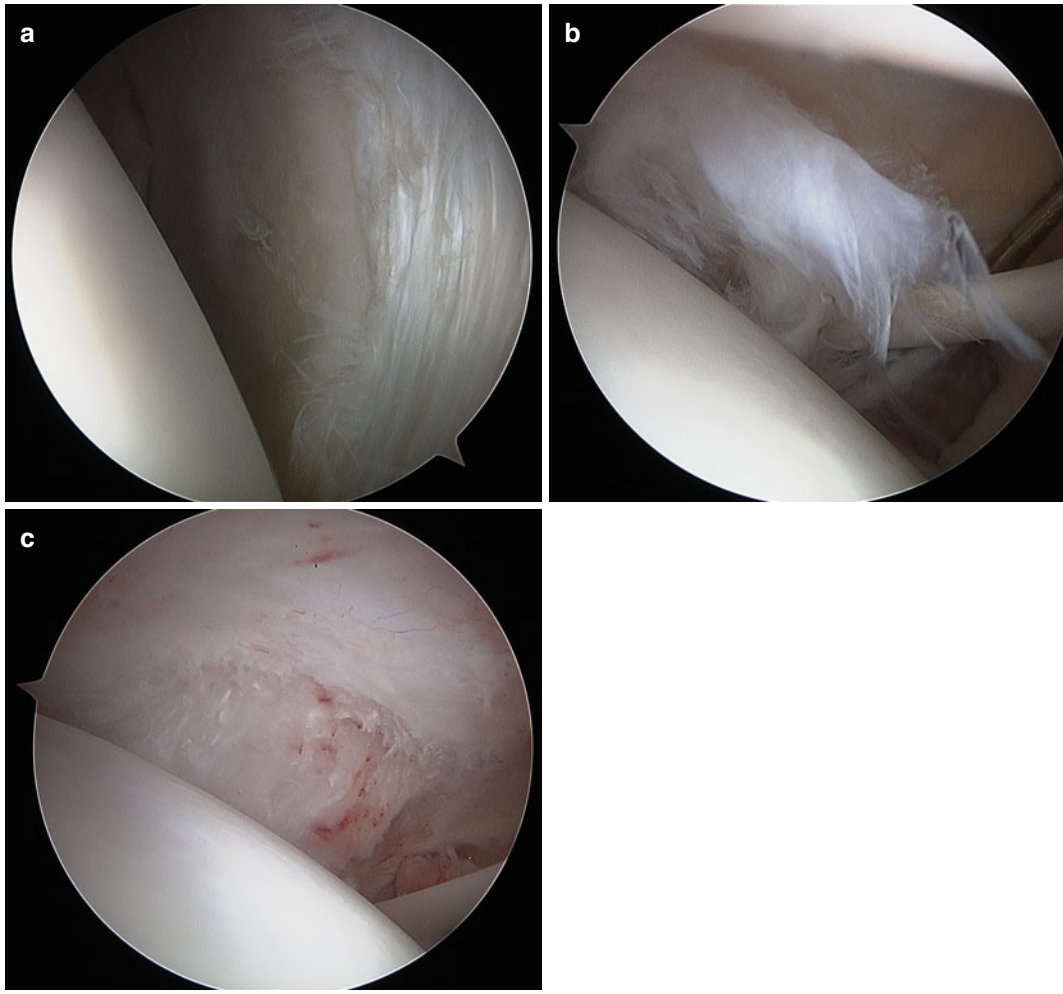

Fig. 5.1 Arthroscopic images of a posterosuperior labral tear (**a**) and an undersurface partial-thickness rotator cuff tear of posterior supraspinatus before (**b**) and after (**c**) arthroscopic debridement in an overhead athlete with internal impingement (Courtesy of Brian Wolf, MD)

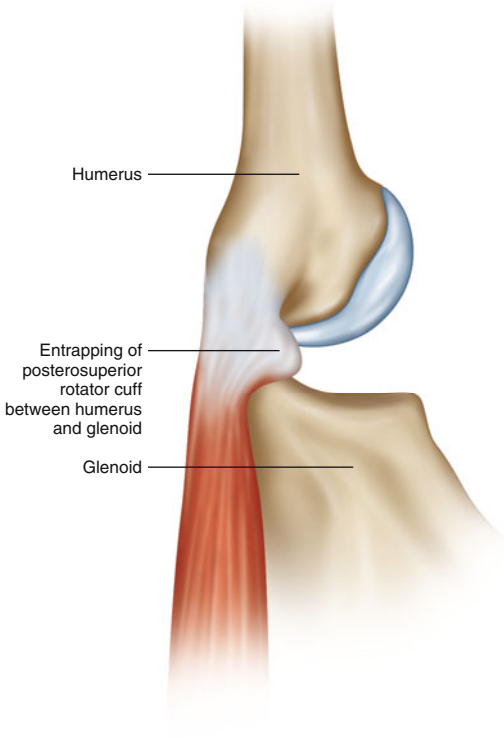

Fig. 5.2 Internal impingement of the posterior rotator cuff between the humerus and glenoid during maximal abduction and external rotation

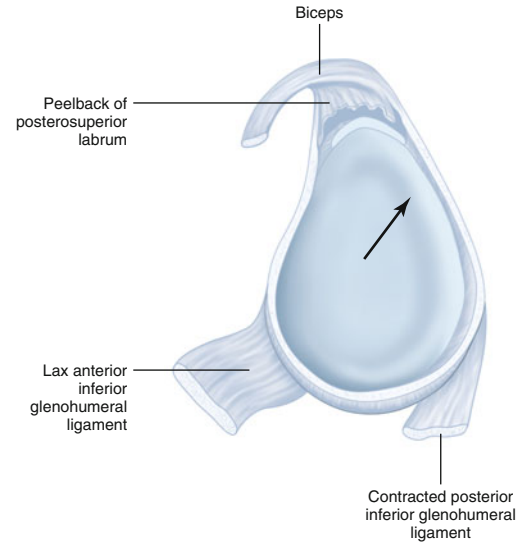

Fig. 5.3 The shift in position of the glenohumeral joint center of rotation (depicted by the *arrow*) in a posterior and superior direction is initiated by contracture of the posteroinferior capsule leading to redundancy of the anteroinferior capsule and ligaments resulting in increased sheer forces at the biceps anchor and superior labrum resulting in a superior labral tear

pathology. Walch hypothesized that this phenomena occurs in all shoulders in the abducted, externally rotated position but may become pathologic in throwers because of the repetitive insult. Jobe theorized that anterior capsular laxity was the main factor in the development of internal impingement, therefore recommending anterior capsular plication as a treatment [25]. Current data actually supports the opposite premise that anterior laxity with anterior glenohumeral subluxation likely lessens internal impingement instead of aggravating it [26].

Most recently, Burkhart et al. has proposed that posterior capsular contracture and a loss of internal rotation are the main factors in the development of internal impingement [27]. Burkhart et al. hypothesized that the posterior capsule becomes injured and thickened due to repetitive tensile forces created during the deceleration phase of throwing [27]. This posterior capsule contraction leads to shifting the center of rotation of the shoulder more posteriorly and superiorly leading to posterosuperior instability with the shoulder abducted and externally rotated. The posterosuperior shift allows humeral hyperexternal rotation which places shear stress on the posterosuperior labral and torsional stress on the posterior cuff leading to undersurface posterior supraspinatus and infraspinatus tear as well as superior labral tears [27] (Fig. 5.3). With a posterosuperior shift of the glenohumeral center of rotation, the space-occupying effect of the proximal humerus on the anteroinferior capsule and ligaments is reduced, leading to redundancy. Prior theories supporting anterior instability as the source of internal impingement were likely a misinterpretation of the redundancy of the anterior inferior glenohumeral ligaments as microinstability. Treatment of pathologic internal impingement concentrates on posterior capsular stretching to reduce the internal rotation deficit as well as strengthening of the posterior cuff to aid in resisting the distracting forces during deceleration and follow-through phases.

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Rotator Cuff Tears in Athletes: Part II. Conservative Management – American Mind

6

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6.1 Introduction

Rotator cuff tears are common injuries in athletes and may occur as a result of acute trauma (such as a fall onto an outstretched arm) or, more commonly, as a result of chronic overuse with repetitive overhead activity. While traumatic conditions are generally treated surgically, chronic overuse injuries are more often treated using a conservative approach.

Overuse injuries of the shoulder are commonly related to microtrauma due to repetitive overhead activities, especially in throwing sports such as baseball or javelin. As these athletes progress to higher levels of competition, greater demands are placed on the glenohumeral joint with increased throwing velocities. Throwing velocity is maximized by increases in shoulder abduction and external rotation range of motion, which leads to anatomic bony and soft tissue adaptations that facilitate hyperabduction and external rotation over time. These anatomic changes can lead to contracture of the posterior capsule with subsequent posterosuperior humeral head migration [1]. This is known as glenohumeral internal rotation deficit (GIRD), which in addition to restricted internal rotation can lead to tearing of the superior labrum (SLAP) via the "peel-back" mechanism [1]. GIRD may also lead to posterosuperior glenoid impingement – an entity characterized by posterosuperior rotator cuff and/or labral tearing as a result of osseous impingement between the greater tuberosity and the glenoid rim in positions of abduction and external rotation (internal impingement). Additionally, muscle imbalances can produce scapular dyskinesis, which may decrease the space available for the rotator cuff tendons to pass beneath the acromion, thus leading to fraying and partial-thickness tearing. The majority of throwing athletes have articular-sided, partial-thickness rotator cuff tears, most of which occur near the interval between the supraspinatus and infraspinatus tendons, presumably due to internal impingement [1].

Athletes who undergo surgery in the midst of a season are commonly excluded from play for the majority of the season. Even off-season

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surgical treatment may limit return to play or a return to the preinjury level. For efficient and successful treatment with physical therapy, it is important to keep in mind some basic considerations and to follow the basic principles of rehabilitation described below.

Although the pathomechanisms surrounding rotator cuff tears in overhead athletes are still heavily debated, conservative management remains a mainstay of treatment [2]. Therefore, the purpose of this chapter is to review the basic principles of conservative management for rotator cuff tears in athletes with a focus on the overhead athlete.

6.2 Basic Considerations

In the overhead athlete with a rotator cuff tear, the primary goal of nonsurgical management is to achieve a return to full competitive sport while also preventing further injury. This is underscored by specific rehabilitation goals such as decreasing pain and inflammation, strengthening surrounding musculature to promote proper joint kinematics, and promoting proper throwing mechanics that maintain a normal scapulohumeral angle. It is unlikely that conservative treatment induces healing of a torn rotator cuff. However, when overhead athletes present with pain as their major symptom, they can often be treated nonoperative with the goal to improve their range of motion and return them to prior competitive levels.

6.3 Basic Principles: Phases of Rehabilitation

6.3.1 Phase 1

In the overhead athlete with a rotator cuff tear not qualifying for initial surgery, phase 1 of rehabilitation should focus primarily on methods to decrease pain and inflammation, which facilitates range of motion while decreasing pain arthrogenic inhibition, so that more advanced exercises may be implemented. In addition to rest, activity modification, cryotherapy, and anti-inflammatory medications, there are several other therapeutic options that may help to decrease pain and inflammation associated with rotator cuff tears. These may include transcutaneous electrical nerve stimulation (TENS), massage therapy, and laser and heat therapy. However, there is a paucity of evidence to support their use specific to shoulder injuries in the overhead athlete. Nevertheless, since these modalities are typically inexpensive and present minimal risk to the patient, subjective and objective improvements as a result of these interventions may warrant their use.

Subacromial and/or intra-articular injections can also be used to decrease pain and inflammation in patients with rotator cuff tears. Injections may include hyaluronic acid [3], corticosteroids [4, 5], platelet-rich plasma [6], or local anesthetic preparations [7]. While local anesthetics and corticosteroids have been shown to be effective at decreasing pain and inflammation in those with rotator cuff tears, the efficacies of hyaluronic acid and platelet-rich plasma injections are still debated.

In addition to decreasing pain and inflammation, it is critical to ensure that throwing athletes maintain appropriate glenohumeral range of motion. Although the throwing shoulder is often found to have increased external rotation and decreased internal rotation, the total arc of motion should be almost equal to that of the non-throwing shoulder [8]. When physical examination reveals a loss of internal rotation with an associated decrease in the total arc of motion (i.e., glenohumeral internal rotation deficit [GIRD]), specific stretching exercises should be implemented to relieve contractures of the posterior structures, pectoralis minor, and short head of the biceps tendon [8, 9]. Cross-body stretching, the sleeper stretch, and the unilateral corner stretch have been found to significantly increase internal rotation capacity in overhead athletes with GIRD [9, 10]. If an athlete returns to throwing activities before achieving their normal arc of motion, symptoms may recur even after completion of a full rehabilitation program [11]. Therefore, active and passive glenohumeral range of motion should be maintained and emphasized throughout all phases of the rehabilitation process to ensure a successful return to throwing sports.

6.3.2 Phase 2

The athlete may progress to the second phase of rehabilitation following the relief of pain and inflammation. In phase 2, strengthening of surrounding musculature (including that of the hand, wrist, and elbow) is initiated while maintaining pain-free active and passive range of motion. Although the specific strengthening program should be individualized according to the needs of each patient, some have shown that specific emphasis on scapular retractors and glenohumeral external rotators may be beneficial during rehabilitation of the overhead athlete [12].

The concepts of neuromuscular control and dynamic stability center around the coordination of agonist/antagonist muscle groups which work together to produce force couples that center the humeral head within the glenoid fossa at all levels of humeral elevation and rotation. Techniques that focus on neuromuscular control, such as plyometrics, perturbation training, proprioceptive neuromuscular facilitation (PNF) exercises, and closed kinetic chain exercises, should be implemented into the rehabilitation program of any overhead athlete to prevent future injury [13].

6.3.3 Phase 3

Progression to phase 3 requires that the athlete demonstrates optimal upper extremity strength, normalization of range of motion deficits, advanced neuromuscular control, a lack of symptoms, and a lack of significant physical examination findings. In phase 3, intensive upper and lower extremity strength and endurance training is initiated along with an introduction to plyometric exercises that are designed to optimize neuromuscular control. Furthermore, core stability has to be strengthened to ensure proper throwing motion to generate efficient forces within the shoulder joint motion, counteracting distractive and compressive work in a synchronous and coordinated fashion. Any mismatch occurring in this kinematic chain may lead to pathological shear stress in the shoulder joint and to injuries [11]. Therefore, to successfully treat any rotator cuff tear with physical therapy, potentially underlying deficits in the athlete's throwing mechanics have to be detected and incorporated. The athlete should be taught to work on a balanced distribution of training exercise for the agonist and antagonist muscles of the upper and lower extremities and the trunk to optimize core stability [14, 15]. To prepare for phase 4, light endurance-like roadwork and cycling as well as throwing activities should be started at this point to help transition the athlete back into overhead activity.

6.3.4 Phase 4

During phase 4, the athlete is gradually returned to sport. A structured interval throwing program should be implemented to ensure a graduate progress because there is an elevated risk of rotator cuff re-injury within this stage [11]. Criteria for return to play should be adequate strength tested with handheld dynamometry [16, 17]; achievement of sufficient thresholds in functional outcome scores, such as the Kerlan-Jobe Orthopaedic Clinic (KJOC) questionnaire [18]; controlled pain; and appropriate ROM and scapulohumeral rhythm before an athlete may be considered "cleared" for full activities. Furthermore, increases in pain during overhead activity should be addressed with rest and activity modification. When indicated, pain-free stretching should be continued after rehabilitation to prevent loss of internal rotation and recurrent symptoms [8]. However, the strength training program should be altered gradually to avoid an overuse injury [19].

6.4 Conservative Treatment's Elements

6.4.1 Passive and Active Range of Motion Exercises

Stretching and strengthening exercises should be conducted under supervision of a professional physical therapist. A specific rehabilitation program should be tailored to the individual athlete

as well as the specific type of tear to eliminate pain, to maintain and maximize range of motion, and to prevent adhesions due to healing processes [20]. Shoulder-specific exercises should involve the rotator cuff muscles, periscapular stabilizers, and the deltoid muscle. Furthermore, depending on the result of the evaluation of throwing mechanics, specific exercises for the lower body as well as for the trunk should be included (body core stability).

Exercises for the rotator cuff and the deltoid include range of motion exercises, proprioceptive exercises, and strengthening exercises (Chap. 33, Sect. 3.1.1).

6.4.2 Selective Stretching

To prevent anterior and posterior capsular tightness, certain stretching exercises are advisable. Posterior shoulder tightness is frequently seen in combination with rotator cuff tears in overhead athletes and is one of the most common causes for shoulder pain [21]. Posterior shoulder tightness can be treated by special exercises. These include internal/external rotation stretch and the "sleeper stretch" (Chap. 33, Sect. 3.1.1).

6.4.3 Core Stability

Underlying scapular dyskinesis should be addressed (see Sect. 6.4.1) to integrate scapulothoracic muscles into any sport-specific motion [22]. Exercises for the trunk and the lower extremity in order to improve and maintain "core stability" are important. This can also be achieved by strengthening and proprioceptive exercises described in Chap. 33.

In addition, exercises for special throwing movements of the athlete should be taught, if applicable (Chap. 33).

6.4.4 Injections

Subacromial and intra-articular injections may be used to decrease inflammation and rapidly assist in the rehabilitation and recovery process [20]. Under sterile conditions, steroids, local anesthetics, or hyaluronic acid can be injected into the glenohumeral joint or the subacromial space, depending on the location of the tear. Usually, a corticosteroid is used in combination with a local anesthetic. Corticosteroids can decrease the inflammation; however, collagen necrosis limits their usage. Even in a young athlete's shoulder, corticosteroids should not be injected more than once every 3 months and, in our opinion, not more than three times in total. Injection of hyaluronic acid may be beneficial in some cases. As a component of the natural synovial fluid, it may help to preserve joint friction at a physiological level [23].

However, for injection of any substances in athletes, particular caution has to be obtained to meet the anti-doping regulations. For most of the national anti-doping agencies, intra-articular injections of steroids and other substances are allowed for many specific reasonable indications. Nevertheless, every indication and every forbidden substance have to be justified and notified to the National Anti-Doping Agency prior to treatment.

6.4.5 Medications

As with systemic medications, common NSAIDs should be used [24]. To obviate gastrointestinal reactions and side effects of NSAIDs, a proton pump inhibitor should be administered in addition. Due to an increased risk of increase of vascular events and myocardial infarction, we do not recommend the use of COX2-inhibitors. In cases of allergic reactions to NSAIDs, paracetamol is an adequate alternative.

6.4.6 Cryotherapy

Cryotherapy is effective for initial short-term pain relief. It may diminish the release of blood and proteins from the surrounding vasculature by reducing tissue metabolism. Ice may be effective for reducing swelling and pain in cases of acute inflammatory tendinopathies by blunting the

inflammatory response [25]. Applications of ice through a wet towel for 10-minute periods seem to be most effective [25].

6.4.7 Duration and Frequency of the Physical Therapy

Conservative treatment is targeted to progressively bring athletes back to their preinjury level and to elite competition as quick as possible.

In our experience, conservative treatment that outlined is a reasonable option for the individual athlete, with duration to not exceed 12 weeks without documented improvement. Furthermore, the athlete should have entered phase 4 of the rehabilitation and conceivably be ready to return to their prior level of competitive sports after 6 months. If these thresholds cannot be met, the athlete will most likely not improve from further conservative treatment and may benefit surgery.

6.5 Conclusion

6.5.1 Principles of Injury Prevention

For injury prevention, athletic cross-training in different recreational activities (as opposed to only working on the throwing motion) exposes the body to various movements and forces with the goal to maximize overall physical and mental well-being of the athlete [11]. Some important principles should be followed to maintain throwing motion capability and to prevent acute injuries in the overhead athlete. The act of throwing in the dominant arm may lead to a reduction of internal rotation, resulting in a higher risk of injury [26]. To decrease this risk, it is necessary to maintain the full range of motion throughout the year. This may be achieved by a specific stretching program. Furthermore, to maintain the proper throwing mechanics, core stability should be emphasized as described (phase 3). Both glenohumeral and scapulothoracic articulation need to be controlled by the athlete neuromuscularly for proper kinematic movements at the elite level (also see Sect. 6.3.2) [13]. Within the off-season, training programs should include rest and exercises specific to the sport in question and position played. A program may include strength, power, range of motion, and/or endurance exercises.

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Rotator Cuff Tear in Athletes: Part II. Conservative Management – European Mindset

7

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7.1 Introduction

Shoulders in athletes, especially throwing athletes, undergo high levels of stress and are therefore more likely to experience rotator cuff tears than other populations. Even though full rotator cuff tears are not common, partial tears are increasing in incidence.

In a study conducted by Jost et al. [29] that imaged the throwing shoulder of professional handball players using magnetic resonance imaging (MRI), they found that 83 % of players had tendinopathies or partial tears of the supraspinatus tendon. However, only 44 % of these players were symptomatic. Symptoms correlated poorly with the abnormalities seen on MRI. Follow-up investigation, on average 6.8 years later, found that all the players that had resigned from playing handball on a professional level did not play anymore due to causes unrelated to the documented shoulder injuries. At follow-up, none of these players reported surgical treatment or showed progression of the rotator cuff pathology. They also had significantly better Constant-Murley Scores [39]. It is therefore reasonable to assume that rotator cuff tears found on MRI are not an indication for surgical rotator cuff repair. Thus, in most cases, nonoperative management remains the mainstay for most athletes as a first approach with arthroscopic debridement and repair when necessary if refractory symptoms are present.

The goal of this chapter is to discuss the conservative management options after rotator cuff tears in athletes and look at prevention options.

7.2 Conservative Treatment Options

Rotator cuff tears have a high prevalence in athletes. However, most of these athletes are not symptomatic [29]. Partial rotator cuff tears are much more common than full-thickness tears in young otherwise healthy athletes [29]. Treatment modalities for these tears can involve (1) initial operative treatment, (2) conservative treatment followed by delayed operative treatment, or (3) conservative treatment only. Good patient selection is crucial for successful conservative treatment of rotator cuff tears. While surgical treatment is initially indicated for athletes with subscapularis tendon tears and all symptomatic full-thickness tears, athletes with partial and asymptomatic full-thickness supraspinatus or infraspinatus tears can initially be treated conservatively. Conservative treatment is usually undertaken for up to 6 months.

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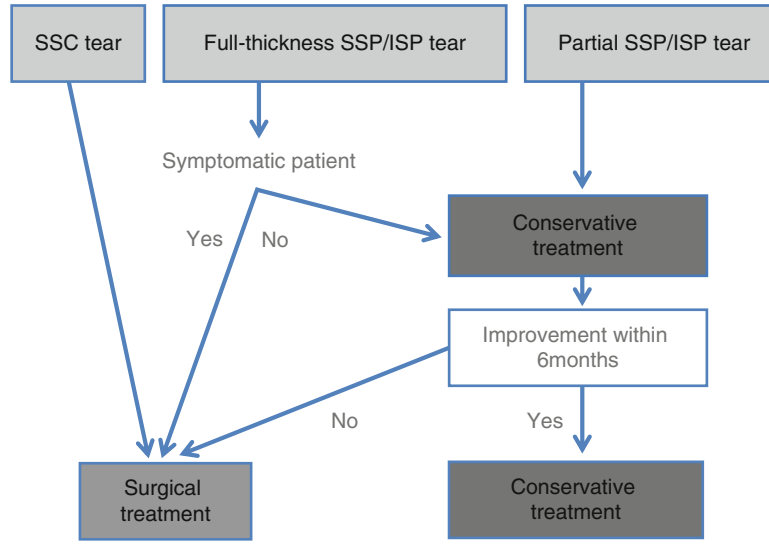

Fig. 7.1 Algorithm for rotator cuff tear treatment in athletes (*SSP* supraspinatus tendon, *ISP* infraspinatus tendon, *SSC* subscapularis tendon)

There is evidence which shows that rotator cuff tear patients with pain lasting longer than 6 months do not respond well to ongoing nonoperative treatment [3]. Therefore, if symptoms persist, patients may be reevaluated for surgery (Fig. 7.1).

As with many orthopedic problems, an abundance of different therapies exist for the conservative treatment of rotator cuff problems. The benefit of some of these treatment options is controversial. It is therefore not surprising that there is little consensus among orthopedic surgeons on what should be the gold standard for the conservative treatment of rotator cuff tears.

The following conservative treatment options will be discussed:

1. Physical therapy
2. Modification of activities/change of position
3. Administration of corticosteroids
4. NSAIDs
5. Extracorporal shock wave therapy
6. Iontophoresis
7. Ultrasound
8. Transcutaneous electrical nerve stimulation (TENS) and pulsed electromagnetic field (PEMF)
9. Acupuncture
10. Manual therapy

Even though there are an abundance of studies investigating the impact of these different treatment options, we did not find level I studies that give reliable and strong evidence. Most of the studies only show a tendency with a moderate level of evidence. This underlines the need for further double-blinded randomized level I studies that investigate the benefit of different conservative treatment options.

7.2.1 Physical Therapy

Today, physical therapy is one of the most important components of conservative treatment despite limited evidence found in the literature about its efficacy. In a multicenter study, Kuhn et al. [33] investigated 452 patients with atraumatic full-thickness rotator cuff tears. These patients were treated with a conservative regimen receiving physical therapy with or without an intra-articular corticosteroid injection depending on the severity of the symptoms. At the final follow-up by telephone after 24 months, 30 % of the patients had elected to undergo surgery, most of the time within 6–12 weeks after diagnosis of the injury. Conservative treatment was effective in approximately 70 % of the patients. However, this study may not be suitable for athletes, especially overhead-throwing athletes, since pain reduction is not the most important factor but rather improvement of strength [33]. Several studies have shown that

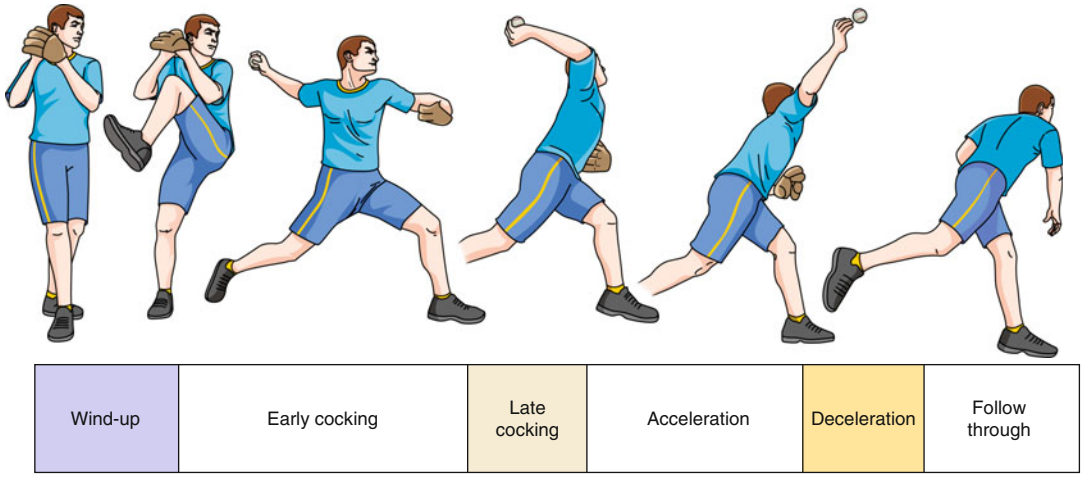

Fig. 7.2 Phases of the baseball pitch

patients with clinical signs of rotator cuff tendinopathy also demonstrate kinematic alterations consistent with scapular dyskinesis [31, 41]. The importance of treating scapular dyskinesis during physical therapy sessions will be discussed later in this chapter.

7.2.2 Modification of Activities/ Change of Position

Throwing motions should be evaluated and if necessary corrected particularly in overhead-throwing athletes. For this reason, it is important to know its phases and the main muscles involved in each phase (Fig. 7.2).

The supraspinatus muscle plays an important role in the late cocking phase. This muscle causes humeral abduction and contributes to the stability of the joint by drawing the humeral head towards the glenoid [22]. During the acceleration phase in unstable shoulders, the supraspinatus tendon is active as well and may help to stabilize the joint in these athletes [22].

The infraspinatus together with the teres minor muscle is responsible for external rotation of the shoulder during the late cocking and follow-through phase [22].

The subscapularis muscle acts as an internal rotator in the acceleration and follow-through phase, helping to carry the arm across the chest. While professional overhead players have the capacity to activate every rotator cuff muscle individually, the amateur players are not able to use every muscle exclusively [22]. This may lead to faster fatigue, eventually overuse and injury. When a rotator cuff tear is diagnosed, part of the training focus should be on the phase in which the damaged tendon is activated.

Although a change of position or change of sport might eliminate the cause of the tear and provide lasting relief, it is not desirable especially for high-level athletes and is therefore not feasible.

7.2.3 Corticosteroids

7.2.3.1 Local Corticosteroids

Corticosteroids are potent pain modulating and anti-inflammatory drugs.

In certain situations, subacromial and intra-articular cortisone injections can be helpful to restore pain-free shoulder function. It has been shown that partial-thickness articular surface rotator cuff tears are at least twice as common as bursal-sided tears [19, 44]. While in patients with articular surface rotator cuff tears an intra-articular corticosteroid injection may improve symptoms, in patients with bursal-sided tears, a subacromial injection is desirable. This may allow the patient to start physical therapy. One

clinical study [12] investigated the outcome of 49 patients with rotator cuff disease that were treated with subacromial corticosteroid injections, NSAIDs, and physical therapy. After 1 year, 40 % of the patients failed conservative treatment and had to be treated surgically. The authors concluded that it is therefore difficult to predict outcomes after this conservative treatment [12].

In addition to the beneficial effects, corticosteroids may have adverse effects such as infections and tendon atrophy. Tendon quality decreases with an increasing number of injections. Speed et al. therefore recommend a maximum of three injections per shoulder with a minimum interval of 6 weeks between each injection [40].

Furthermore, there is evidence that blind injection techniques are reported to reach the desired space (e.g., subacromial space) in only 30–80 % of cases [26].

Both Chen et al. [9] and Naredo et al. [37] reported less pain and improved shoulder function when comparing ultrasound-guided subacromial injections with blind injections.

7.2.3.2 Systemic Corticosteroids

Systemic corticosteroids have shown to be as effective as local corticosteroids applied to the shoulder. Ekeberg et al. [18] compared the short-term effectiveness of ultrasound-guided corticosteroid injections into the subacromial bursa and systemic corticosteroid injections. The results of this study do not indicate that systemic corticosteroid injections are less effective than local injections. However, systemic corticosteroids may have more systemic side effects.

When given systemic corticosteroids, it was observed that there was not only a reduction of inflammation, but there was also an associated positive impact on the muscular changes that occur after a rotator cuff tear, namely, fatty infiltration, atrophy, and retraction. In a sheep rotator cuff tear model, the sheep that received local and systemic nandrolone decanoate (group I, $n=6$) and the sheep that received local steroid injections (group II, $n=7$) showed less retraction when compared to the untreated group (group III, $n=7$). Furthermore, group II showed less muscle atrophy of the supraspinatus muscle compared to the control group [21].

7.2.4 Nonsteroidal Anti-inflammatory Drugs

Nonsteroidal anti-inflammatory drugs (NSAIDs) are commonly used for the management of pain and reduction of swelling in orthopedic patients. Despite their positive anti-inflammatory and pain-relieving effects, NSAIDs also have been reported to have a negative effect on rotator cuff tendon-to-bone healing in the animal model. In a study by Cohen et al. [11], Sprague–Dawley rats underwent acute rat rotator cuff reconstructions. Postoperatively, these rats either received a cyclooxygenase-2-specific NSAID ($n=60$) or a nonselective NSAID ($n=60$). The rats in the control group received no treatment ($n=60$). The cyclooxygenase-2-specific as well as the nonselective NSAID group showed significantly decreased ultimate load to failure modes when compared to the control group at 2, 4, and 8 weeks. No differences were seen between the two NSAIDs used in this study suggesting that this inhibition of tendon-to-bone healing may be caused by cyclooxygenase-2 [11].

Similar results were reported in a smaller study where rats receiving daily intraperitoneal NSAID injections starting from postoperative day 11 after a rotator cuff repair displayed a significantly lower mean maximal load to failure at 21 days postoperatively when compared to the control group which received only saline injections and the group that received injections from postoperative day 1 to 11 [8]. No differences were found with respect to cellularity, vascularity, and collagen orientation though [8].

In a meta-analysis, Boudreault et al. [4] showed that even though only moderate grade evidence exists regarding the efficacy of NSAIDs for rotator cuff tendinopathy, these drugs are effective in reducing short-term pain but not in improving function. Furthermore, NSAIDs are as effective as corticosteroid injections for pain relief [4].

7.2.5 Extracorporeal Shock Wave Therapy

Extracorporeal shock wave therapy has been proposed as an alternative treatment for surgical rotator cuff reconstruction. Harniman et al. [27] concluded in a systematic review that there is moderate evidence showing no effect of low-energy ESWT (<0.2 mJ/mm^2) in chronic non-calcific rotator cuff tendonitis and moderate evidence in support of high-energy ESWT (generally 0.2–0.4 mJ/mm^2) for chronic calcific rotator cuff tendonitis. Similar results were published by Huisstede et al. in 2011 [28]. Due to the fact that significant methodological weaknesses were found in the two randomized controlled trials, further well-designed studies will be necessary to prove the benefit of extracorporeal shock wave therapy.

7.2.6 Iontophoresis

Iontophoresis is a process in which ions flow driven by an electric field. These molecules (e.g., drugs) have to have an electric charge or need to be linked to a charged molecule. The benefits of iontophoresis are controversial as a treatment for different musculoskeletal disorders in both in vitro and in vivo studies.

However, there are no studies investigating the impact of iontophoresis on rotator cuff pathologies. Thus, it is not possible to draw any conclusions on the efficacy of iontophoresis in the treatment of rotator cuff disease.

7.2.7 Ultrasound

There is insufficient evidence to merit the use of ultrasound therapy in shoulder pathologies. Several authors have reported improving pain and quality of life in patients that were treated with ultrasound [1, 17, 36]; others have reported no differences in outcome [16, 35, 38, 42]. One big problem in ultrasound treatment is that there is no accepted standard method with respect to the frequency and treatment intensity. In the above-cited studies, the treatment intensity, frequency, and duration were not the same, making a comparison of the different studies difficult.

7.2.8 Transcutaneous Electrical Nerve Stimulation (TENS) and Pulsed Electromagnetic Field (PEMF)

There is little literature that investigates the impact of transcutaneous electrical nerve stimulation (TENS) and pulsed electromagnetic field (PEMF) on rotator cuff pathologies.

Eyigor et al. [20] were able to show that intra-articular corticosteroid injections as well as TENS are efficient in the treatment of rotator cuff tendinitis. When compared, corticosteroid injections were slightly more efficacious than TENS with regard to pain relief, ROM, and disability in the first weeks [20].

PEMF has been shown to improve the short-term clinical outcomes in patients with therapy refractory rotator cuff tendinitis over a 4-week treatment period [2]. To our knowledge no other studies investigate the impact of PEMF on rotator cuff tears.

7.2.9 Acupuncture

Especially in the treatment of pain, acupuncture has gained increasing attention. Unfortunately, there is lack of well carried out clinical studies in the literature that investigate the benefit of acupuncture for rotator cuff healing. The studies that exist show controversial results.

In a randomized clinical trial, Kleinhenz et al. [32] reported that acupuncture with penetration of the skin was more effective in improving the Constant–Murley Score than when performing placebo needling in patients with rotator cuff tendinopathy [32]. In a meta-analysis published in the Cochrane Library, the authors found no significant difference in short-term improvement associated with acupuncture when compared to placebo. This may be explained by type II error due to a rather small sample size of the two included trials [24].

7.2.10 Manual Therapy

There is only one randomized clinical study investigating the impact of manual therapy on rotator cuff disease [23]. The authors found an improved clinical outcome (pain relief, strength, and shoulder function) after 2 months in the group, where patients received manual therapy in combination with supervised shoulder exercises when compared to the group where only supervised shoulder exercises were performed.

7.3 Rehabilitation Regimes

The rehabilitation program of an athlete needs to be formulated by the physical therapist to the specific deficits and pathologies of every athlete. One of the main factors for successful rehabilitation is to understand the factors contributing to the rotator cuff disease.

Independent of the specific sport, the conservative treatment of a rotator cuff tear can be divided into four phases [5, 7]. In the first phase, the main objective is to control pain and inflammation. After this acute phase of about 2 weeks, the second phase begins, which focuses on achieving full range of motion, isometric and dynamic muscle strength, and neuromuscular function of the rotator cuff. It is also important not to provoke either apprehension or pain [5]. Generally, the exercises should be adapted to sport-specific movements of the shoulder. In throwers, for example, contractures of the posterior capsule are very common and should be addressed by therapy because they may lead to glenohumeral internal rotation deficit, displacement of the center of rotation, and an anterior tilt of the scapula [6, 30, 31]. Normally this phase takes about 4–6 weeks. The main goals of the third phase are to increase strength and joint stability throughout the kinetic chain, as well as gradually introducing sport-specific skills. This begins with plyometric training for throwers and sport-specific interval programs for all athletes. Finally, in phase four, the goal is the return to sports, with the ability to fully throw or strike and have the use of the upper extremity without symptoms or apprehension. Braun et al. [5] suggested starting the third phase within 3 months and return to competitive sports within 6 months; otherwise, a surgical intervention should be considered (Table 7.1).

7.4 The Role of the Scapula

The scapula plays an essential role in any shoulder injury and therefore in rehabilitation. The scapula is the origin of several muscles and has an important role in the energy transfer from the trunk to the upper arm. Weakness of the serratus anterior muscle or the lower and middle part of trapezius can lead to scapular instability, as is observed in two-thirds of all rotator cuff problems and in every glenohumeral instability disorder [34, 43]. These muscles are very important in scapular function because scapular dyskinesis may result in overuse with impingement and rotator cuff injury [25].

7.4.1 Scapular Dyskinesis

Several studies have shown that patients with clinical signs of rotator cuff tendinopathy also demonstrate kinematic alterations consistent with scapular dyskinesis [31, 41]. A recent study investigated risk factors for overuse shoulder injuries among male professional handball players. The authors found a significant correlation between obvious scapular dyskinesis and shoulder problems [10]. It remains unclear whether scapular dyskinesis is the cause of these problems or rather a result of it. The scapula is closely linked to the function of the rotator cuff since all of the rotator cuff muscles originate from the scapula. The position in relation to the chest influences the activity of the rotator cuff. Excessive external rotation of the humerus in relation to the acromion can result in a decrease of the subacromial space under the coracoacromial arch, which can increase the chance of experiencing rotator cuff tears. In this case, scapular dyskinesis may be the cause of the rotator cuff pathology [6]. As an indirect sign, Burkhart et al. proposed that a prominence of the superomedial border of the scapula can be associated with impingement and rotator cuff symptoms [6]. In the case where the dyskinesis is

Table 7.1 Summary of the goal and procedure of each phase as described by Braun et al. [5]

Phase 1	Reduction of pain and inflammation	Passive- and active-assisted ROM exercises
	Minimizing range of motion deficits	Nonsteroidal anti-inflammatory drugs
		Lymphatic drainage
		Cryotherapy
Phase 2	Normal range of motion	Specific stretching regimen
		Strengthening and neuromuscular exercises
Phase 3	Strength	Intensive strength and endurance training
	Stability	Neuromuscular training
	Sport-specific skills	Introduction of plyometric training
		Initial adapted sport-specific interval program
Phase 4	Back to sport	Strength and neuromuscular maintenance program
		Advanced interval throwing program
		Full throwing velocity

caused by the rotator cuff tear, it could be considered that dyskinesis further alters the biomechanics of the rotator cuff exacerbating the pathological changes. In either case, scapular dyskinesis not only correlates with rotator cuff disease but also with lower function scores. Therefore, its identification is crucial, and it should be treated in both the conservative and also surgical treatment plan.

7.4.2 SICK Shoulder Syndrome

If this dyskinesis is not corrected and progresses further, it may lead to SICK shoulder syndrome, a syndrome which particularly affects throwing athletes. This acronym SICK consists of **S**capular malposition, **I**nferior medial border prominence, **C**oracoid pain and maldisposition, and dys**K**inesis of scapular movement. If an athlete with a malpositioned SICK scapula keeps training or playing, the dysfunction will increase and intra-articular structural damage may occur as described by Burkhart et al. [6]. The rehabilitation of scapular dyskinesis should focus on stretching and mobilizing soft tissue, as well as regaining muscular strength and control due to the fact that the athlete may have problems with flexibility or muscle performance or both [14]. Based on the clinical assessment, the individual problems should be addressed by specific therapies. In order to improve flexibility, the scapular muscles such as pectoralis minor and levator scapulae should be stretched, and it is essential that the posterior capsule be mobilized, especially in throwing athletes. As these athletes have the same range of motion on both sides with less internal rotation to gain more external rotation, we personally think that scapular setting is more important on glenohumeral positioning and force transmission than stretching of the posteroinferior capsule. In order to improve muscle performance, the lower and middle trapezius muscles have to be trained, together with the serratus anterior muscle to obtain a better balanced force ratio, a good position of the scapula, and enhanced energy transfer to the upper arm. De Mey et al. [15] reported improved scapular function and less pain due to a significant functional improvement in overhead athletes after a 6-week training phase according to the method of Cools et al. [13].

7.5 Kinetic Chain

Kinetic chains connect the muscles and their respective forces through the body, from the foot through to the trunk and up to the shoulder. So if, for example, there is weakness of the hip abductors or trunk stabilizers, this may influence the shoulder by kinetic chain transmission [25]. Regarding shoulder function, abnormalities in the kinetic chain can cause unfavorable position out of the safe zone as described by Greiwe and Ahmad [25]. When the shoulder is hyperabducted and in an externally rotated position, this may increase compressive and shear forces on the rotator cuff, glenoid, and the capsule–labral complex. Therefore, preventing further injury begins with preservation of the kinetic chain coordinating transmission of forces from the legs and trunk to the upper extremity. As the kinetic chain plays an important role, especially for throwing motion, a

training program strengthening all elements of the chain and linking them together is crucial. All athletes should be educated to work on a well-balanced distribution of exercises for the agonist and antagonist muscles of the upper extremities, and should add strength and stability exercises for the lower extremities and trunk [11].

7.6 Injury Prevention

All throwing and striking athletes should integrate a rotator cuff injury prevention program in their training. These programs have to address flexibility and muscle strength. They also have to restore scapular stabilizers and external rotators of the cuff. Furthermore, in throwers as well as volleyball or tennis players, the program should focus on core strengthening and stability training of the lower limb, especially of the hip abductors and the foot.

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Rotator Cuff Tear in Athletes: Part III. Surgical Treatment

8

Maxwell C. Park

8.1 Introduction

The management of a rotator cuff tear in the athlete can be challenging, particularly when accounting for sport-specific performance demands and seasonal considerations. Perhaps the most critical distinction between types of rotator cuff tears in athletes is the chronicity of the symptomatic tear: Is the tear acute versus chronic? Acute tears are generally associated with contact athletes, while athletes that perform repetitive overhead throwing motions are at risk for chronic overuse degenerative-type tears; the management of each type can be very different. Acute tears typically have a relatively better prognosis when considering that overuse injuries can involve anatomic adaptations that develop over time after repetitive cycles of use that can contribute to or cause the injury to begin with. The other distinction that can be made, which affects treatment and prognosis as well, is between partial- and full-thickness tears. Given another chapter is devoted to partial articular-sided rotator cuff tears, this chapter will primarily focus on full-thickness tears.

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8.2 Pathoanatomy, Biomechanics, and Preferred Classification

Pathoanatomy can be characterized as arising from acute versus chronic injuries. For acute injuries, from a direct blow or fall on the shoulder, or a fall onto an outstretched hand, the proximal humerus may forcibly contact the acromion [8], creating tendon displacement or tearing, typically involving the supraspinatus tendon, which can be otherwise healthy without prior symptoms. Dislocation of the glenohumeral joint may cause rotator cuff tears involving the supraspinatus and infraspinatus tendons (and more rarely the subscapularis tendon) in the acute traumatic setting as well, particularly in the older patient. For chronic repetitive overuse tears, typically in the athlete who performs repetitive exertional overhead activity, tearing of the posterior supraspinatus and anterior infraspinatus can occur, for example, as a result of chronic internal impingement. Understanding the nature of injury can help frame the expectations of any particular repair that might be performed—acute injury arguably having a better prognosis when the torn tissue is not degenerative, as may be the case in the chronic overuse setting.

The biomechanics of repair should be understood in order to optimize the technical management of various tear patterns. In recent years, the transosseous-equivalent (TOE) repair technique has become a standard from which

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other repairs have been measured [22, 27, 30]. Arguably the primary benefit of this technique lies in its ability to restore footprint dimensions after repair [28]. Other biomechanical benefits include improved load to failure [22], self-reinforcement [26], gap formation resistance [23], interconnectivity [29], and resistance to fluid extravasation [1].

Footprint restoration requires understanding the unique anatomy of the native tendon. For example, the supraspinatus has an inherent asymmetry. The anterior region is cord-like, while the posterior region is strap-like. This creates an asymmetry in loading stresses, which can affect repair constructs [10]. The anterior region experiences more loading forces, and additional anterior fixation may be helpful. The strains experienced anteriorly are enhanced with external rotation [24]. While it is generally understood that more tendon suture passes will lead to better fixation [13], this needs to be weighed against physiological necessity and surgical times [19].

Technical surgical efficiency can be measured using a so-called technical efficiency ratio to help gauge biomechanical sufficiency in the context of technical ease for a given repair construct [19]. The original description of the TOE repair requires at a minimum two medial horizontal mattresses, two knots, and four anchor implants. This can be quantified by the ratio: ((# tendon suture passes + # suture limbs requiring management + # knots)/# implants used); for the original TOE, the ratio is (4 + 4 + 2)/4 = 2.5. This provides a basis for comparing like repair constructs, providing a measure for technical difficulty in a clinical setting, and may help in creating hypotheses when biomechanically testing newly proposed repair constructs in the laboratory [27].

Recently, an optimized (from a technical standpoint) TOE repair construct has been biomechanically tested and validated [27]. It involves a broad medial inter-implant mattress created from a medial pulley (MP-TOE) configuration, instead of two separate and isolated focal mattresses (Fig. 8.1). Because only two tendon suture passes are necessary, the technical efficiency ratio is (2 + 4 + 2)/4 = 2.0, with the smaller number representing improved efficiency or less difficulty when compared to a similar construct such as the original TOE repair. The broad medial mattress can demonstrate a unique "purse-string" mode of failure without medial tearing at the musculotendinous junction with failure loading, suggesting improved load-sharing capacity; with tendon loading, the central

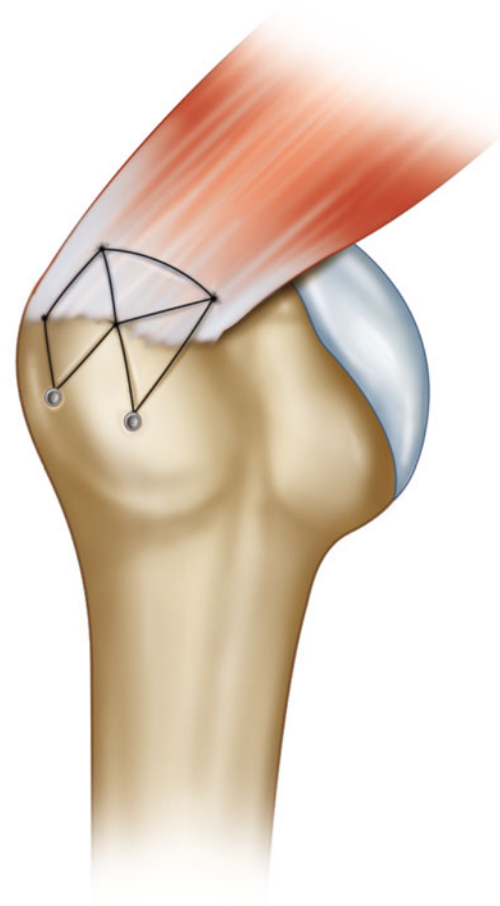

Fig. 8.1 "Medial pulley transosseous-equivalent" repair depicting a modified TOE construct with a broad medial inter-implant mattress configuration. This construct only requires two tendon suture passes, without compromising biomechanical performance compared to the original TOE construct (which requires a minimum of four tendon suture passes). Technical efficiency is therefore improved (Adapted from Park et al. [27])

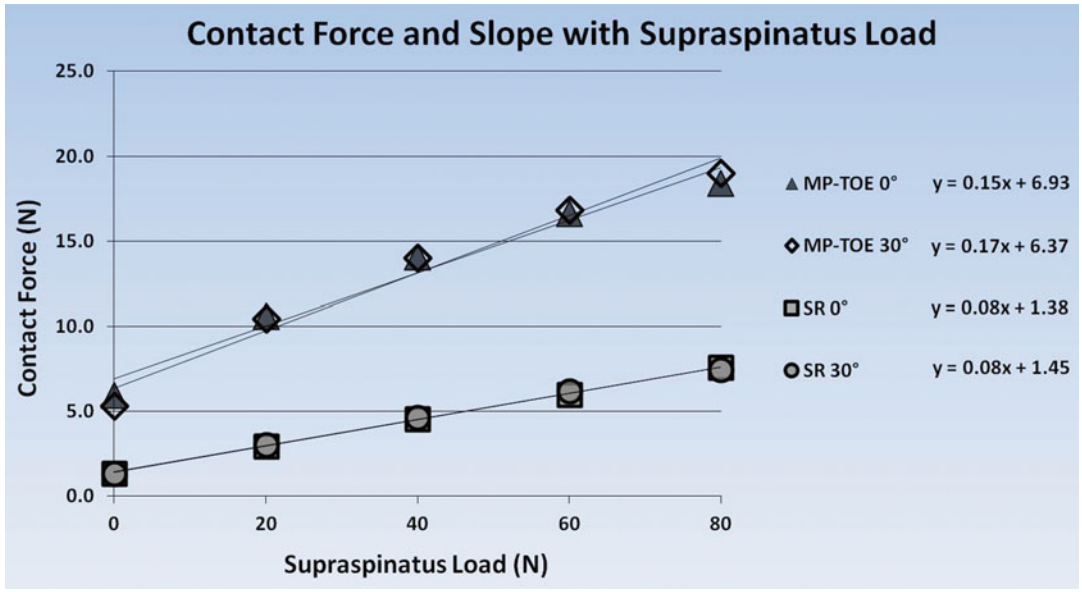

Fig. 8.2 Footprint contact force with progressive tendon loading. At each load, the MP-TOE repair provided significantly more contact force ($p<0.05$) compared to single-row (SR) repair. With increasing loads, the MP-TOE repair had a significantly higher progression (slope) of footprint contact force compared with single-row repair at both 0° ($p=0.025$) and 30° ($p=0.014$) abduction. If the slopes were not significantly different between repairs, the "self-reinforcement" effect would not have been validated

repair converges across the broad medial mattress configuration giving the appearance of a purse-string effect. The original TOE repair with separate spot-weld medial mattress configurations can demonstrate medial tear patterns [6].

Tendon suture-bridging constructs have been theorized to be "self-reinforcing" in the face of potentially destructive tensile loading forces, mimicking a Chinese finger trap where distraction forces also contribute to resistance to separation [3]. This concept has been biomechanically characterized and validated [26]. Force sensors were placed on the footprint prior to repair with the MP-TOE technique in ten cadaveric specimens. With progressive tendon loading, the frictional forces increased as well. Using the same methodology, a standard "single-row" (SR) repair demonstrated the same relationship; however, the progression of resistive frictional force was disproportionately more with the MP-TOE repair (Fig. 8.2). The SR repair creates a "focal loop wedge" effect, where the focal lateral suture loop elongates creating obligatory tendon compression over the lateral footprint with tendon force simulation (Fig. 8.3). With SR repair, tendon loading creates suture loop elongation and coincidental construct failure with increasing gap formation. In this context, SR repair cannot be optimally self-reinforcing as it does not fix the tendon-bridging suture loop medially whereby a more complete footprint "wedge" effect [3] can occur to include the medial footprint (Fig. 8.4).

Rotator cuff tear classification for full-thickness tears can be based most intuitively on footprint anatomy: Stage 0 or normal, Stage I supraspinatus only, Stage II overlap area (supraspinatus and infraspinatus), Stage III the anterior half of the infraspinatus, and Stage IV the entire supraspinatus and infraspinatus tendons [17]. While this classification is based on functional anatomy, intraoperative technical decision-making is based on tendon mobility and tissue quality (tendon and bone).

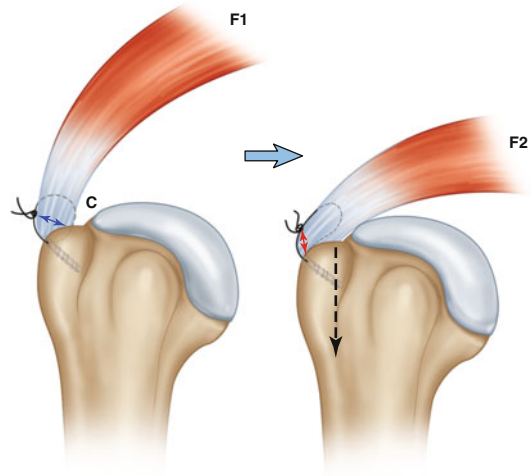

Fig. 8.3 Schematic rendering showing the "focal loop wedge" effect. With increasing tendon load (F1 to F2), focal loop stitch configurations (as seen with single-row repairs) elongate, thus compressing the focal loop stitch (*blue to red double arrows*), while exposed footprint contact area C is relatively decreased. In turn, the F2 load is creating a focal compression vector (*down arrow*) onto the footprint. This is an example of a rotator cuff "self-reinforcement" effect. However, this effect is significantly improved with tendon-spanning constructs that are fixed medially to secure a "loop" that can bridge the entire footprint; this has been shown to provide disproportionately more progressive footprint frictional resistance even with disruptive tendon loading forces [26]

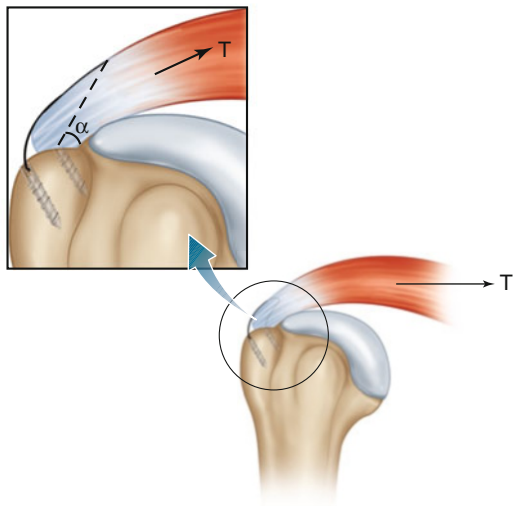

Fig. 8.4 Medial fixation secures a "loop" of suture that spans the footprint, allowing for a more complete footprint restoration, which increases the compressive vector over the insertion even with tendon loading. As the load T increases, the angle decreases, wedging the tendon between the tendon-bridging sutures and bony insertion ("wedge" effect). With single-row repair, the suture loop is not fixed medially, as the tendon is secured over the isolated anchors only (From Burkhart et al. [3])

8.3 Clinical Presentation and Essential Physical Examination

The athlete with a rotator cuff injury will typically present with either an acute traumatic injury or a chronic history of more than 6 months having had persistent and progressive pain. The tendon regions commonly involved include the supraspinatus tendon and anterior aspect of the infraspinatus tendon. For an acute injury, the mechanism of injury may point to the type and extent of injury. For chronic injuries, the athlete can usually be participating in sports that involve repetitive overhead motion, such as baseball, volleyball, tennis, water polo, and the like. The prognosis for acute injury after repair is arguably more predictable than for chronic injury with a repetitive overuse etiology insofar as return to sport in the latter setting involves recreating the very mechanism that contributed to the threshold injury. Furthermore, truly chronic tears may be associated with muscle atrophy and fatty infiltration that may be irreversible. Each patient may present different functional goals after surgery largely

based on the level of play (e.g., "weekend warrior" to professional), and this will frame the reasonable expectations that can be achieved after repair.

A careful physical examination begins with inspection. The affected extremity may manifest atrophy; and gross atrophy within the scapular fossae could be diagnostic for a rotator cuff tear—a spinoglenoid notch cyst with suprascapular nerve compression would be in the differential diagnoses, however, and careful examination for labral pathology must be considered particularly in the athlete who performs repetitive exertional overhead motion. Passive range of motion is critical to assess to eliminate a concurrent diagnosis of frozen shoulder or glenohumeral osteoarthritis. Excessive external rotation relative to the normal shoulder may signify a torn subscapularis tendon. Active range of motion testing generally will gauge the functional limitations the patient may have.

Motor testing can isolate the muscles involved. Resisted forward elevation in the scapular plane can help assess supraspinatus involvement, although a negative test does not mean a tear is not present, especially when the tear is only partial full-thickness, leaving a tear that is functional on examination. In addition to elevation, the supraspinatus assists rotation of the humerus internally and externally. Weakness to external rotation at the side generally means the patient has supraspinatus and infraspinatus involvement—the patient may elevate the elbow to compensate. However, a negative test does not preclude a supraspinatus tear, even though the tear may be full-thickness as it may be only partially torn in the anterior-posterior dimension. The belly-press, lift-off, and bear-hug tests can help measure subscapularis tendon pathology, although this tendon is not commonly injured relative to the supraspinatus and infraspinatus tendons. The belly-press test is performed with the hand pressing maximally into the abdomen and the elbow in line with the trunk in the sagittal plane—a positive test is manifested by a relative weakness compared to the normal side or the elbow dropping posteriorly in the sagittal plane. The lift-off test is performed by placing the dorsum of the hand against the mid-lumbar spine—a positive test is apparent when the patient is unable to internally rotate and lift the hand away from the back. The bear-hug test is performed with the hand placed on the contralateral shoulder over the acromioclavicular joint—a positive test results when the examiner can externally rotate the hand away from the initial position.

Neer and Hawkins tests are typically used to assess external impingement underneath the coracoacromial ligament arch. Pain posteriorly with abduction and external rotation, with or without instability, indicates the possibility of internal impingement, particularly in an overhead throwing athlete, for example. Other provocative tests can be used to assess for non-tendinous concurrent pathologies, including labral tears, which can dictate the type of imaging study that is obtained, and the surgical approach.

8.4 Essential Radiology

Radiographic x-ray imaging can provide baseline information regarding the athlete's shoulder. When rotator cuff pathology is suspected in the athlete, true anterior-posterior, outlet, and axillary views are routinely obtained. Arthritic changes of the glenohumeral and acromioclavicular joints will give a measure of relative overuse. Glenohumeral osteoarthritic changes would lend to a poor prognosis with respect to rotator cuff tendon pathology. In a more senior athlete, a high-riding humeral head would also be a poor prognostic factor, consistent with chronic overuse, suggesting a large to massive rotator cuff tear involving both the supraspinatus and infraspinatus tendons. The acromiohumeral interval (AHI), the distance between the acromion and humerus, has been inversely related to rotator cuff tearing and fatty infiltration [33]. Fatty infiltration can be classified by using computed tomography (CT). Grade 0 has been defined as normal muscle, grade 1 fatty streaking, grade 2 more muscle than fatty infiltration, grade 3 equal amounts of muscle and fat, and grade 4 more fatty infiltration than muscle. Increasing grade of infiltration has been correlated with worsening function [4, 11,

Type	Description	Preoperative MRI Findings	Treatment	Prognosis
1	Crescent	Short and wide tear	End-to-bone repair	Good to excellent
2	Longitudinal (L or U)	Long and narrow tear	Margin convergence	Good to excellent
3	Massive contracted	Long and wide, >2 × 2 cm	Interval slides or partial repair	Fair to good
4	Cuff tear arthropathy	Cuff tear arthropathy	Arthroplasty	Fair to good

Fig. 8.5 Geometric classification (Adapted from Davidson and Burkhart [7])

12], and high grades have been associated with irreversible changes, even after repair.

Magnetic resonance imaging, in addition to gauging fatty infiltration, can directly assess the degree of tendon involvement, and a geometric classification has been proposed to aid in predicting repair potential and prognosis after repair [7] (Fig. 8.5); care should be taken to identify the anterior cord of the supraspinatus tendon. T2-weighted coronal and sagittal oblique views should be evaluated. In the type 1 tear, the tear is retracted on the coronal view more than it is wide on the sagittal view, with retraction <2 cm; this would be equivalent to a crescent-type tear, with a relatively good to excellent prognosis. The type 2 tear is more retracted than it is wide, with the width being <2 cm; this is equivalent to a longitudinal tear ("L" or "U") also with a good to excellent prognosis. Massive contracted tears would define the type 3 tear with retraction and width being greater than or equal to 2 cm, and the prognosis is more guarded. Cuff tear arthropathy defines the type 4 "tear" using this classification. This geometric classification allows for recommendations on repair technique based on the classification type: Type 1 would allow for end-to-bone repair, type 2 margin convergence, type 3 interval slides or partial repair, and type 4 arthroplasty.

8.5 Disease-Specific Clinical and Arthroscopic Pathology

The spectrum of injury involving the athlete's rotator cuff includes tendonitis, partial tears (bursal- and articular-sided), and full-thickness tears. The athlete with a torn rotator cuff will typically have pain that prevents full participation; sport-specific activity will usually be limited with respect to range of motion and strength. Arm activity above shoulder level is particularly affected. Athletes with chronic tears may participate until threshold injury occurs, which may involve some degree of tear progression. Either the athlete will cycle back to a rest-and-participation cycle or threshold injury prevents return.

During arthroscopy, the diagnosis can be confirmed. A spectrum of injury may be encountered from tendonitis and interstitial tearing, to partial tearing, to full-thickness tears. In tears that occur from acute injury, pathology may be isolated to the rotator cuff. Acute tears can arise when the force between the greater tuberosity and acromion displaces the rotator cuff, usually seen more in the contact athlete. Either partial- or full-thickness tears may occur. Chronic tears may manifest structural degeneration of the coracoacromial ligament (external impingement), in which case acromioplasty should be carefully considered [35].

In the overhead throwing athlete, the labrum and biceps tendon must be carefully inspected and characterized. Adaptive changes may give false-positive findings, and care must be taken not to aggressively treat all findings beyond simple debridement; otherwise, there may be an increased risk for over-constraining the shoulder. This may lead to a return to sport, but at a lower level, or possibly be career-ending [21]. Internal impingement from overuse throwing can manifest as partial articular-sided tears of the infraspinatus tendon and partial tearing of the posterosuperior labrum.

8.6 Treatment Options

A spectrum of pathology may be present in the athlete, whether arising from an acute or chronic setting: tendonitis and interstitial tearing, to partial bursal- or articular-sided tearing, to full-thickness tearing. In general, the smaller or lesser the extent of injury, the more likely nonoperative management

may be effective. And acute trauma may have a better prognosis for both nonoperative and operative management, relative to chronic overuse injuries. For overuse injuries, return to sport can be difficult when the sport activity itself contributes to the injury.

In general, all types of rotator cuff injuries can be considered for nonoperative management. Perhaps most importantly, a requisite healing period must be respected, to give the biology of healing an opportunity. Range of motion must be maintained. Typically passive and active range of motion should be progressed in a graded manner; in the throwing athlete, attention toward stretching the posterior capsule may be necessary. Routine nonsteroidal anti-inflammatory medications and icing may be initiated. A cortisone-type injection trial can be considered, with strict counseling to avoid a premature return to play and the risk of relative overuse. Once pain is controlled, progressive conditioning may be initiated. Generally, rotator cuff injuries can take 3–6 weeks minimum prior to return to sport; injuries that involve loss of structural integrity usually take longer, up to 12 weeks minimum. Each patient's progress must be individualized, and attention should be given to the timing (symptom duration), the sport-specific requirements, and the patient's goals. Although individualizing treatments is recommended, generally, full-thickness tears in the athlete warrant surgical intervention or strong consideration with a relatively lower threshold to perform surgery, as the natural history is for tears to progress [39].

When nonoperative management fails, arthroscopic surgery can be considered. For tendonitis or tears involving less than 50 % of the tendon substance, including either bursal- or articular-sided tears, careful debridement and subacromial decompression can be considered. However, there remains some controversy regarding the indications for acromioplasty; generally when coracoacromial ligament changes exist, acromioplasty should be strongly considered. For partial articular-sided tears involving more than 50 % of the footprint, in situ trans-tendon repair and completing the tear followed by repair have been described [14]—discussion here is reserved for the following chapter 9. For bursal-sided tears involving more than 50 % of the medial-lateral substance, including full-thickness tears, a repair can be considered. For small 10 mm tears without retraction, single-anchor repair can be sufficient. For larger tears, SR and TOE repairs can be considered with tissue loss and tendon retraction being important factors in repair strategy decision-making.

8.7 Author's Preferred Treatment

The lateral decubitus position is preferred with 10 lbs of arm traction and approximately 45° arm abduction; simultaneously, 5 lbs of traction is placed at approximately 30° abduction via a second pulley to allow another option for footprint exposure and implant approach as needed. A standard posterior portal is used to access the glenohumeral joint. Under direct visualization, an anterior portal is made at the rotator interval using needle localization; this is cheated slightly superior to allow for potential anchor placement through this portal as needed. A nerve hook probe is used to perform routine diagnostic arthroscopy with a 30° arthroscope; relatively rarely, a 70° arthroscope can be used for subscapularis tears. In the athlete, attention must be given to the capsulolabral structures including the biceps tendon. The rotator cuff tear can be thoroughly characterized by careful inspection and probing, with both intra- and extra-articular arthroscopy. After switching the posterior cannula to the subacromial space, an anterolateral working portal can be established after needle localization; care must be taken to not place this portal too superior or inferior, accounting for access and exposure with respect to suture passing and distal-lateral anchor placement. If a kissing lesion on the coracoacromial ligament is encountered (usually seen in athletes with a chronic history), the ligament is released with a heat probe; acromioplasty when indicated is performed at the end of the case to limit bleeding and risk for adversely affecting visualization during the repair. A posterolateral portal, also established with needle localization, is often used to achieve a bird's-eye view of the torn tendon.

For full-thickness supraspinatus tendon tears less than 10 mm in the anterior-posterior dimen-

sion, a single-anchor knotless repair is preferred [20]. A tear that is non-retracted and without tissue loss are both prerequisite indications. After debridement of the footprint and gentle decortication, a tape-type suture such as FiberTape (Arthrex, Naples, FL) is used to create an inverted-mattress repair. It is important to emphasize that trial reductions must be performed prior to passing any sutures through the tendon to fully characterize the tear pattern. Each tendon suture pass is approximately 12 mm medial to the tear and 7–8 mm apart from one another in the anterior-posterior dimension; if the suture passes are too far apart from one another, a central "dog ear" malreduction can occur. The anterior-most suture pass should ideally capture the anterior cord. A self-grasping suture passer is preferred. Through the anterolateral portal, the FiberTape is then incorporated into a knotless anchor and placed 10 mm distal-lateral to the footprint edge, thus preserving the entire bony footprint for potential healing (compared to placing the anchor on top of the footprint). Given the tear size is relatively small, the load to failure need not be high as a significant portion of the footprint is intact. Concern may arise from the fact that abduction could reduce tendon contact; however, given the relatively small tendon tear dimension, this may not be clinically relevant. Further, significant loss of contact has not been shown to occur with 0° and 30° abduction, but only with 60° abduction which is not a static postoperative position [28]. This type of repair has been shown to better restore tendon repair morphology compared to a repair using a suture anchor on top of the footprint (Fig. 8.6) [20].

This repair strategy can be used for high-grade bursal-sided tears and superior subscapularis tendon tears that are 10 mm or less as well, as long as tendon suture passes can be achieved with the appropriate dimensions [18]. For subscapularis tendon tears, the anterior portal, or an accessory portal, may be used as the working portal. For tears with tendon tissue loss or significant retraction, the anchor should be placed on top of the footprint—simple suture configurations with routine knots are commonly used.

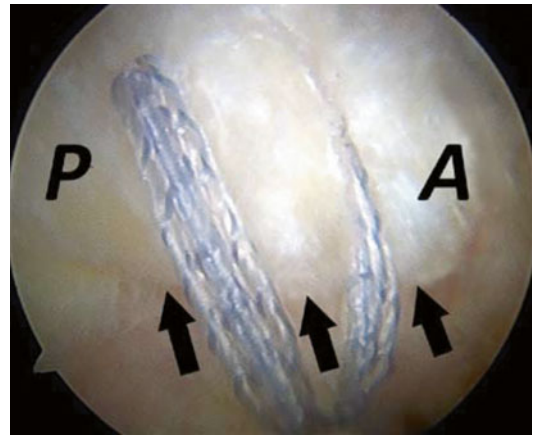

Fig. 8.6 Inverted-mattress single-anchor repair using tape-type suture incorporated into a knotless anchor placed 1 cm distal-lateral to the footprint. This has been shown to provide improved tendon morphology compared to single-anchor repair with the anchor placed on top of the footprint. Further, the entire bony footprint is available for tendon-to-bone healing when the anchor is not occupying the footprint itself [20]. The lateral footprint margin (*arrows*) is restored (*A* = anterior, *P* = posterior)

For full-thickness tears greater than 10 mm in the anterior-posterior dimension or involving the entire supraspinatus tendon, and having sufficient mobility and adequate footprint coverage, the MP-TOE repair is preferred. This involves a 2-suture-pass TOE repair involving a broad medial inter-implant mattress configuration [27]. This construct is believed to be an evolved product of the original description of the TOE repair; although technically easier to do with fewer tendon suture passes, biomechanical performance is not compromised. Further, this repair has demonstrated less trauma at the musculotendinous junction where failure has been a concern [6]. After footprint preparation, two single-loaded medial anchors are placed 12–15 mm apart. An accessory superior portal is made to place both anchors; depending on the patient's anatomy, the anterior and posterior portals may be used to place the anterior and posterior medial implants, respectively. Using a suture shuttle (FiberSnare, Arthrex, FL), both suture limbs from each anchor are shuttle-passed *simultaneously* over each anchor approximately 12 mm medial to the torn tendon edge. A suture limb from each anchor is

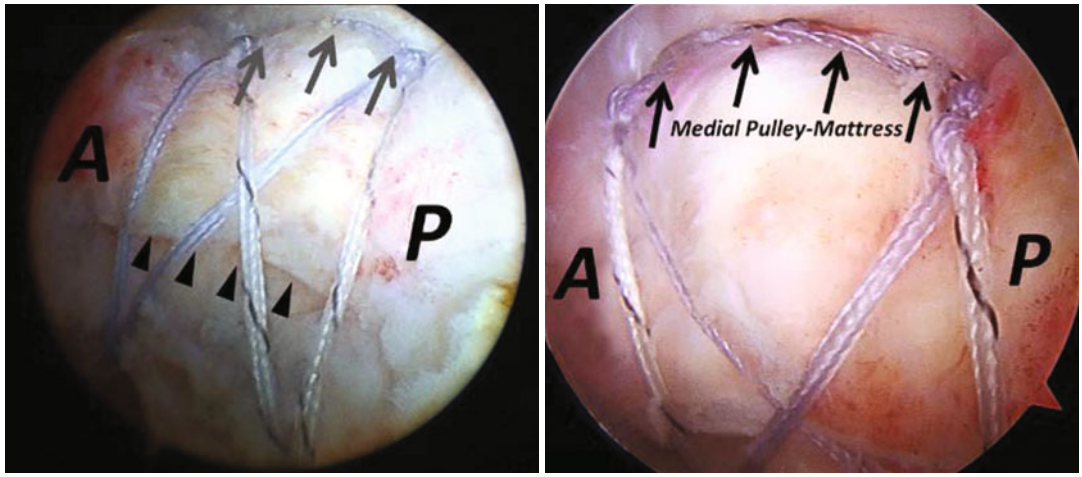

Fig. 8.7 Examples of MP-TOE repair arthroscopic pictures taken from the posterolateral portal. The lateral footprint (*arrowheads*) is restored. Medially, a broad 2-suture-pass inter-implant mattress configuration (*arrows*) creates a construct which has demonstrated improved load-sharing capacity compared to the original TOE repair which utilizes 2 separate and focal medial mattress configurations (4 tendon suture passes) [27] (*A* = anterior, *P* = posterior)

then tied to one another outside the anterolateral cannula. The free suture limb posteriorly is then pulled to bring the knot to the posterior anchor. The posterior limb is retrieved through the anterolateral cannula with the free anterior suture; these sutures are then tied over the anterior anchor using a non-sliding knot. With a single knot over each anchor, a suture limb from each knot is then bridged posteriorly using a knotless anchor. The remaining suture limbs from each anchor are bridged anteriorly; both lateral anchors are fixed 10–12 mm distal-lateral to the footprint (Fig. 8.7). Given biomechanical analyses, single-loaded medial anchors have been shown to be sufficient, optimizing technical efficiency (limiting unnecessary suture passes or management), and without compromising biomechanical performance. Double-loaded anchors may be used based on surgeon preference, but this would be technically more involved and may put the repair at risk for medial failure.

For tears that are larger than 10 mm in the anterior-posterior dimension and that also involve tissue loss and undue tension after trial reductions, a simple SR repair is preferred, with additional suture augmentation (e.g., margin convergence, "rip-stop") as the tear pattern dictates. Creating a tension-free repair takes priority over complete footprint restoration—in this way, medial failure can be avoided. The tear pattern should always dictate the repair strategy.

8.8 Rehabilitation

After surgical repair, the patient's shoulder is immobilized with an abduction pillow. Irrespective of tear size, progressive range of motion is all that is prescribed for the first 12 weeks postoperatively. Progressive resistive exercises are initiated no sooner than 12 weeks from the time of surgery. Sport-specific reconditioning may be initiated at 12 weeks as clinical progress dictates.

For all repairs, it must be emphasized to the athlete that the recovery is the most important variable with respect to healing biology. Noncompliance in the eager or overzealous athlete, no matter how well the repair was performed at time zero, will increase the risk for tear persistence and recurrent failure.

8.9 Advantages, Pitfalls, and Complications

The advantage of the MP-TOE repair is that biomechanical performance does not need to be compromised; although technically easier to perform, insofar as fewer suture passes through the tendon are necessary compared to the original TOE repair, gap resistance and load to failure are not compromised [27]. Further, a self-reinforcing effect has been validated for this construct [26]. Pitfalls are related to incorrect tear pattern recognition and insufficient tendon trial reduction—"dog ear" malreduction may be corrected with accessory fixation, but it is best to avoid this altogether with careful trial reductions. A complication that has been well documented is medial failure [5, 6]; should this occur, salvage may prove to be very challenging as there may be limited or no tissue to receive additional sutures in the revision setting. The MP-TOE repair has been shown to potentially provide improved load-sharing capability medially, and less medial tissue failure has been characterized with failure load testing; a "purse-string" mode of failure has been demonstrated with tissue convergence across the broad medial inter-implant mattress configuration. Spot-weld medial mattress fixation has demonstrated medial tissue failure.

A fundamental complication may be related to noncompliance with rehabilitation timelines. Often range of motion and pain are improved well before repair healing is fully optimized locally over the footprint. Failure to achieve sufficient healing prior to exerting the repaired tendon can limit or negate the opportunity to heal, increasing the risk for predictable failure. With seasonal return to play considerations, this is a mandatory discussion with the patient-athlete.

8.10 Experience in Treatment of Athletes

In my experience, the results with single-anchor repair, single-row repair, and MP-TOE repair in athletes have matched the general results seen in the literature with 85–90 % good to excellent results [2, 9, 25, 31, 32, 34, 37, 38]. Repair success is predicated on tear pattern recognition and intraoperative trial reductions. Overhead throwing athletes are a special subpopulation of athletes in the context of rotator cuff injury. One study reviewed the results of recreational and amateur overhead throwing athletes and found that arthroscopic rotator cuff repair led to good clinical results with a high rate of return to sports, despite a re-tear rate of 23.8 % [15]. In the case of the overhead athlete with a full-thickness rotator cuff tear, especially pitchers, the prognosis for a predictably satisfactory outcome is limited [16, 36]; the repetitive overhead activity itself contributes to the injury, thus making return to the same activity challenging.

Beyond appropriate repair selection, the single-most important factor contributing to success in my experience has been emphasizing patient education and compliance—this applies both pre- and postoperatively. Educating the patient-athlete is fundamental to a satisfactory outcome and frames the expectations and in turn the perceived results. Optimizing the healing environment, which largely involves preventing premature return to sport, in active and eager patients, is a primary challenge—especially in the context of seasonal calendars that can shorten perceived healing timelines. Practical timelines may not match true healing timelines which can put rotator cuff repair at risk in athletes, unless the surgeon proactively manages sport-specific rehabilitation. Engaging the trainer or therapist is essential to optimizing the outcome therefore.

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9 Current Concepts: Arthroscopic Treatment of Articular-Sided Partial-Thickness Rotator Cuff Tears

Teruhisa Mihata

9.1 Introduction

Surgical treatment of articular-sided partial-thickness rotator cuff tears is typically indicated after failed conservative management, and various approaches, including debridement of partial-thickness tears with or without acromioplasty [3, 8, 21, 35, 38, 41], trans-tendon repair [6, 11, 15, 22, 39, 42, 46], or conversion to a full-thickness tear followed by repair [10, 11, 19, 37, 39], have been reported. The best surgical option for each patient will differ and needs to be determined on the basis of the patient's background (including their gender, age, sport, and job) and the results of physical examination, including those of shoulder laxity and stiffness.

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9.2 Pathoanatomy and Biomechanics

The articular-sided partial-thickness rotator cuff tear is traditionally considered to be a variant of the rotator cuff tear. However, a recent anatomical study has shown that the superior shoulder capsule is attached to a substantial area (30–61 %) of the greater tuberosity [32]. This suggests that articular-sided partial-thickness tears of the supraspinatus and infraspinatus tendons include detachment of the superior shoulder capsule from the greater tuberosity. It also suggests that low-grade partial tears found to be less than 50 % of the tendon thickness are not rotator cuff tears but just superior capsule tears.

Because shoulder capsule tears can result in increased glenohumeral translation [1, 2, 5, 33, 34, 43, 45], articular-sided partial-thickness supraspinatus and infraspinatus tendon tears are associated with increased glenohumeral joint laxity. A biomechanical study by Ishihara et al. has shown that a tear in the superior capsule at the greater tuberosity—as can occur with articular-sided partial-thickness rotator cuff tears—increases anterior and inferior translation [16]. Increased shoulder laxity contributes to shoulder symptoms or further glenohumeral injury in throwing athletes [13]. Therefore, shoulder laxity needs to be considered in the treatment of articular-sided partial-thickness supraspinatus and infraspinatus tendon tears.

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9.3 Clinical Presentation and Essential Physical Examinations

Both careful assessment of the patient's complaint and physical examination for shoulder instability or pathological shoulder laxity are the most important factors in treating articular-sided partial-thickness rotator cuff tears. If the patient has symptoms of shoulder instability or a history of shoulder dislocation, the torn labrum or capsular ligaments should be treated. If the patient has pain at a position of maximum external rotation of the abducted shoulder, pathological shoulder laxity—including internal impingement or peel back of the superior labrum—should be suspected. Scapular function, muscle strength, and capsular condition should also be evaluated.

The Hara test (Fig. 9.1) is useful for assessing the upper-extremity kinetic chain for abnormalities leading to shoulder pain in patients with articular-sided partial-thickness rotator cuff tears. The Hara test comprises 11 physical examinations relevant to the scapular and humeral kinetic

Hara test scoring sheet

Date of Examination _____

Name _____ Age_____ Sex _____

Dominant Hand (R) _____ (L) _____

Sport_____ Position _____ Years Played _____

Instructions to examiners:

Please perform and score the following 11physical examinations and then calculate the total score (i.e.,the number of "intact" results).

Scapular function		
Scapula–spine distance	Abnormal	Intact
Elbow extension test	Abnormal	Intact
Elbow push test	Abnormal	Intact
Manual muscle strength		
Abduction	Abnormal	Intact
External rotation	Abnormal	Intact
Internal rotation	Abnormal	Intact
Posterior tightness		
Combined abduction test	Abnormal	Intact
Horizontal flexion test	Abnormal	Intact
Capsular laxity tests	Abnormal	Intact
Subacromial impingement tests	Abnormal	Intact
Hyper external rotation test	Abnormal	Intact
Total Hara test score (number of "intact"results)	_____	

Fig. 9.1 Hara test scoring sheet

chain: (1) scapula–spine distance (Fig. 9.2), (2) elbow extension test (Fig. 9.3), (3) elbow push test (Fig. 9.4), (4) manual muscle strength of abduction, (5) manual muscle strength of external rotation, (6) manual muscle strength of internal rotation, (7) combined abduction test (Fig. 9.5), (8) horizontal flexion test (Fig. 9.6), (9) capsular laxity tests, (10) subacromial impingement tests, and (11) hyper-external rotation test (Fig. 9.7). The total score (i.e., the number of "intact" results—see Fig. 9.1) for the Hara test and the abnormalities in each examination are evaluated.

The scapula–spine distance, elbow extension test, elbow push test, subacromial impingement tests, and manual muscle tests of shoulder abduction, external rotation, and internal rotation are assessed while the subject is sitting. Patients are supine for the combined abduction test, horizontal flexion test, capsular laxity tests, and hyper-external rotation test.

In the scapula–spine distance test, the distance from the medial edge of the scapular spine to the

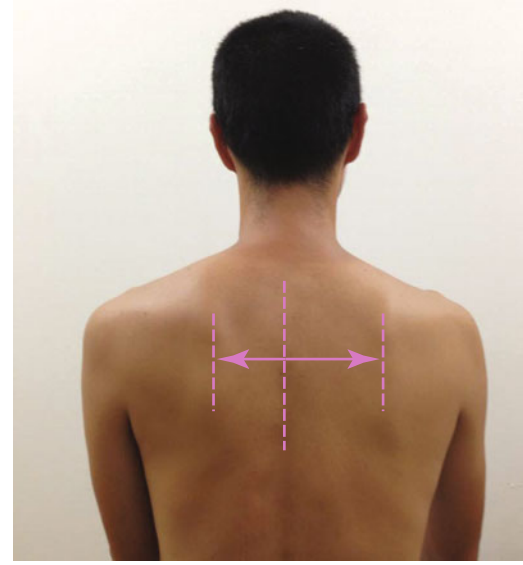

Fig. 9.2 In the scapula–spine distance test, the distance from the medial edge of the scapular spine to the spinous process of the thoracic spine is measured with the arms at the sides. The reference point on the thoracic spine is defined as the nearest spinous process. A difference of more than 1.0 cm between the left and right sides is considered abnormal

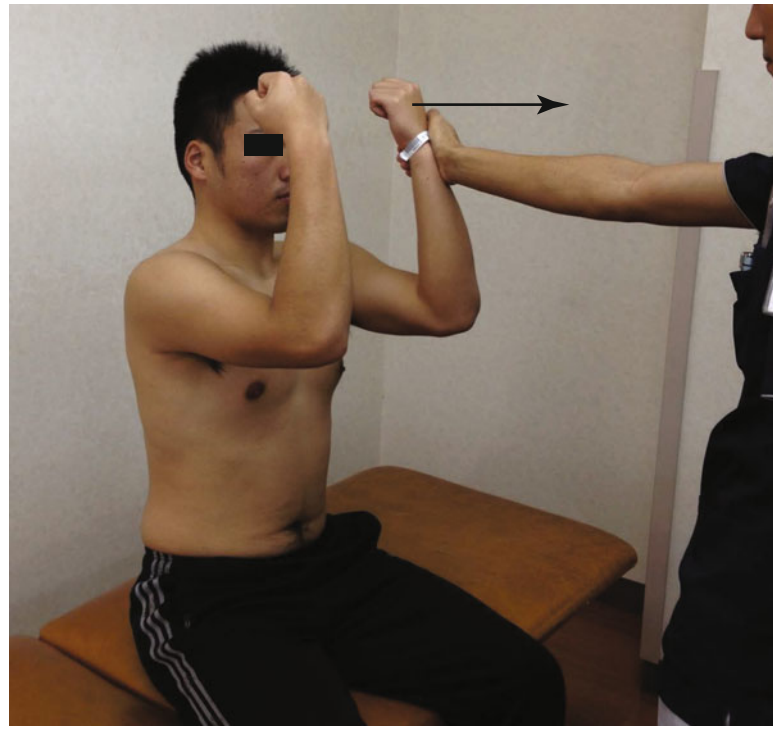

Fig. 9.3 Elbow extension test for assessment of scapular stability. The elbow extension test is performed with the shoulders in 90° of forward flexion. The subject extends the elbow joint from 90° of flexion with maximum force while the examiner holds the subject's forearm to resist his extension force. The test is considered abnormal when the muscle strength on the dominant side is less than that on the nondominant side

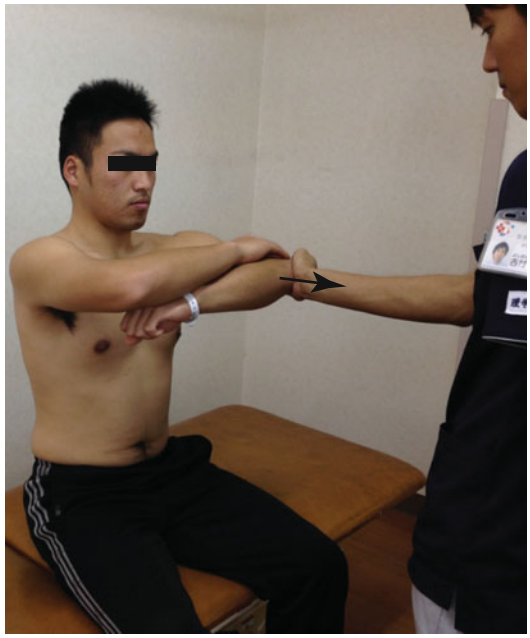

Fig. 9.4 Elbow push test for assessment of scapular stability. The elbow push test is performed with the shoulders in 90° of forward flexion. While grasping the contralateral elbow with each hand, the subject pushes each elbow in turn anteriorly with maximum force. The examiner resists this pushing by holding the elbow. The test is considered to be abnormal when the muscle strength on the dominant side is less than that on the nondominant side

spinous process of the thoracic spine is measured with the arms at the sides (Fig. 9.2). The reference point on the thoracic spine is defined as the nearest spinous process. A difference of more than 1.0 cm between the left and right sides is considered abnormal. To assess the scapular stabilizers, the elbow extension test and elbow push test are performed with the shoulders in 90° of forward flexion (Figs. 9.3 and 9.4). For the elbow extension test, the subject extends the elbow joint from 90° of flexion by using maximum force, while the examiner holds the subject's forearm to resist his extension force (Fig. 9.3). For the elbow push test, while grabbing the contralateral elbow with each hand, the subject pushes each elbow in turn anteriorly with maximum force as the examiner resists his pushing by holding the elbow (Fig. 9.4). Muscle strength is evaluated by manual muscle testing on a scale of 0–5. We assess the muscle strength of shoulder abduction with the subject's thumb up; this is known as the "full can position" [20, 25]. We measure external rotation strength with the subject's arm at his side [9]. To assess internal rotation strength, we record the subject's strength in lifting his hand off his back [12]. We consider the results of the elbow extension test, elbow push test, and manual muscle tests of abduction, external rotation, and internal rotation to be abnormal when the muscle strength on the dominant side is less than that on the nondominant side. To assess the posterior tightness of the shoulder joint, subjects perform the combined abduction test and horizontal flexion test while the examiner fix the scapula and prevent it from moving by holding it. The humerus is passively abducted in the coronal plane for the combined abduction test (Fig. 9.5) and horizontally flexed for the horizontal flexion test (Fig. 9.6). If the subject's upper arm fails to touch his head during glenohumeral abduction with a fixed scapula, the combined abduction test is graded as abnormal. The horizontal flexion test is considered abnormal when the subject is unable to reach around the other shoulder to touch the bed during horizontal flexion with a fixed scapula. Capsular laxity is evaluated by load-and-shift testing in the anterior, posterior, and inferior directions; anterior apprehension and relocation tests are also done. When the dominant side shows increased laxity or when the subject feels that the shoulder is unstable during any test, capsular laxity is considered abnormal. To evaluate subacromial impingement, we perform the Neer [31], Hawkins [14], and Yocum tests [24]. If the subject feels shoulder pain during any of these tests, subacromial impingement testing is graded as abnormal. The hyper-external rotation test (Fig. 9.7), which evaluates peel back of the superior labrum [4, 27, 28] and pathologic internal impingement [18, 26, 47], is performed in 90° of shoulder abduction with the elbow flexed at 90° in the supine position. The test is considered to be abnormal when a subject feels pain as the examiner applied external rotation torque beyond the maximum external rotation position. The number of "intact" results among the 11 physical examinations is recorded as the total

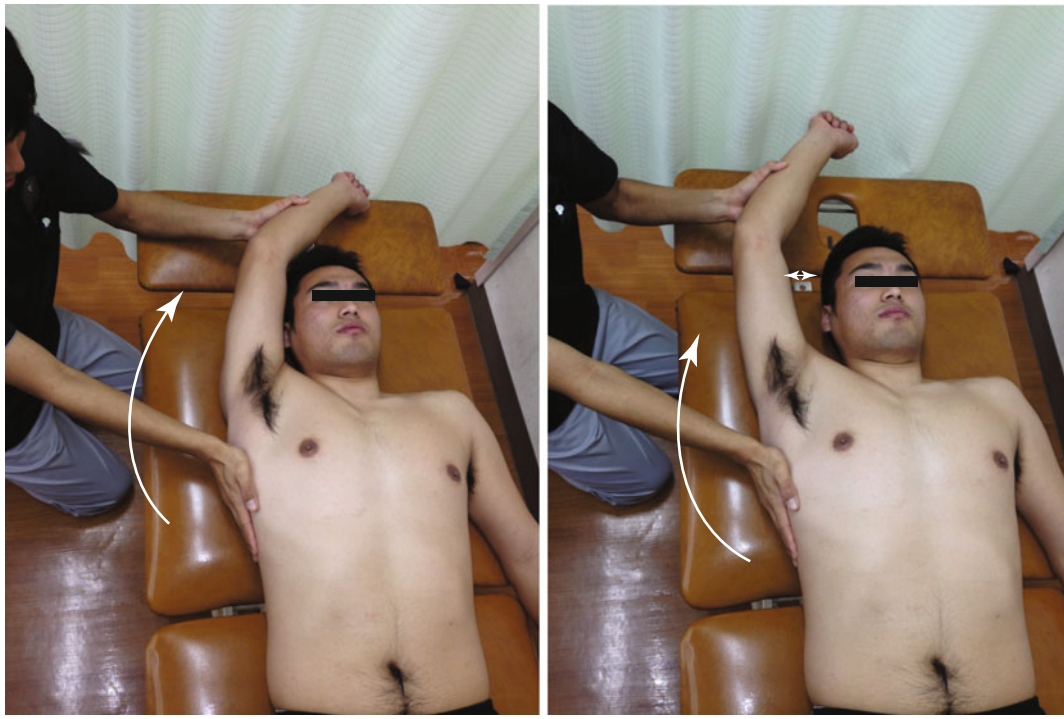

Fig. 9.5 Combined abduction test for assessment of posterior shoulder tightness. The examiner completely prevents any movement of the scapula by holding it. The humerus is passively abducted in the coronal plane. This test is considered abnormal when the upper arm fails to touch the head during glenohumeral abduction with a fixed scapula. *Left*: intact; *right*: abnormal

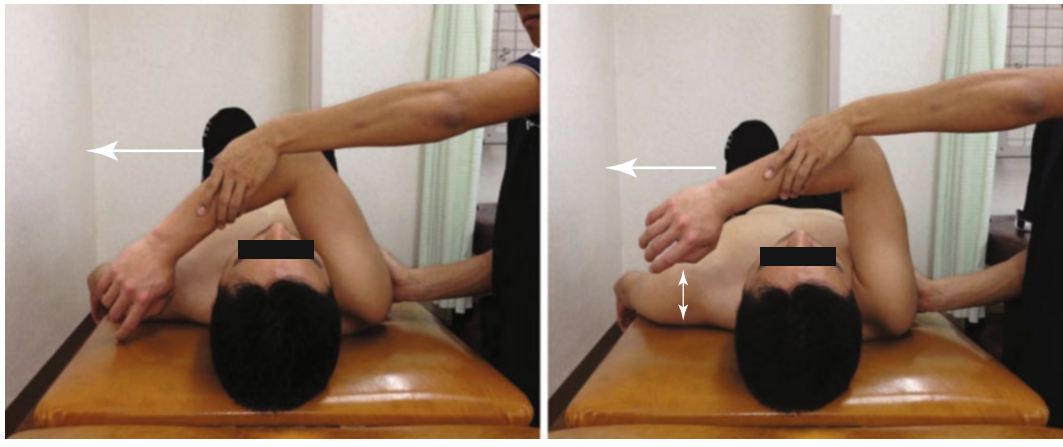

Fig. 9.6 Horizontal flexion test for assessment of posterior shoulder tightness. The examiner completely prevents any movement of the scapula by holding it and horizontally flexes the humerus. This test is considered to be abnormal when, during shoulder horizontal flexion with a fixed scapula, the subject is unable to reach around the other shoulder to touch the bed. *Left*: intact; *right*: abnormal

Hara test score for each subject. The maximum total score (11 points) represents all "intact" results (i.e., no abnormality found) for all tests; subjects with lower scores are considered likely to have a problem in the upper-extremity kinetic chain.

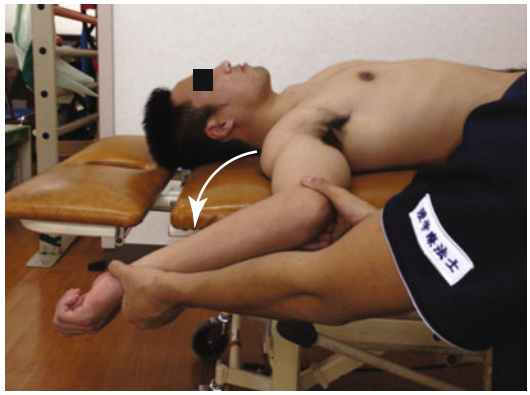

Fig. 9.7 The hyper-external rotation test, which evaluates peel back of the superior labrum and pathological internal impingement, is performed in 90° of shoulder abduction with the elbow flexed at 90° in the supine position. The test is considered abnormal when the subject feels pain as the examiner applies external rotation torque beyond the maximum external rotation position

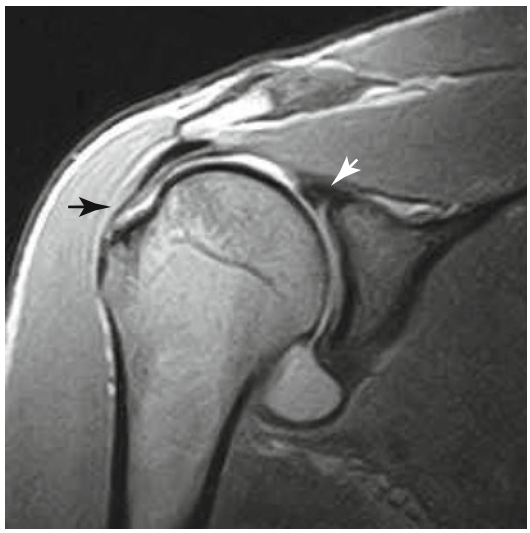

Fig. 9.8 Magnetic resonance arthrography of partial-thickness articular-sided rotator cuff tear (*black arrow*) and SLAP lesion (*white arrow*)

9.4 Essential Radiology

We prefer to use magnetic resonance arthrography to evaluate capsular laxity and labral injuries, as well as articular-sided partial-thickness rotator cuff tears (Fig. 9.8). Stress x-rays are also useful for assessing shoulder laxity. Bony deformities or defects in the glenoid or humeral head can be assessed accurately by using three-dimensional computed tomography.

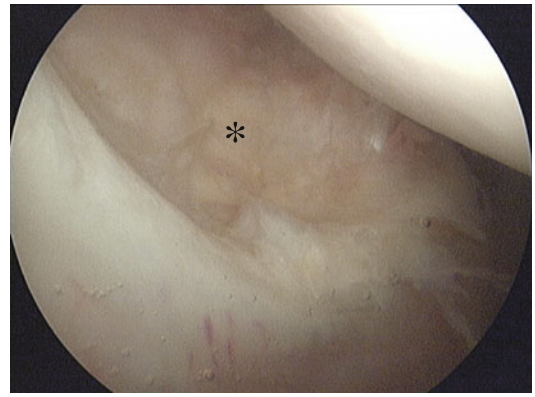

Fig. 9.9 Arthroscopic findings in partial-thickness articular-sided rotator cuff tear (*)

9.5 Disease-Specific Clinical and Arthroscopic Pathology

The main clinical symptom of articular-sided partial-thickness rotator cuff tears in throwing athletes is shoulder pain during sport. The shoulder pain is related to scapular dyskinesis, muscle imbalance (including imbalance of the rotator cuff muscles), posterior capsule tightness, and excessive anterior capsular laxity, as well as to the rotator cuff tear itself. In most patients with articular-sided partial-thickness rotator cuff tears (Fig. 9.9), a middle glenohumeral ligament tear (Fig. 9.10) or elongation and tearing of the anterior inferior glenohumeral ligament (Fig. 9.11) as well as a SLAP (superior labral tear from anterior to posterior) lesion (Fig. 9.12) and a thickened posterior capsule can be found during arthroscopy.

9.6 Treatment Options

Physical therapy is effective in most throwing athletes with articular-sided partial-thickness rotator cuff tears. When the scapular dyskinesis, muscle imbalance, posterior tightness, and increased

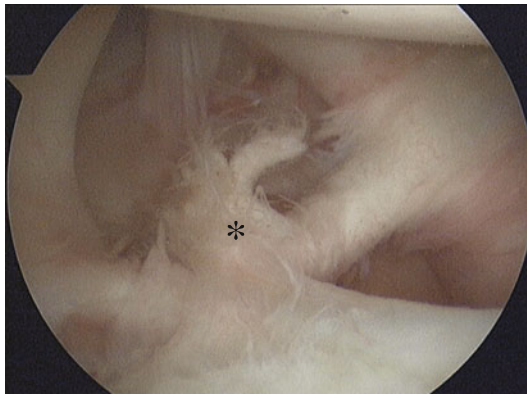

Fig. 9.10 Arthroscopic findings in middle glenohumeral ligament tear (*)

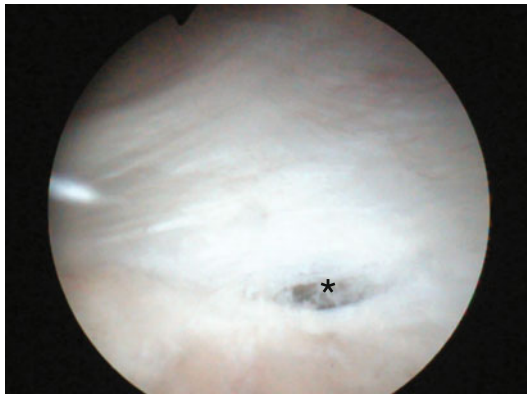

Fig. 9.11 Arthroscopic findings in tear and elongation of the anterior band of the inferior glenohumeral ligament (*)

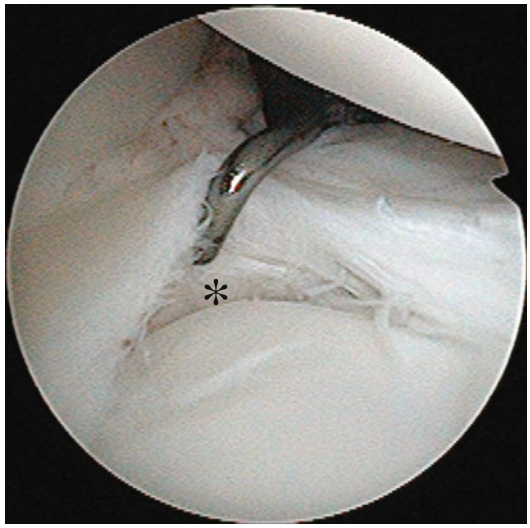

Fig. 9.12 Arthroscopic findings in a type II SLAP lesion (*)

anterior laxity are ameliorated with physical therapy, shoulder pain during throwing decreases or disappears in most cases. If physical therapy fails, surgical treatment typically is indicated. Various approaches, including debridement of partial-thickness tears with or without acromioplasty [3, 8, 21, 35, 38, 41], trans-tendon repair [6, 11, 15, 22, 39, 42, 46], or conversion to a full-thickness tear followed by repair [10, 11, 19, 37, 39], have been reported.

9.6.1 Debridement with or Without Acromioplasty

The superior shoulder capsule and undersurface of the supraspinatus and infraspinatus tendon insertions are evaluated for degenerative changes and debrided with a shaver until normal tendon is reached. Anterior acromioplasty and release of the coracoacromial ligament with debridement may be added. In patients with pathological shoulder laxity, which can cause shoulder symptoms, debridement alone is not a good option because it does not change shoulder laxity. However, for some throwing athletes who have no symptoms of pain associated with their pathological shoulder laxity or instability, debridement is a good surgical treatment and carries a low risk of postoperative shoulder stiffness.

9.6.2 Trans-tendon Repair

Snyder [40] proposed the use of an arthroscopic trans-tendon technique to treat partial-thickness articular-sided rotator cuff tears with the intention of restoring the medial footprint but preserving the tendon fibers remaining on the bursal side. Biomechanical studies have shown that trans-tendon repair results in higher footprint contact pressure and higher ultimate failure load compared with completion of the tear followed by repair [36]. Nevertheless, most clinical studies show that arthroscopic trans-tendon repair has a good outcome in terms of pain relief and shoulder score [6, 11, 15, 39, 42, 46].

9.6.2.1 Operative Technique

The frayed parts of the torn tendon are removed to expose good-quality tendon tissue. The footprint of the greater tuberosity is debrided to bleeding bone. A subacromial bursectomy is then performed to expose the bursal side of the torn tendon. The arthroscope is returned to the glenohumeral joint through the posterior portal, and a working portal is made anteriorly through the rotator interval. A spinal needle is passed percutaneously through the partial-thickness rotator cuff tear to identify the location for anchor insertion. One or two 4.5-mm suture anchors are then passed through the partial-thickness rotator cuff tear and screwed into the greater tuberosity under visualization from the glenohumeral joint. (In case of a longitudinal tear, a side-to-side suture repair is performed without using any suture anchor.) One suture limb is retrieved through the anterior portal. A 16-gauge spinal needle is passed through the anterolateral portal 5 mm medial to the intact margin of the torn rotator cuff tendon to guide the suture limb and one suture limb from the anterior portal pulled through the intact healthy portion of the rotator cuff. The other limb is passed through 5–10 mm posterior to the first suture limb in the same fashion to make a mattress stitch. The suture limbs are tied by using a non-sliding rotator cuff knot [29]. If a second anchor is used, its sutures are passed through and tied in the same fashion.

9.6.3 Conversion to Full-Thickness Tear Followed by Repair

Clinical studies have shown that repair of articular-sided partial-thickness rotator cuff tears after tear completion has a good outcome. Itoi and Tabata investigated the clinical results of 38 shoulders in 36 patients with incomplete rotator cuff tears that were repaired after tear completion [17]. The overall results were satisfactory in 31 shoulders (82 %). Deutsch prospectively evaluated the clinical outcomes in 41 patients who underwent arthroscopic repair after completion of partial-thickness supraspinatus tears [10]. Significant improvements were demonstrated in terms of American Shoulder and Elbow Surgeons scores, pain relief, and satisfaction. Forty patients (98 %) were satisfied with their outcomes. Kamath et al. also reported a high rate of patient satisfaction with arthroscopic repair after conversion to full-thickness tears [19]. Thirty-seven of 42 shoulders (88 %) had intact rotator cuffs (as seen on postoperative ultrasound), with improved American Shoulder and Elbow Surgeons scores and a 93 % patient satisfaction rate.

9.6.3.1 Operative Technique

Diagnostic arthroscopy is performed to assess the rotator cuff tendon and any associated pathology. Subacromial bursectomy is performed in all patients. Acromioplasty is limited to the removal of acromial spurs. Frayed rotator cuff tissue is debrided. A spinal needle is placed at the site of the articular-sided tear to identify its location from the bursal side. From the bursal side, the torn tendon is then checked with a probe. If the probe penetrates the residual tendon very easily, this means that the torn tendon cannot be repaired without tear completion because it is too thin and degenerated; the thin tendon tissues are therefore removed to expose the good tendon tissues. The remaining, stronger, torn tendons are then repaired by using a single-row, double-row, or suture bridge technique.

9.6.4 Rotator Cuff Repair in Throwing Athletes

Conway reported excellent clinical results of arthroscopic repair of a partial-thickness rotator cuff tear in nine baseball players [7]. Seven were professional players and two were college players. Eight players (89 %) returned to play at the same level or a higher one. However, most reports of rotator cuff repairs in professional baseball players have demonstrated a poor prognosis, with substantial difficulty in returning to preinjury levels of play [23, 30, 44].

Mazoue and Andrews reported that many pitchers were able to return to pitching with good velocity and control but fatigue early, so that they could pitch effectively for only a short period of

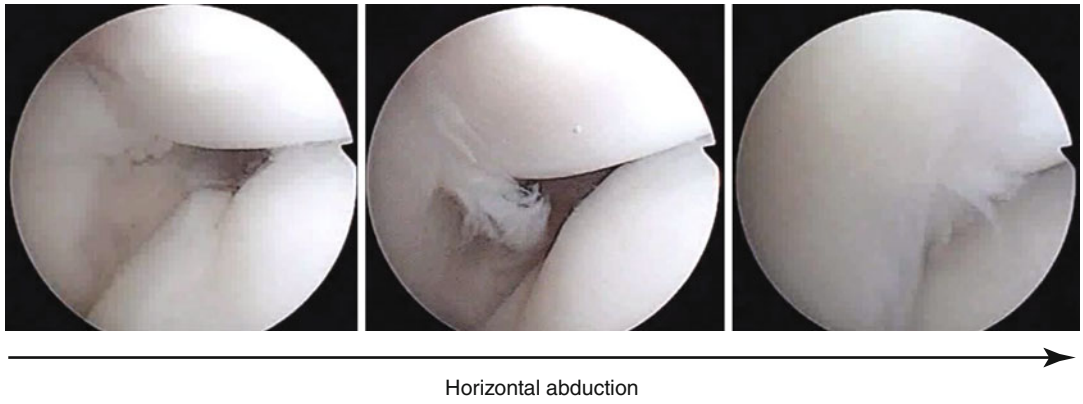

Fig. 9.13 Arthroscopic findings in internal impingement of the torn rotator cuff tendon

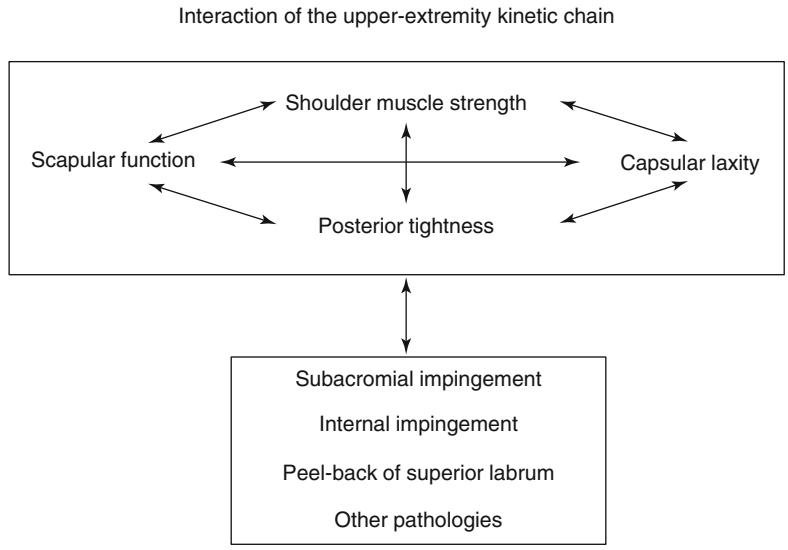

Fig. 9.14 Interactions in the upper-extremity kinetic chain

time [23]. Several complained of pain during competition, but most did not feel that the pain limited their ability to pitch.

9.7 Author-Preferred Treatment

Partial-thickness articular-sided rotator cuff tears are often seen in overhead-throwing athletes. Most tears are caused by shoulder internal impingement (Fig. 9.13). Scapular dysfunction, shoulder instability or pathological shoulder laxity, posterior shoulder tightness, and muscle imbalance are associated with pathological internal impingement (Fig. 9.14). Therefore, I use physical therapy to normalize the upper-extremity kinetic chain. Approximately 90 % of throwing athletes with partial-thickness articular-sided rotator cuff tears can obtain pain relief and return to their previous levels of sport. If the athletes cannot return to their sports, we will consider arthroscopic surgery.

When we treat these overhead-throwing athletes surgically, we need to evaluate their shoulder laxity and stiffness before surgery. If the patient has severe posterior tightness, the posterior labrum should be debrided without repair. If the patient has anterior capsular laxity, the anterior or superior labrum is repaired (Fig. 9.15). The choice of surgery for the torn rotator cuff

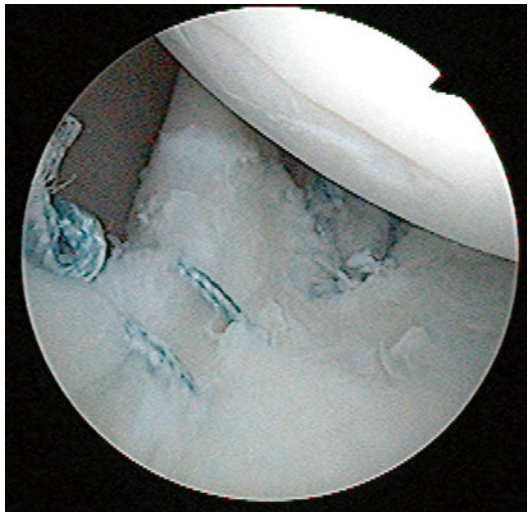

Fig. 9.15 Arthroscopic SLAP repair

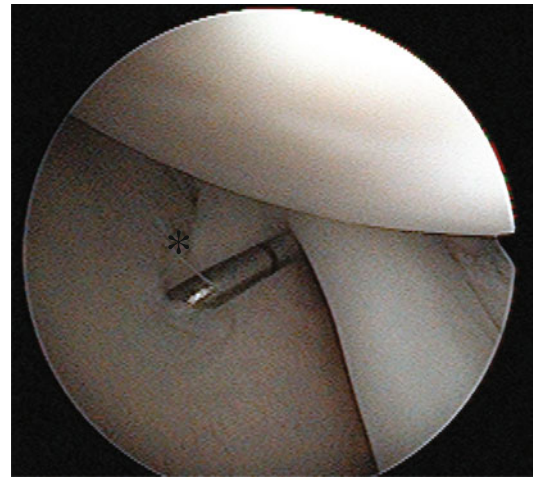

Fig. 9.16 Arthroscopic findings in partial-thickness articular-sided rotator cuff tear (*) in case 1

tendons is based on the tear thickness. If the tear constitutes less than 50 % of the thickness of the tendon, the tendon is debrided. If the tear constitutes more than 50 %, trans-tendon repair is considered in the case of young athletes (less than 30 years old) or in those with unstable shoulders. However, if the patient is a professional player and is 30 years or more, debridement is the method of choice.

9.8 Rehabilitation

Rehabilitation is the most important treatment for throwing athletes with or without surgical treatment. First, scapular function, capsule condition, muscle strength, and function of the trunk and lower extremity are evaluated very carefully. The Hara test (see Fig. 9.1) is useful for assessing the kinematic chain in throwing athletes. The rehabilitation protocol is based on the abnormalities found in the physical examination of each player. Most symptomatic players with partial-thickness articular-sided rotator cuff tears can recover with appropriate physical therapy.

The choice of postoperative rehabilitation is based on the choice of surgery. A range of motion (ROM) exercises are performed as soon as possible after the debridement of partial rotator cuff tears. Once ROM and muscle strength have recovered, a throwing program is started. In contrast, after rotator cuff repair with or without tear completion, the shoulder is immobilized for 3 weeks. Passive and active assistive ROM exercises are then started. Muscle strengthening starts 3 months after surgery if full ROM has been obtained. The throwing program starts 4 months after surgery.

9.9 Experience in Treatment of Athletes

Case 1

A college baseball player aged 18 years had severe shoulder pain during throwing. Arthroscopic trans-tendon repair of a partial-thickness articular-sided rotator cuff tear without using suture anchors (Figs. 9.16 and 9.17), along with SLAP repair, was performed after failure of 3 months of physical therapy. Eight months after surgery, the patient returned to competitive baseball as an infielder.

Case 2

A professional baseball pitcher aged 35 years had shoulder pain in the late cocking phase and acceleration phase of throwing. Although he was able to return to baseball after our physical therapy, he wanted to undergo arthroscopic surgery to

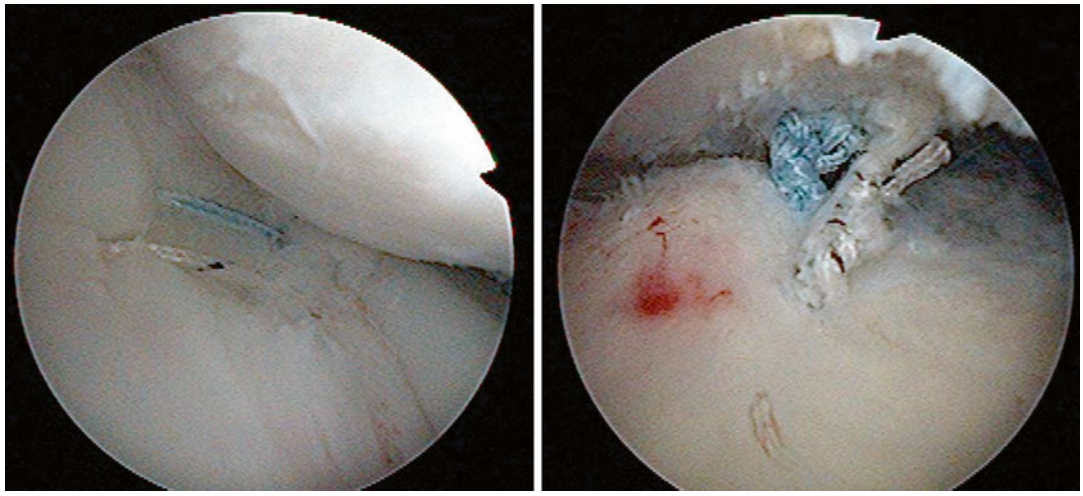

Fig. 9.17 Arthroscopic trans-tendon repair of a partial-thickness articular-sided rotator cuff tear, without the use of suture anchors, in case 1. *Left*: glenohumeral view; *right*: subacromial view

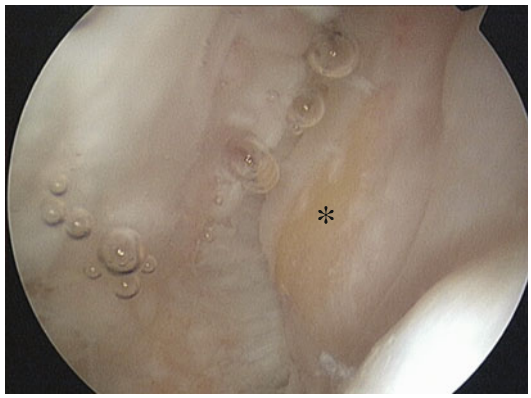

Fig. 9.18 Arthroscopic debridement of a partial-thickness articular-sided rotator cuff tear (*) in case 2

decrease his shoulder pain. After the season had ended, he underwent debridement of a SLAP lesion and partial-thickness articular-sided rotator cuff tear (Fig. 9.18). Ten months after surgery, he returned to professional baseball.

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SLAP Lesion: Part I. Pathophysiology and Diagnosis

10

Brian Grawe, Asheesh Bedi, and Answorth Allen

10.1 Pathophysiology

10.1.1 Anatomy and Function

The glenoid labrum is comprised of fibrocartilaginous tissue that surrounds the glenohumeral joint in a circumferential fashion [1, 2]. Anatomically, the labrum provides the glenoid attachment site for both the glenohumeral ligaments (GH) and the long head of the biceps [3]. Functionally, the glenoid labrum allows for improved stability of the glenohumeral joint by effectively deepening the osseous glenoid and enhancing the concavity-compression mechanism, thus limiting translation of the humeral head [4]. In the thrower's shoulder, this stabilizing effect becomes uniquely important, as biomechanical studies have demonstrated that tension within the bicipital–labral complex may improve the torsional rigidity of the abducted, externally rotated arm [5].

Histological investigation has revealed that the superior labrum demonstrates elastin fibers that are sparsely intertwined within a matrix [6], with a distinct fibrocartilaginous transition zone that links the hyaline cartilage of the glenoid to the more fibrous tissue of the labrum at the 12 o'clock position [1]. The superior labrum is typically triangular in appearance and may frequently have an inner free edge that partially covers the glenoid articular surface (meniscoid). The articular cartilage of the bony glenoid extends over its rim superiorly and medially. As a result, the superior labrum may attach to the glenoid rim or more commonly medial to the articular margin. This relatively medial attachment creates a subsynovial recess that can extend several millimeters medial to the glenoid edge (Fig. 10.1) [1, 6]. Several blood vessels arborize in both a radial and circumferential configuration to supply the peripheral labrum. These vessels include the suprascapular, circumflex scapular, and posterior humeral circumflex arteries. Similar to the meniscus of the knee, the innermost margin of the glenoid labrum is known to be avascular, and the anterosuperior quadrant demonstrates the poorest blood supply (Fig. 10.1) [1, 4, 7]. These facts, coupled with the superior labrum's relative mobile nature, make SLAP tears difficult to both diagnose and treat with modalities that result in reliable healing.

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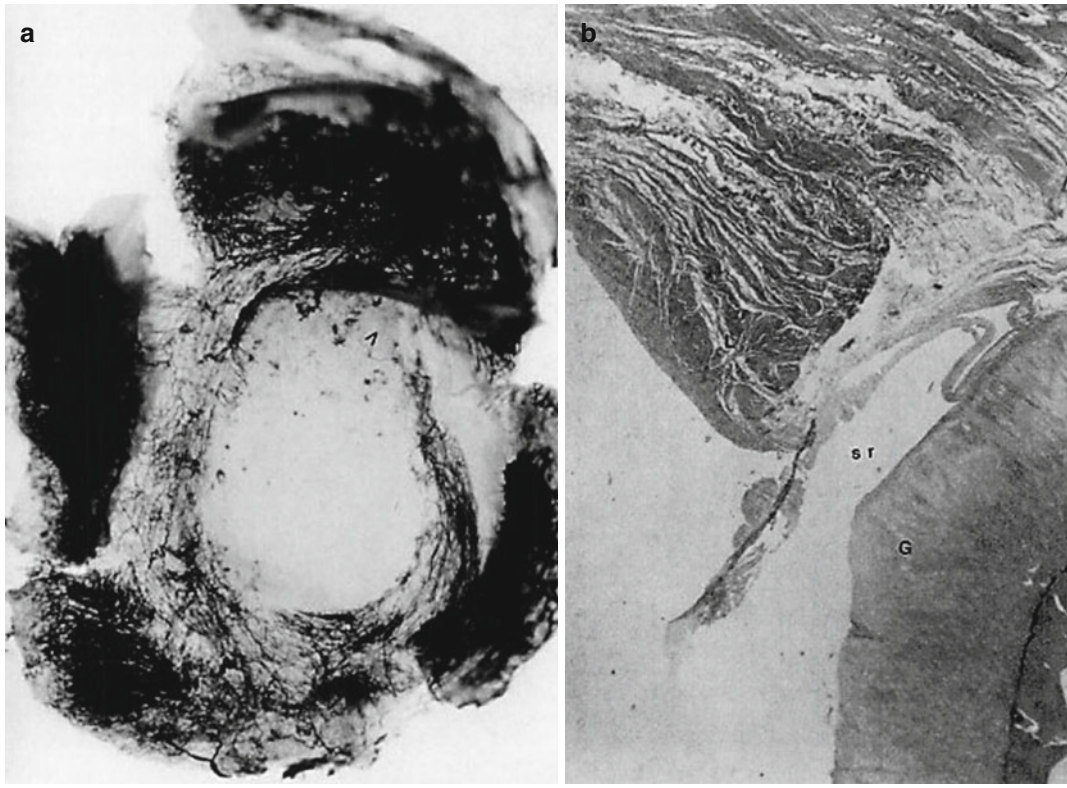

Fig. 10.1 (**a**, **b**) Demonstrates vascular architecture of the glenoid labrum, via a sagittal section of a human cadaveric specimen (**a**). Note the watershed zone in the anterosuperior region. A clear synovial recess (*sr*) is shown in (**b**), located between the labrum (*L*) and glenoid (*g*) (Adapted from Cooper et al. [1]. Reprinted with permission)

Numerous cadaveric and clinical studies have documented important distinct anatomic variations in both the biceps anchor attachment and the superior labrum, respectively. These variations must be appreciated when attempting to distinguish a "true" SLAP (superior labrum anterior posterior) lesion and further respected when providing appropriate treatment to pathology about the superior labral complex. Roughly 50 % of shoulders will demonstrate the biceps tendon originating from the supraglenoid tubercle, with the remaining half demonstrating its fibers originating directly from the superior labrum [7]. Furthermore, the majority of shoulders display a biceps with a posterior or posterior dominant glenoid labrum insertion, with only a minority of shoulders demonstrating an equal distribution of fibers inserting on the anterior and posterior aspect of the labrum [7, 8]. Grossly, the biceps anchor will usually demonstrate normal physiologic mobility, and overconstraint after repair may be an important factor leading to postoperative stiffness [6].

Normal anatomic variations to the superior labrum have also been reported with differing degrees of incidence, based on arthroscopic findings during surgery. Three distinct variants are classically described that included a sublabral foramen, a sublabral foramen with a cord-like middle glenohumeral ligament (MGHL), and an absent anterosuperior labrum with a cord-like middle glenohumeral ligament (Buford complex) [9, 10]. Aside from a sublabral "hole," the aforementioned sublabral recess may represent a potential space underneath the superior labrum adjacent to the biceps attachment site – which is also commonly associated with a cord-like MGHL [11]. These variations have been noted to occur in 13 % of the population, with the "Buford complex" being the least common [9]. From a clinical standpoint, these variations have been observed to play a role in the pathogenesis and predilection

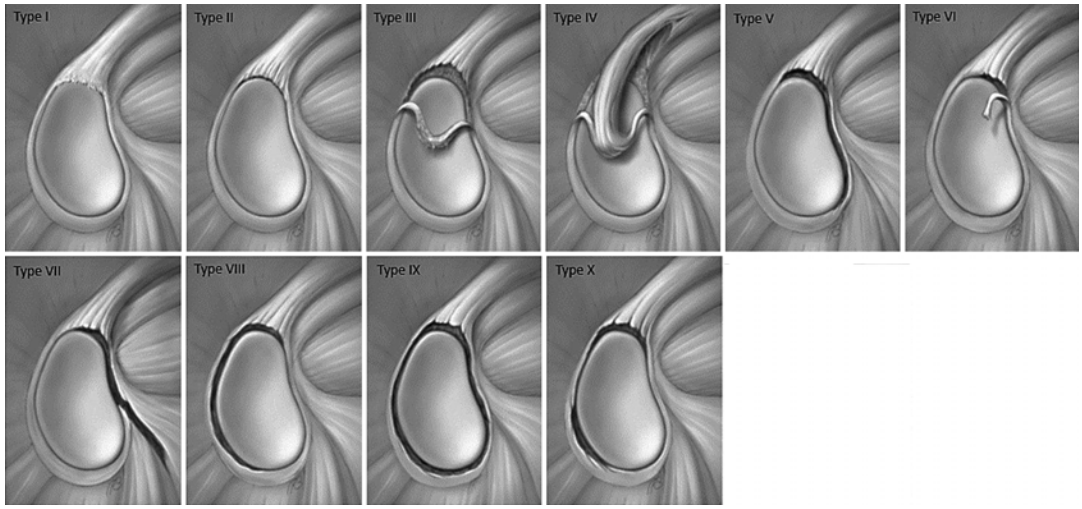

Fig. 10.2 Shows illustrations of the various SLAP lesions, based upon the original classification system (with modifications) (Adapted from Powell et al. [19]. Reprinted with permission)

for lesions about the superior labral complex and influence abnormal biomechanics about the glenohumeral joint. In a large prospective series of 546 patients, Kim and colleagues revealed that the presence of a superior labrum anatomic variant had a positive association with anterosuperior labral fraying, an abnormal superior glenohumeral ligament, and increased passive external rotation with the arm in abduction [12]. Furthermore, both the sublabral foramen variant and the "Buford complex" were shown to have an increase association with type II SLAP lesions [4, 12]. It is critical to recognize and understand the significance of these variants and ultimately distinguish them from pathologic lesions, as errant repair will often result in postoperative pain and stiffness, with an inferior clinical outcome [4].

10.1.2 Classification (Fig. 10.2)

Since the first description on a superior labral lesion near the biceps tendon origin in an overhead throwing athlete by Andrews and colleagues [13], numerous classifications systems have been developed to aid in the understanding and treatment of these injuries [14–19]. Snyder et al. coined the termed "SLAP" lesion to denote a tear of the superior labrum anterior posterior and reported an incidence of 6 % in over 2,000 shoulder arthroscopies [14, 15]. These authors also developed the most commonly utilized classification system with four distinct types of tears [14].

Type I lesions describe degenerative fraying of the superior labrum's free edge with an intact and stable biceps anchor. This particular entity is usually the result of age-related degenerative changes and should not necessarily be considered the primary pathology in patients with underlying shoulder pain [20]. Type II tears represent an unstable lesion in which the superior labrum and biceps anchor are detached for the glenoid rim – frequently this complex will be symptomatic and displaced into the glenohumeral joint. These lesions are reported to be the most common subtype, representing 41 % of SLAP tears in Snyder's original article [14]. A bucket-handle tear of the superior labrum with an intact biceps anchor represents a type III tear. Depending on the size and morphology (meniscoid superior labrum) of the torn labrum, mechanical symptoms may ensue, as the torn fragment will often displace into the joint. Type IV lesions represent a bucket-handle tear of the superior labrum that extends into the biceps anchor. Variable amounts of biceps tendon proper may be involved in the pathology, which may ultimately affect surgical management.

Type II lesions are often described as the most clinically relevant subtype based on their frequency [2, 6], and as a result Morgan and

colleagues developed a subclassification system for the type II SLAP tears [17]. These authors proposed tears to be further quantified based on location and extension of the tear, "A" being more anterior, "B" being more posterior, and "C" being a combined anterior posterior lesion. Furthermore, a type IIB lesion may develop posterosuperior instability with glenohumeral "pseudolaxity" [17]. Choi and Kim have also described a type II variant where destabilization of the superior bicipital–labral anchor complex is accompanied by a concomitant articular cartilage avulsion that can lead to loose bodies within the glenohumeral joint [18]. The so-called combined lesions have also been described and classified by Maffet and Powell, respectively [16, 19]. Maffet initially expanded upon the original Snyder classification system, noting that a review of his own patients demonstrated that only 62 % fit within the original schema. He describes type V tears as a Bankart lesion that extended into the superior labrum. Type VI lesions are denoted by a type II tear with an unstable labral flap, and finally type VII lesions represent tears that extend through the MGHL – resulting in an incompetent capsuloligamentous complex [16]. Powell further described types VIII through X, which involve a type II lesion with posterior extension, circumferential extension, or a concomitant posteroinferior labral disruption (reverse Bankart), respectively [19].

More recently, a myriad of studies have tried to enumerate the agreement between observers when diagnosing SLAP lesions, based upon Snyder's initial criteria, with varying results [21–23]. Gobezie et al. utilized video vignettes to establish inter- and intraobserver agreement for both the diagnosis and treatment of SLAP lesions. Findings of this study demonstrated considerable interobserver variability and only moderate intraobserver variability ($\kappa = 0.54$ & $\kappa = 0.45$) in regard to both treatment and diagnosis of a SLAP lesion. Furthermore, surgeons had difficulty distinguishing a normal shoulder from type I and II SLAP tears. Interestingly, arthroscopists were more likely to agree on treatment of the lesion, rather than how they would classify it based upon the Snyder criteria [21]. Jia and colleagues, in a similar study, were able to demonstrate improved intraobserver and interobserver agreement of SLAP tear diagnosis and classification ($\kappa = 0.67$ and 0.804), among experienced shoulder arthroscopists. Simplifying the labrums into normal or abnormal increased absolute agreement and intraobserver reliability, and utilization of the Morgan subclassification system did not affect the average correlation coefficient. Of note, quality of the video vignettes significantly affected the clinician's ability to make a confident diagnosis [22]. The lack of surgeons' ability to physically probe the labrum and perform arthroscopic impingement maneuvers ("peel off" of the labrum) has been cited as an intrinsic limitation of these studies [21, 22].

In a more clinically relevant study, Wolf et al. investigated the influence of multiple patient variables (via clinical vignettes) on the classification and treatment of superior labral complex injuries [23]. The variables included were age, sex, job activity, sports participation, and history/physical examination findings. Surgeons included in the study were part of the MOON (Multicenter Orthopedic Outcomes Network) shoulder group. Based on those surgeons surveyed, age, vocation, sporting activity, and physical examination findings were determined to be the most critical variables to affect treatment choices. These variables resulted in a treatment change 36 % of the time and a Snyder classification system change 28 % of the time [23]. Importantly, it must be noted that all these studies are colored by the fact that universal treatment standards do not exist for the various SLAP pathologies and that age and activity level often significantly influence the treatment algorithm, for the patient and surgeon alike.

10.1.3 Pathogenesis

Numerous etiologies have been proposed for the pathogenesis and underlying shoulder biomechanics responsible for the creation of a SLAP lesion. Frequently accepted mechanisms of injury include forceful traction loads to the arm, direct compression loads, and repetitive overhead throwing activities [6]. Acute traumatic injuries

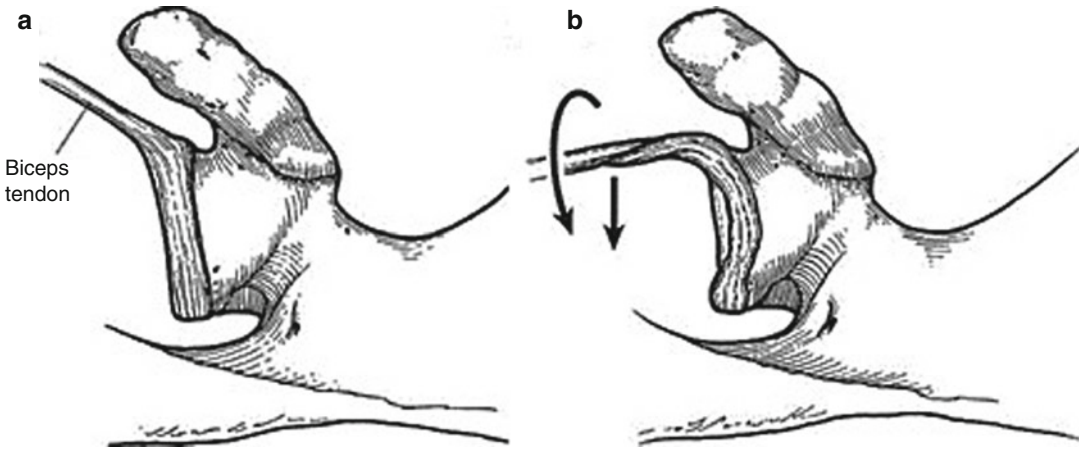

Fig. 10.3 Depicts the "peel-back" mechanism. Resting position of the biceps anchor viewed superiorly (**a**). The biceps will move posteriorly and twist at its base in the abducted and externally rotated position, resulting in labral "peel back" (**b**) (Adapted from Burkhart et al. [58]. Reprinted with permission)

may be responsible for a SLAP tear in a contact athlete – typically the result of a direct blow to the adducted shoulder [24]. Biomechanical studies have demonstrated that impaction loading to a forward flexed arm is more likely to produce an acute SLAP lesion, in comparison to the arm in the extended position [25]. Inferior traction injuries, weightlifters or fall while water skiing, have also been described as clinically and biomechanically culpable for the acute SLAP injury [16].

The overhead athlete usually presents as a distinct and unique patient population, at particular risk for the development of a SLAP lesion. Regardless of the precise mechanisms, lesions are the direct consequence of repetitive overhead throwing activities that occur as the result of overuse. The motions of hyperabduction and external rotation result in an increase in shear and compressive forces on the glenohumeral joint and ultimate strain on the rotator cuff and capsulolabral structures [26]. The dominant arm of young male high-performance overhead athletes appears to be most at vulnerable patient population [27]. The position of the shoulder has been shown to play a role in both biceps stability and injury pattern to superior labrum/biceps anchor. Although some controversy exists as to whether or not late cocking or the deceleration phase of throwing places the superior labrum at risk for injury, the biceps tendon insertion demonstrates 20 % less strength during late cocking [28]. Further biomechanical data that mimics throwing motion was only able to demonstrate increase strain in the superior labrum during the late cocking phase [29].

Specific to the throwing athlete, various anatomic and biomechanical factors may result in a predisposition to development of injury patterns in the superior labral complex. These athletes often develop a shift in shoulder range of motion, with an increase in external rotation, which can be accompanied with or without maintenance of total arc range of motion. These motion changes can be associated with bony changes, capsular changes, or both. This underlying phenomenon has been termed "GIRD" (glenohumeral internal rotation deficit) [26, 30, 31]. Wilk and colleagues have demonstrated that pitchers with a diagnosis of GIRD, based on physical examination, have an increased risk of shoulder injury. In this 3-year prospective study of 122 pitchers, those carrying a diagnosis of GIRD were twice as likely to develop a shoulder injury [32]. Numerous biomechanical mechanisms have been postulated to result in SLAP tears of the overhead athletes; Burkhart's [33] theory of a proposed "peel-back" mechanism is one such etiology (Fig. 10.3) [33]. This theory suggests that the inciting events of posterior and inferior glenohumeral capsular contractures lead to repetitive microtrauma in the

overhead athlete, with a relative posterior and superior shift of the humeral head during the cocking phase of throwing. Such a shift in glenohumeral kinematics then marks an increase in shear forces at the posterosuperior labrum. The biceps will then adopt a more vertical position that creates a "vicious cycle" where torsional forces are then generated at the posterosuperior labrum. This recurrent shear and torsional force combination at the bicipital–labral complex will then lead to a "peeling back" of the labrum toward the scapular neck [27, 33]. The sine qua non of such a lesion is the posterior capsular contracture, which must be addressed during the treatment phase with dedicated stretching.

A second proposed biomechanical mechanism for the production of a SLAP lesion is that of internal impingement. This theory implies that the superior labrum is subjected to shear and direct contact stresses in the late cocking position of throwing. The SLAP lesion is ultimately a result of impingement of the articular portion of the rotator cuff and posterosuperior labrum between the humerus and glenoid rim [34, 35]. The inciting event, however, appears to be subtle anterior shoulder instability, secondary to muscle fatigue or ligamentous injury. Such instability will allow the humeral head to shift anteriorly during abduction and external rotation (late cocking), and the aforementioned impingement ensues. Such a shift in glenohumeral mechanics has been corroborated in biomechanical studies mimicking anterior capsular laxity and concomitant posterior capsular contracture [36]. Champions of this model emphasize the need for treatment of the labral tear and anteroinferior instability.

A final method for the production of a SLAP injury is that of a "weed-puller" mechanism, initially described by McLeod and Andrews [13]. In this theory torsion produced by the long head of the biceps brachii tears the labrum away from the glenoid. Distinct from the other proposed mechanisms is that this particular theory suggests that the deceleration phase of throwing is the underlying culprit producing the SLAP tear. This theory was first developed on the basis of biomechanical cadaveric data that showed peak biceps muscle activity during the deceleration motion [37].

It is imperative for the clinician to evaluate the athlete as a whole when attempting to discern the underlying etiology of the SLAP lesion. Throwing requires a complex series of coordinated movements that ultimately transmit large amounts of energy from the lower trunk to the arm – the so-called kinetic chain [38]. Alterations in this cascade can result in motions and stresses that injure the labrum. In a similar fashion the role of the scapula and its overall contribution to shoulder motion and the kinetic chain must also be respected. The scapula's synchronized relationship with the humerus allows for a stable center of rotation of the glenohumeral joint. The overhead thrower can become susceptible to scapular dyskinesis, which may eventually lead to the "SICK" scapula (scapular malposition, inferior medial border prominence, coracoid pain, malposition, and dyskinesis of scapular movement) [39]. This abnormal position of the scapula can lead to abnormal kinematics of the glenohumeral joint and pathologic stress across the labrum, ultimately leading to disability of the throwing shoulder.

10.2 Diagnosis

10.2.1 History

The clinical diagnosis of a SLAP tear can often pose a challenge, even to the most experienced surgeon. Patients will often display concomitant pathology of the shoulder, based on preoperative history, physical exam, and imaging, with symptoms consistent with an insidious onset of nondescript pain. A thorough history, including the mechanism of injury, must be elicited, as the significance of a SLAP lesion, even at the time of surgery, can often be unclear.

Pain is the most common clinical complaint and is usually located anteriorly. Athletes will associate the pain with athletic impairment, including loss of throwing velocity or difficulty with overhead motions [27, 40]. In the overhead athlete mechanical symptoms can predominate, and the sensation of catching, popping, or clicking will be present with rotational movements.

Symptoms of weakness and instability may be the result of other underlying pathologies such as partial-thickness rotator cuff tears, capsulolabral injuries, biceps tendinopathy, and internal impingement [41]. Weakness should be carefully evaluated, as it may be the result of a ganglion cyst formation and compression of the suprascapular nerve. Additionally, "dead arm syndrome," although typically associated multidirectional instability of the glenohumeral joint, has also been described in athletes with SLAP tears [42].

10.2.2 Physical Examination

The physical examination of the athlete, in the face of a potential SLAP lesion, should commence with assessment of both glenohumeral and scapulothoracic motion of the affected shoulder. It is imperative that glenohumeral range of motion be assessed with the scapula stabilized and compared to the contralateral extremity. As previously discussed overhead throwing athletes often exhibit findings consistent with GIRD, defined as a deficit of internal rotation of at least 20° of glenohumeral motion when compared to the contralateral side [26]. As a result, shoulder rotation must be evaluated in adduction and 90° of abduction and should be performed in the supine position to assist with scapular stabilization. If two examiners are available, one can stabilize the scapula by placing a hand over the coracoid and acromion while the other measure the arc of motion. Alternatively, these maneuvers can be successfully performed with a single examiner as well. Judicious evaluation of shoulder stability must also take place, as combined lesions of anterior capsulolabral structures are not uncommon in the overhead athlete. Anterior instability can be assessed, with maintenance of the supine position and utilization of the load shift and apprehension relocation testing. It is also very important to evaluate for possible inferior and posterior instability, with the sulcus sign and posterior apprehension or jerk testing, respectively. Manual rotator cuff strength testing must also be documented, as these muscles function as important dynamic stabilizers of the glenohumeral joint.

Aside from careful inspection of the glenohumeral joint, a thorough exam of the scapulothoracic joint must also take place, to allow for a complete evaluation of the athlete with a suspected SLAP lesion. Scapular asymmetry can be noted in the resting position, and dynamic evaluation, by having the patient forward elevate their arms, can effectively demonstrate the presence of winging. Throwing athletes will often demonstrate a protracted scapula as a normal adaptation; however, this alteration has been reported to lead to other shoulder injuries, namely, dynamic outlet impingement and tears of the rotator cuff [4, 43]. The SICK scapula and scapular dyskinesis must also be ruled out. The SICK scapula will appear lower with a prominent inferior medial border, compared to the contralateral side. This position will allow the coracoid to tilt inferiorly and tighten the pectoralis minor and biceps – as a result, the athlete will complain of coracoid pain and posterior scapular pain. Scapular dyskinesis, on the other hand, can be determined by comparing side-to-side scapular kinematics – forward elevation is most often the motion of interest that will reproduce the dyskinesis [3]. If significant dynamic winging is present, the proximal or distal etiology must be ascertained and treated appropriately.

Numerous special tests have been described in the literature to detect the presence of pathologic injury to the superior labrum/biceps complex. These tests vary in terms of accuracy and reliability for the correct diagnosis of a SLAP lesion, and a combination of these tests may prove more useful than one single maneuver [3]. Current tests include the O'Brien active compression test, anterior slide test, compression rotation test, resisted supination external rotation test, the speed test, crank test, biceps load tests I and II, and major shear test [44–49]. Numerous authors have investigated the clinical utility of these tests, in terms of sensitivity and specificity, and no single test reaches the same diagnostic value as its original authors first reported. In fact, Cook et al. recently conducted a prospective case-control to test the diagnostic accuracy of 5 such tests (O'Brien active compression, biceps load test II, labral tension test, O'Driscoll/dynamic labral

shear test, and speed test) for the detection of a SLAP lesion. The authors ultimately concluded that none of the 5 tests, either stand-alone or clustered together, provided any diagnostic value for detection of a SLAP tear [50]. Recent evidence, put forth by a meta-analysis, does suggest that the active compression test is the most sensitive and predictive for ruling out a superior labral tear, followed by the crank and speed test [51]. A concise review of multiple tests for distinguishing SLAP lesions, with reported test performance, has been adapted from a recent systematic review [52]. The overarching theme is that the diagnosis of SLAP can be difficult, and the clinical examination, alone, cannot be solely relied for an accurate diagnosis.

10.2.3 Imaging

Further assessment of an athlete with a suspected SLAP tear, with appropriate imaging, should begin with high-quality orthogonal radiographs of the shoulder. Standard views include a true AP of the glenohumeral joint, a scapular AP, an axillary, and an outlet. Roentgenograms do not have specific findings consistent with a SLAP tear; however, they may rule out other pathologies responsible for the patient's shoulder pain. Common coincident pathology that must be ruled out includes acromioclavicular abnormalities, outlet impingement, or the presence of a Bennett lesion (mineralization of the posterior band of inferior glenohumeral ligament) – commonly seen in athletes diagnosed with GIRD.

Magnetic resonance imaging (MRI), with or without arthrography, remains the gold standard for the advanced imaging of a shoulder with a suspected diagnosis of a SLAP tear. Furthermore, MRI can be very useful for the evaluation of other concomitant conditions associated with superior labral tears, including tendinosis or frank tearing of the undersurface of the rotator cuff and spinoglenoid notch cysts. It is imperative that multiplanar images, in the axial, coronal, and parasagittal plane, be obtained. Recent evidence suggests that images obtained with the affected arm in the abducted externally rotated position (ABER) may improve diagnostic accuracy by mimicking the "peel-back" effect on the labrum. Borrero and colleagues recently investigated this technique in patients with comparison of MRI findings to arthroscopic findings, and the ABER position improved diagnostic accuracy in the subgroup of patients who were overhead athletes [53]. It must be noted that discriminating discrete SLAP tears, from the previously mentioned anatomic variants associated with superior bicipital–labral anchor complex, can be difficult. It is imperative that MRI findings always be correlated with subjective findings reported in the history and objective findings gleaned from the physical examination. MRI findings that should alert the physician of a possible SLAP tear include high signal intensity and/or intra-articular contrast extension under the superior labrum/biceps origin, laterally curving high signal intensity in the superior labrum (represented as a deep cleft between the superior labrum and glenoid), fluid extravasation between labral fragments, and anteroposterior extension of high signal intensity at the biceps root [6]. Coronal and axial sequences will be most germane for these respective findings.

Despite recent advances and sophistication in MRI technique, the diagnostic precision of a SLAP tear remains variable in the reported literature. In comparison with arthroscopic findings, conventional MRI has a reported sensitivity, specificity, and accuracy range of 84–98 %, 63–91 %, and 74–96 %, respectively [54–57].

Conclusion

The diagnosis of a clinically significant SLAP tear remains a challenge. Correlating mechanism of injury, based on a thorough history, with discrete physical exam findings will improve diagnostic accuracy. Furthermore, appropriate MR imaging techniques can alert the clinician of other concomitant pathology and aid in the diagnosis of a SLAP tear. It is necessary that surgeons be aware of the clinical significance of all anatomic variations that may be present at the superior labral biceps complex, and errant treatment can be a common source of postoperative pain and stiffness.

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11 SLAP Lesions Part II: Acute Lesion Versus Chronic Lesion Resulting from Repetitive Motion (or Microtrauma)

Lennard Funk and Puneet Monga

11.1 Introduction

Since the original description of labral tears near the biceps origin by Andrews et al. [1], SLAP tears have been an increasingly recognized source of shoulder pain and dysfunction, and the frequency of SLAP repairs performed by shoulder surgeons has been increasing progressively [16]. The term "SLAP tear" was coined by Snyder et al. [14] following a review of 700 shoulder arthroscopies. The diagnosis, pathophysiology, and management of SLAP tears caused by two distinct mechanisms of an acute versus chronic injury causing SLAP tears are vastly different. This chapter aims to compare and contrast these two broad categories of patients with the diagnosis of SLAP tears.

11.2 Pathoanatomy, Biomechanics, and Classification

Snyder et al. [14] described two discreet mechanisms of injury causing SLAP tears. A superior compression mechanism results in an acute traumatic superior compression force to the shoulder, usually due to a fall on the outstretched arm, with the shoulder in the abducted and slightly forward-flexed position. The second mechanism of acute SLAP tears has been described as an inferior traction mechanism, which involves a sudden, traumatic inferior pull on the arm. It is not uncommon for such forces to cause associated injuries, such as anterior and posterior labral tears and rotator cuff tears. The third mechanism, which is commonly observed in practice, is association of acute SLAP tears with shoulder dislocations or subluxations. Such injuries are commonly seen in contact sports, such as rugby [8]. Commonly the SLAP tear in such a scenario extends into either the anterior or posterior labrum. An acute SLAP tear can also be explained by a deceleration injury causing traction along the length of the biceps tendon, resulting in a SLAP tear, especially during the follow-through phase of the throwing action [1] (Fig. 11.1).

Chronic SLAP tears from repetitive microtrauma are commonly seen in overhead sporting activities [6]. The pathophysiology of chronic SLAP tears has been much debated, and various mechanisms have been proposed. All these mechanisms are centered around a repetitive overhead motion of hyperabduction and external rotation, which produces large forces and increased shear and compression forces on the glenohumeral joint and strain on the rotator cuff and capsule-labral structures [5]. One of the suggested pathophysiology is alteration in the kinetic

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Fig 11.1 The mechanisms of injury

chain. Throwing requires a complex series of coordinated motions resulting in the transmission of large forces from the legs, the core, via the shoulder, onto the arm and hand. If such coordinated effort of the "kinetic chain" is altered, such alteration can lead to abnormal forces, leading to injury to the labrum or the rotator cuff [11]. An overhead athlete is particularly predisposed to such alterations due to the "thrower's paradox," i.e., a thrower must possess sufficient laxity to allow excessive external rotation, yet sufficient stability to prevent glenohumeral joint subluxation [15]. It is commonly noted in overhead athletes that the range of external rotation is increased and the internal rotation is correspondingly decreased [2]. This pattern is termed as glenohumeral internal rotation deficit or GIRD. Such changes, although advantageous to an elite training athlete, may predispose the athlete to labral injuries (Fig. 11.2).

Fig 11.2 Image showing a muscle model in the extreme overhead position of a baseball pitch, with arching of the back and hyperextension of the shoulder. Kinetic chain – throwing requires a complex series of coordinated motions resulting in the transmission of large forces from the legs, the core, via the shoulder, onto the arm and hand

Another patho-mechanical explanation of a chronic SLAP tear may well be as a result of contracture of the posterior shoulder capsule, resulting in a relative posterior superior migration of the humeral head [5]. Such a posterior contracture appears with repetitive overhead external rotation and can result in a secondary posterior superior position of the humeral head. This, in turn, increases the shear forces across the glenohumeral joint, leading to internal impingement. Burkhart et al. [5] also described the "peel-back" mechanism, which may potentially explain chronic SLAP tears. During the abduction and external rotation of the shoulder, torsional forces in the biceps and labrum increase as the arm

Fig 11.3 The peel-back mechanism in a throwing athlete with tight posterior capsule

moves to this extreme position, leading to a twist at the base of the biceps, which transmits such a force onto the labrum, resulting in a "peel back." Furthermore, in athletes with such altered mechanics of posteroinferiorly contracted capsule, a "peel-back" mechanism is exacerbated if the position of the scapula is protracted and laterally rotated (Fig. 11.3).

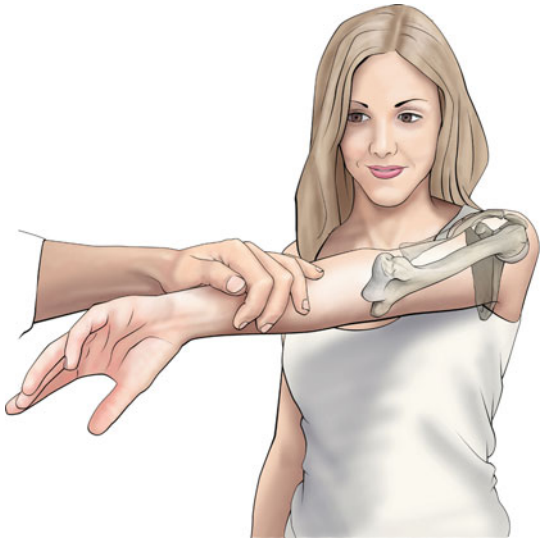
It is likely that all the above mechanisms have a role to play in the pathophysiology of the chronic SLAP tear seen in an overhead athlete. Snyder et al. [14] developed an arthroscopic classification for SLAP tears, which is common to both acute traumatic SLAP tears and chronic SLAP tears caused by repetitive micromotion. Various modifications to the original SLAP classification by Snyder et al. [14] have been proposed, notably type 5, where a Bankart lesion extends into the superior labrum and biceps anchor, and type 8 with a posterior labral extension into a 6 o'clock position. Such type 5 and type 8 lesions are commonly seen in acute SLAP tears, resulting from a concomitant shoulder dislocation, as indeed the type 9 (more severe labral tears with circumferential involvement). SLAP tears, in contrast, as a result of chronic repetitive overhead motion, would classically lead to a type 2 SLAP tear. Such type 2 SLAP tears have been further classified into type 2A, B, and C [13]. The most frequent lesion seen in chronic SLAP tear is a type 2B SLAP tear.

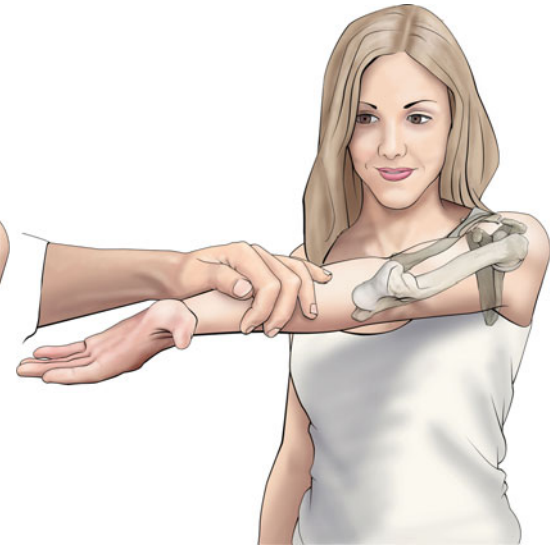
11.3 Clinical Presentation and Physical Examination

Patients presenting with an acute SLAP tear give a history of sudden onset pain following an episode of fall, traction force along the arm, a throwing action, or a dislocating force. They commonly report pain in the anterior aspect of the shoulder and describe this as "deep." This pain can sometimes be associated with a click and is commonly exacerbated by forward flexion and internal rotation and recreation of the throwing action. The athlete commonly reports sudden pain, a popping sensation, and loss of function.

Chronic SLAP tears resulting from repetitive overhead action commonly present with insidious onset pain, which is usually noted at the time of abduction, external rotation, or the late cocking phase of throwing. Such symptoms are usually located along the biceps tendon or posterosuperiorly. However, overhead athletes may report a "dead arm" [11] with a loss of power during throwing. Patients may also present with rotator cuff insufficiency, especially in internal impingement, where rotator cuff tears are commonly associated with such SLAP tears.

Physical examination begins with assessment of core stability, scapular kinematics, and assessment of range of motion. A glenohumeral internal deficit (GIRD) should specifically be sought, and tests for shoulder instability and rotator cuff strength should be performed. A wide variety of physical examination techniques have been described to clinically diagnose SLAP tears. Such tests include the O'Brien's active compression test, anterior slide test, biceps load test, dynamic labral shear test, and labral tension test. None of these tests, however, are diagnostic of SLAP tears, either as stand-alone or in combination [12] (Figs. 11.4 and 11.5).





Figs. 11.4 and 11.5 Assessment of gird – see here – https://www.dropbox.com/s/kbdfkijx3qgg9y3/GIRD%20swimmer.wmv. O'Brien's test: resistance is tested with the arm forward flexed to 90° and adducted 10° with the thumb pointing down (**a**) and subsequently with the thumb pointing up (**b**)

11.4 Imaging

The gold standard for diagnosing a SLAP tear, acute or chronic, remains MRI arthrography. In this technique contrast is injected into the glenohumeral joint under radiographic control and leads to controlled distension of the glenohumeral joint. Seepage of contrast under the labrum is confirmatory of a SLAP tear. It has been suggested that MRI arthrography in the ABER (abduction, external rotation) position can recreate the posterior superior labral "peel back," hence increasing the detection of SLAP tears [4]. It is, however, extremely important that the findings of such sensitive investigations are interpreted and analyzed with caution. It is not uncommon for athletes to have MRI abnormalities [9]. Clinical correlation of MRI arthrography should be carried out alongside the clinical findings to avoid overdiagnosis of such lesions as the presence of sublabral recess can lead to false-positive reports.

Although standard radiographs are not useful in diagnoses of SLAP tear, they are commonly performed to exclude associated bony injuries and concomitant pathology such as A-C joint disease, fractures, and extrinsic subacromial impingement (Fig. 11.6).

11.5 Arthroscopic Pathology

The surgeon should be aware of the common anatomical labral variance of the superior sublabral recess, sublabral foramen, and Buford complex [10] in order to avoid false-positive diagnosis of SLAP tears. The Snyder classification for SLAP tears is commonly used to describe superior labral tears, with the essential pathology being disruption of the superior labral tissue from the underlying hyaline cartilage. Acute SLAP tears are commonly type 2, type 5, type 8, or type 9. However, all forms of labral injury can be associated with such acute traumatic labral tears. Chronic SLAP tears are commonly associated with type 2B lesions. The superior labral lesions as a result of overhead repetitive activity commonly appear frayed. Such chronic tears may well be associated with partial-thickness rotator cuff tears. It is useful to perform intraoperative "peel-back" mechanism where the arm is taken

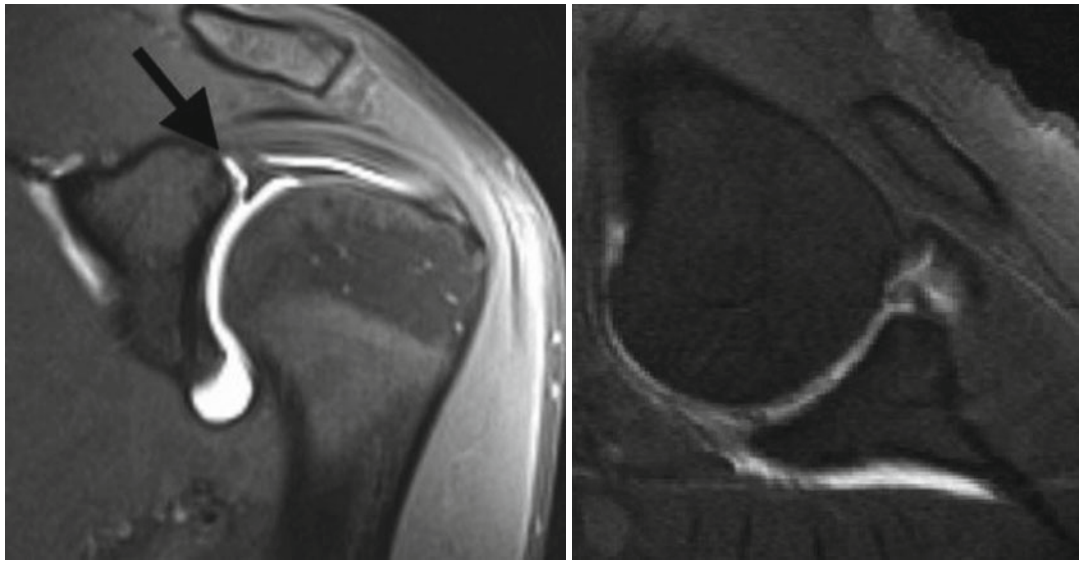


Fig. 11.6 MRA of SLAP in neutral and ABER position

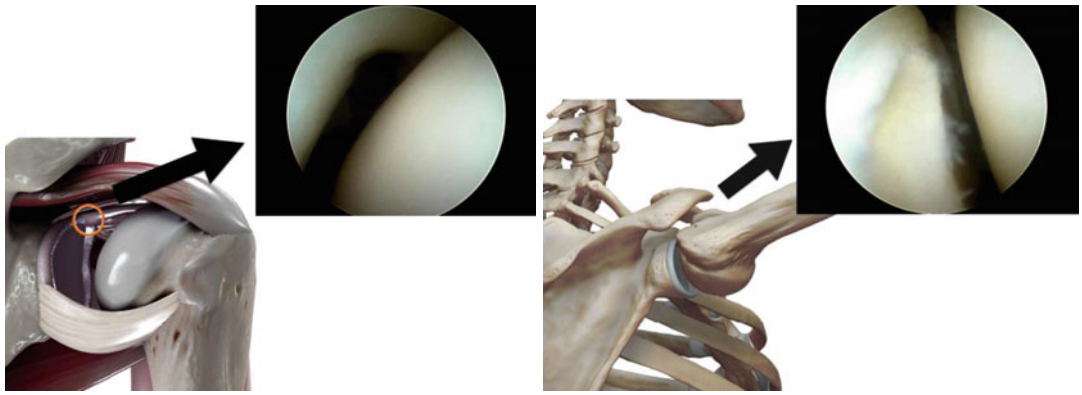


Fig. 11.7 Arthroscopy images of peel back

into a position of abduction and external rotation and the superior labrum closely observed for instability. This maneuver is useful in demonstrating the labrum peeling off the posterior superior aspect of the glenoid. Visual demonstration of "peel back" leading to exposure of the underlying glenoid bone would prompt the surgeon to perform labral repair [5]. A systematic examination of the joint should also include examination of the rotator cuff, the rest of the labrum, and, in particular, the status of the biceps tendon (Figs. 11.7 and 11.8).

11.6 Treatment Options

It is important to differentiate acute from chronic SLAP tears, primarily because the treatment for these two conditions remains significantly different. For an acute SLAP tear in an athlete, with or without association of a dislocation, early surgery is warranted; a chronic SLAP tear can usually be managed without surgical intervention in most cases.

An acute symptomatic SLAP tear is best managed with early arthroscopic repair. Such

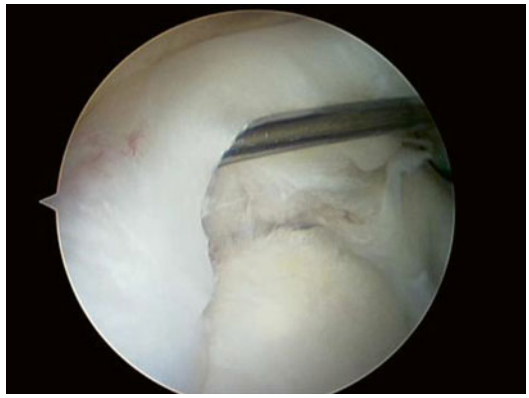


Fig. 11.8 SLAP lesion at arthroscopy – lifting off with the probe

treatment involves identification and confirmation of the clinico-radiological diagnosis intraoperatively. This superior labrum tear is repaired using suture anchor/s, along with management of any other concomitant pathology. The principle of early repair is to restore anatomy and to optimize the athlete for an early return to sports.

Management of chronic SLAP tears, on the other hand, should be with a trial of nonoperative treatment initially. Nonoperative treatment for overhead throwing athletes in the form of initial rest, followed by appropriate rehabilitation, can result in return to sports in two-thirds of the cases [7]. Specific deficiencies identified during physical examination, especially with regard to core stability, scapular dyskinesis, and GIRD, should be addressed during this phase of nonoperative care.

Operative treatment for chronic SLAP tears is reserved for an overhead athlete where symptoms persist despite good rehabilitation efforts. Even in the absence of typical MRI arthrogram findings, a strong clinical suspicion and positive provocative tests are sufficient to justify arthroscopic management of such injuries. Treatment of a chronic SLAP tear is either in the form of a SLAP repair or biceps tenodesis. Even though arthroscopic repair is the preferred treatment option for younger patients with good quality biceps tendon [12], biceps tenodesis has been shown as a reliable alternative, with a higher proportion of patients returning to sports in one report [3]. Biceps tenodesis may be used in a revision situation, in patients with associated biceps tendon damage, or based on surgeon preference.

11.7 Authors' Preferred Treatment

In our experience in contact athletes, the SLAP tear is usually associated with other injuries requiring treatment, such as Bankart tears, rotator cuff tears, and posterior labral tears. Isolated SLAP tears are uncommon in these patients. Our preferred clinical tests include the O'Brien's test and dynamic labral shear tests; however, the absence of positive findings does not exclude a SLAP tear. The history is a more important indicator of a probable SLAP tear. An MRI arthrogram with ABER views is obtained as the imaging of choice. If the patient is unable to return to sports and training, early arthroscopic assessment is preferred, commonly culminating in a repair of the superior labrum using suture anchors. The repair of associated injuries, such as labral tears and rotator cuff tears, is carried out at the same time as the repair of the superior labrum.

We prefer to use a single suture anchor with the suture posterior to the long head of biceps tendon. In our experience, the anterior suture adds little to the stability of the repair and can tighten the rotator interval or a high middle glenohumeral ligament leading to postoperative stiffness. For tears extending posteriorly we will add a second posterior suture anchor via a posterolateral portal. Following repair, we like to perform an arthroscopic peel back to ensure labral stability.

Chronic SLAP tears commonly present with posterosuperior joint pain and loss of power during overhead play. The clinical examination is carried out to assess core stability, scapular kinematics, and, in particular, assessment of GIRD. These features are specifically identified,

and the involvement of a specialist shoulder therapist is instituted in the treatment, alongside the team therapist. This is to ensure the complex scapula issues are managed with the sport-specific rehabilitation simultaneously. Indications for surgery include persistent labral symptoms and signs with a failure to progress with therapy and to return to sports.

A careful examination under anesthesia is performed, specifically looking for any differential capsular laxity or tightness compared to the opposite shoulder. A "peel-back" test is commonly performed intraoperatively to confirm the diagnosis. Any associated significant anterior capsular laxity and anterior labral tears are managed with small capsular shift and labral repair. Associated partial-thickness rotator cuff tears are debrided. If the humeral bony insertion footprint of the rotator cuff is exposed by the tear, the rotator cuff is repaired. Biceps tenodesis is reserved for failed SLAP repairs, biceps tendon involvement (e.g., SLAP type 4), or in older patients (generally over 40 years of age).

Postoperatively full active range of motion can commence under the guidance of the therapist. Any resistance activities and forced overhead movements are avoided for 6 weeks following surgery.

11.8 Rehabilitation

The phases of rehabilitation for SLAP repair are similar in both acute and chronic tears. However, acute SLAP tears usually are accelerated through the rehabilitation stages, with the desire for early return to sports, as the sports demands of contact athletes are not quite so specific for repetitive overhead activities. In a chronic SLAP tear, it is also important to continue appropriate management of issues related to the kinetic chain, core strength, scapular dyskinesis, and GIRD, as failure to address these can lead to continued symptoms, delay in return to play, and recurrence of the lesion.

Below is our standard rehabilitation protocol, which is adjusted depending on the patients' response to rehabilitation and their sporting requirements:

- <3 weeks
  - Wean off sling over 3 weeks.
  - Assess kinetic chain control and provide exercises as required.
  - Regain scapula and glenohumeral stability, working for shoulder joint control rather than range.
  - Active assisted motion as tolerated.
  - Closed chain exercises as tolerated.
  - Core stability exercises (no resisted biceps exercises).
- 3–6 weeks
  - Begin active biceps exercises.
  - Progress from closed chain to active glenohumeral movement.
  - Scapular stabilizer exercises.
  - Strengthen rotator cuff muscles.
  - Posterior complex stretching.
- 6 weeks+
  - Ensure posterior capsule mobility.
  - Manual therapy if indicated to eliminate any stiffness.
  - Progress to eccentric biceps exercises with scapula control if required.
  - Progress to work-/sport-specific rehab.

11.9 Experience in Treatment of Athletes

In our experience and publications, an early return to contact sports can be expected for SLAP tears at approximately 3 months [8], but repetitive overhead athletes can take in excess of 6 months to return to sports with a lower overall satisfaction rate due to the chronic, repetitive nature of the injury and sport. In the chronic SLAP tear, the presence of rotator cuff injuries and associated labral pathologies are negative prognostic factors in returning to sports. This, perhaps, is a reflection of the advanced stage of the process, and the pathophysiology and surgical repair of a SLAP

Table 11.1 Key differences between acute and chronic SLAP tears

	Acute SLAP tear	Chronic SLAP tear
Mechanism	Fall/traction/dislocation/following throw	Repetitive abduction/external rotation
Presentation	Sudden onset	Insidious onset
Pain	Deep, acute, severe	Dull, during activity, "dead arm"
Arthroscopy	Commonly type 2, 5, or 8	Typically type 2B
Management	Early surgery	Trial of rehab
Return to sports	High	Challenging

lesion are unable to restore athletes back to a high sporting level.

A summary of key differences is listed in Table 11.1.

Despite a large volume of work being published and improvement in understanding the pathophysiology of SLAP tears over the last few years, the diagnosis remains difficult and returning the overhead athlete to sports especially challenging.

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SLAP Lesions Part III: Return to Play After SLAP Repair

12

Edward S. Chang, T. David Tarity, and Christopher C. Dodson

12.1 Introduction

The shoulder joint relies on both static and dynamic stabilizers for stability given its ball-and-socket configuration. The glenoid labrum is a fibrocartilaginous tissue which allows for deeper seating of the humeral head relative to the glenoid socket [1]. Tears in the superior aspect of the glenoid labrum were first described in the literature by Andrews et al. in 1985 [2]. The common nomenclature, superior labral anterior-posterior or "SLAP" tear, was described by Synder et al. [3] in 1990. The true incidence of SLAP lesion may be difficult to discern, however; numbers have ranged from 6 to 26 % according to a recent review of the literature [4]. A certain subset of patients may benefit from surgical repair of SLAP tears. Athletes, and in particular overhead athletes who participate in baseball, tennis, handball, badminton, softball, swimming, volleyball, and squash among others, may have variable levels of satisfaction after SLAP repair [5].

12.2 Pathoanatomy/ Biomechanics/Preferred Classification

The glenoid labrum has been implicated by some authors to have less vascularity in the anterosuperior portion with its supply stemming from the suprascapular, circumflex scapular, and posterior circumflex humeral arteries [6]. A transition zone of fibrocartilaginous tissue extending from the hyaline articular cartilage attaches the labrum to the glenoid boney rim [6]. Shearing forces may also contribute to the pathogenesis of the SLAP lesion as suggested by Clavert et al. in a cadaveric model [7].

Also implicated in the etiology of SLAP tears is the scapula. Kibler et al. have advocated the important role the scapula has in normal shoulder function and outlined that altered scapular mechanics may contribute to multiple shoulder pathologies including SLAP tears [8, 9].

Several mechanisms have been suggested in the literature defining the etiology of SLAP tears. Andrews et al. postulated that the superior labrum is injured as a result of the deceleration traction from the pull of the biceps tendon on the labrum during the follow-through phase of throwing [2]. Burkhart et al. suggested a tight posteroinferior capsule causing a glenohumeral rotation deficit

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(GIRD) and a shift in the glenohumeral rotation point in a posterosuperior direction [10] as the primary mechanism in the development of SLAP tears. Further, a biomechanical study by Grossman et al. concluded that a posterior capsular contracture with decreased internal rotation prevents the humerus from externally rotating into its normal posteroinferior position in the cocking phase of throwing, resulting in the risk for developing Type II SLAP tears [11].

The preferred classification for SLAP tears was described by Snyder [3]. Type 1 lesions are characterized by fraying and degeneration of the superior labrum with an uninvolved biceps anchor. Type II tears include fraying and degeneration of the superior labrum with detachment of the biceps anchor. Type III tears involve a bucket handle tear of the superior labrum with an intact biceps tendon, while a Type IV lesion includes the bucket handle tear with a displaced biceps tendon root. This classification has been modified to include several subgroups in Types III and IV variants [12].

12.3 Clinical Presentation and Essential Physical Exam

Patients with SLAP lesions present challenges to diagnose given frequent nonspecific history and physical exam findings [4]. Patients who are ultimately diagnosed with arthroscopically proven SLAP lesions typically present with pain during overhead activities and mechanical symptoms of catching, locking, popping, or grinding [3, 13, 14].

Calvert et al. published a systematic review concluding that the current literature as of 2008 lacks the validity necessary to be useful and suggested that no good physical examination tests exist for effectively diagnosing a SLAP lesion [15]. Burkhart et al. reported that the Speed test and Active Compression Test (O'Brien's test) were found to be highly specific for anterior Type II SLAP lesions, whereas the modified Jobe relocation test was highly specific for posterior SLAP lesions [16]. To add further confusion to the utility of clinically detecting a SLAP lesion, Meserve et al. in a meta-analysis of six studies concluded that clinicians should choose the active compression test first, followed by the crank test, and the Speed test as the third best option when suspecting labral pathology. In our experience, we typically find the O'Brien's test to be most predictive, although all physical examination maneuvers are routinely performed.

12.4 Essential Radiology

The conventional radiographs such as anteroposterior, true anteroposterior in the scapular plane, scapular Y, and axillary views are utilized during the initial evaluation of athletes with shoulder ailments. Magnetic resonance imaging (MRI) and magnetic resonance arthrography (MR arthrograms) offer superior visualization and more sensitivity and specificity than plain radiographs or computed tomography [17]. In a study comparing the diagnostic accuracy of MR arthrography of the shoulder in professional baseball players with conventional MRI, Magee and colleagues reported that MR arthrography yields considerably more diagnostic information than MRI [18]. More recent data suggests that advanced imaging MRI (3.0-T systems), which has a higher signal-to-noise ratio than 1.5 T MRI, may be a better modality to evaluate SLAP lesions. Magee et al. also reported that with a 3.0 T MRI, sensitivity was 90 with 100 % specificity. Data published in 2013 suggests that in community settings, there exists a low sensitivity and high specificity in the diagnosis of Type II SLAP tears using noncontrast MRI including 1.5 and 3.0 T strength systems [19].

12.5 Management and Indications for Surgical Intervention

12.5.1 Nonoperative Treatment

Nonsurgical techniques generally focus on the pathology seen by physical examination and on the imaging studies. Common treatment modali-

ties include rotator cuff strengthening, posterior capsule stretching, and scapular stabilization. We typically employ a rehabilitation protocol emphasizing these exercises.

For overhead athletes, we would then begin a progressive throwing program once the patient is pain-free. It is also important to analyze and correct the patient's throwing mechanics at this time.

12.5.2 Operative Indications and Treatment

At our institution, surgery is considered if the patient fails conservative treatment lasting more than 3 months and has clinical and radiographic signs of a SLAP lesion. In the special case of the overhead athlete, persistent pain during or following a throwing program warrants surgical consideration. Earlier intervention is warranted for patients with evidence of a spinoglenoid notch cyst, causing compression of the suprascapular nerve.

Treatment of SLAP lesions is generally dictated by the intraoperative arthroscopic findings. For the purposes of this chapter, we will assume that the patient being treated is a young, overhead athlete. In Type I lesions, the frayed labrum is typically debrided. Type II lesions are repaired with many fixation options and techniques available to the surgeon. Type III lesions once again are treated with debridement of the unstable labral flaps. Lastly, Type IV lesions depend on the extent of biceps tendon involvement. If <30 % of the tendon is involved, these lesions are treated with debridement of the labral tear and its extension into the biceps tendon. If >30 % of the tendon is involved, the labral tear is repaired along with a biceps tenotomy or tenodesis.

In special cases of a labral tear with a concomitant rotator cuff tear, repair of both in the young athlete has yielded good clinical outcomes [20]. However it is important to take age into consideration for treatment of these unique injuries. In a randomized control study, it was shown that in patients >50 years old with a combination of a superior labral and rotator cuff tear, a combination of rotator cuff repair and biceps tenotomy generated superior outcomes than combined rotator cuff and labral repair [21].

12.6 Authors' Preferred Treatment (Type II SLAP Tear)

The authors' preference for arthroscopic repair of SLAP tears is in the beach-chair position. A standard posterior portal is established, followed by an anterior rotator interval portal using a spinal needle under direct visualization above the subscapularis and lateral to the middle glenohumeral ligament.

Diagnostic arthroscopy with the aid of a probe to evaluate the superior labral tear should be conducted. This step also allows for visualization of associated intra-articular pathology that may or may not need to be addressed including the long head of the biceps tendon, the rotator cuff, and the glenohumeral ligaments.

A high anterolateral portal is established. Using a combination of arthroscopic instruments, devitalized and detached labral tissue is mobilized and debrided (Fig. 12.1). The long head of the biceps tendon root is then decorticated to a healthy bleeding cancellous surface in preparation for repair (Fig. 12.2).

A Neviaser portal is established under direct visualization using a spinal needle followed by a #1 Prolene (Ethicon, New Brunswick, NJ, USA) stitch. We then shuttle a # 2 Fiberwire (Arthrex, Naples, Florida, USA) and create a luggage tag stitch.

While the anchor and suture configuration is individualized based on the tear pattern, the authors prefer a knotless technique. We drill a pilot hole for a 2.9 Pushlock (Arthrex, Naples, Florida, USA) inserted at the 12 o'clock position. We then switch the arthroscope to the anterolateral portal to create a small accessory posterolateral portal with a 4 mm cannula. The aforementioned steps are then repeated to complete placement of a 2.9 Pushlock anchor at the 2 o'clock position (Fig. 12.3).

The repair site is then tested with traction applied to the biceps tendon with an

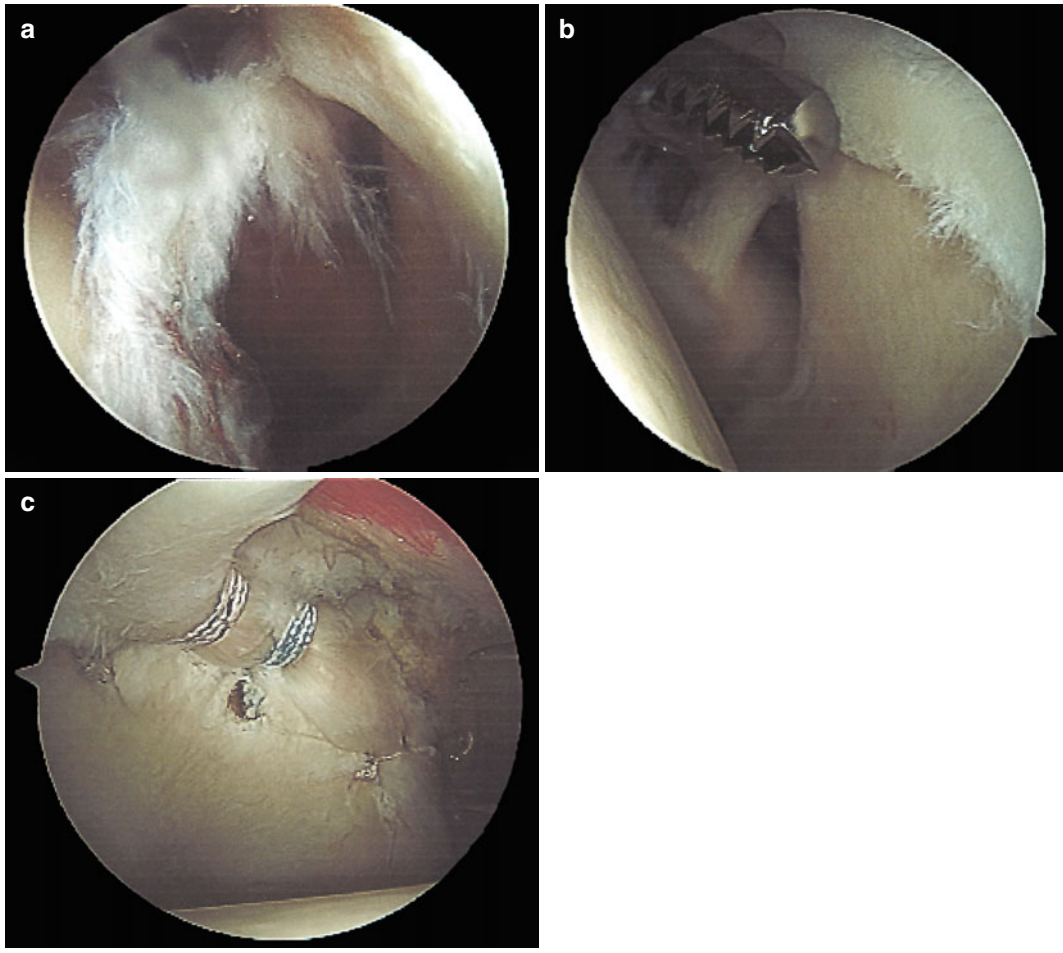

Fig. 12.1 Arthroscopic repair of SLAP lesion. (**a**) Standard posterior viewing portal demonstrating SLAP tear. (**b**) Mobilized labrum following debridement. The superior glenoid surface is also prepared using an arthroscopic rasp. (**c**) SLAP repair performed using a knotless technique with 2.9 mm Pushlock anchors (Arthrex Inc, Naples, FL)

arthroscopic probe to ensure attachment of the labrum. This is followed by confirmation of a negative peel-back sign with the arm in abduction and external rotation. Associated injuries are then addressed on an individual patient basis.

12.7 Rehabilitation

Progression through each rehabilitation phase is both criteria-based and patient-specific. Phases and time frames are designed to give the clinician a general sense of progression.

12.7.1 Postoperative Phase I (Weeks 0–4)

12.7.1.1 Objectives

- Promote healing: reduce pain and inflammation
- Elevation in plane of scapula to 90°
- External rotation: surgeon directed
- Independent home exercise program provided

12.7.1.2 Safeguard

- Immobilizer at all times, except when exercising or bathing
- External rotation and extension limited to neutral

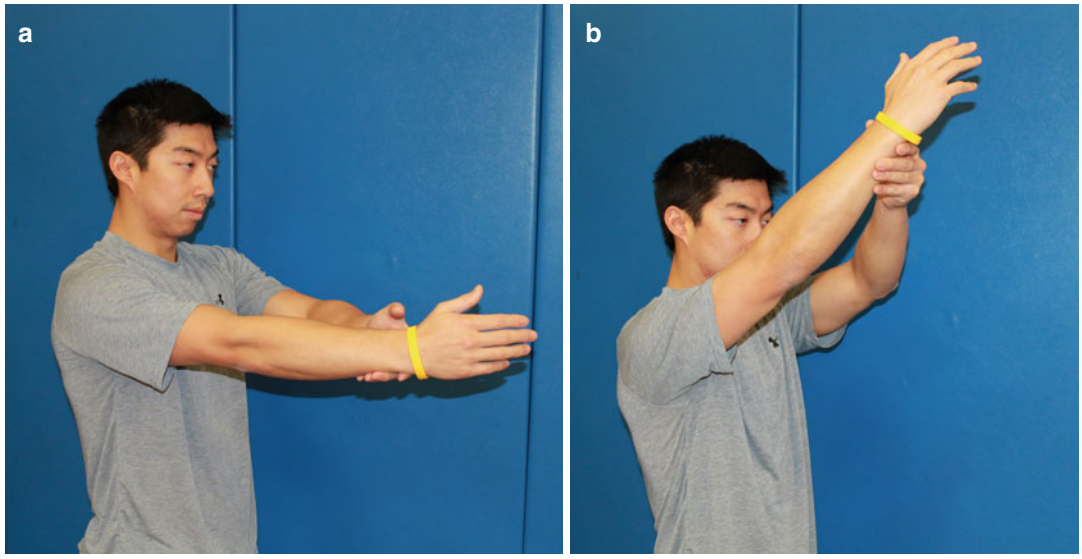

Fig. 12.2 (**a**, **b**) Active assisted forward flexion to 145°

Fig. 12.3 (**a**, **b**) Rotator cuff strengthening with external rotation in 90° of abduction

12.7.1.3 Evaluation and Treatment

- Gripping exercises while in immobilizer
- Active assisted range of motion from external rotation to neutral in the plane of the scapula
- Active range of motion of the wrist/elbow (supported to avoid biceps stress)
- Scapular mobility and stability (side-lying, progressing to manual resistance) pain-free

12.7.1.4 Minimum Criteria for Advancement to Phase II

- External rotation: at least to neutral (surgeon directed)
- Elevation in plane of scapula to 90°
- Minimal pain or inflammation

12.7.2 Postoperative Phase II (Weeks 4–8)

12.7.2.1 Objectives
- Continue to promote healing
- Active assisted forward flexion to 145° (Fig. 12.2a, b)
- External rotation to 60°
- Begin to restore scapula and upper extremity strength
- Restore normal scapulohumeral rhythm

12.7.2.2 Safeguard
- Limited external rotation to 30° until 6 weeks.
- Avoid excessive stretch to the labrum and biceps.
- Avoid rotator cuff inflammation.

12.7.2.3 Evaluation and Treatment
- Discontinue immobilizer (surgeon directed).
- Continue active assisted range of motion elevation in the plane of the scapula with external rotation limited to 30° until 6 weeks.
- Isometric internal/external rotation (submaximal and pain-free) progressing to isotonic internal/external rotation strengthening at 6 weeks.
- Begin humeral head stabilization exercises such as scapular plane elevation with emphasis on scapulohumeral rhythm.
- Begin latissimus strengthening, limited to 90° forward flexion.

12.7.2.4 Minimum Criteria for Advancement to Phase 3
- Elevation in plane of scapula to 145°
- External rotation to 60°
- Internal rotation/external rotation strength 5/5
- Normal scapulohumeral rhythm with minimal pain and inflammation

12.7.3 Postoperative Phase III (Week 8–14)

12.7.3.1 Objectives
- Restore full shoulder range of motion
- Restore normal scapulohumeral rhythm
- Isokinetic internal/external strength 85 % of uninvolved side
- Restore normal flexibility

12.7.3.2 Safeguard
- Avoid rotator cuff inflammation.
- Avoid excessive passive stretching.

12.7.3.3 Evaluation and Treatment
- Aggressive scapular strengthening. Begin latissimus and biceps strengthening.
- Progress internal/external rotation to 90/90 position (Fig. 12.3a, b).

12.7.3.4 Minimum Criteria for Advancement
- Normal scapulohumeral rhythm
- Minimal pain and inflammation
- Full upper extremity range of motion
- Isokinetic internal/external rotation strength 85 % of uninvolved side

12.7.4 Postoperative Phase IV (Weeks 14–18)

12.7.4.1 Emphasize
- Monitoring symptoms

12.7.4.2 Objective
- Restore normal neuromuscular function
- Maintain strength and flexibility
- Isokinetic internal/external strength equal to the unaffected side

12.7.4.3 Safeguard
- Pain-free plyometrics
- Significant pain with a specific activity
- Feeling of instability

12.7.4.4 Evaluation and Treatment
- Continue full upper extremity strengthening program and flexibility exercises.
- Activity and sport-specific plyometrics program. Analyzing and correcting throwing mechanics are critical to prevent re-injury (Fig. 12.4a, b).

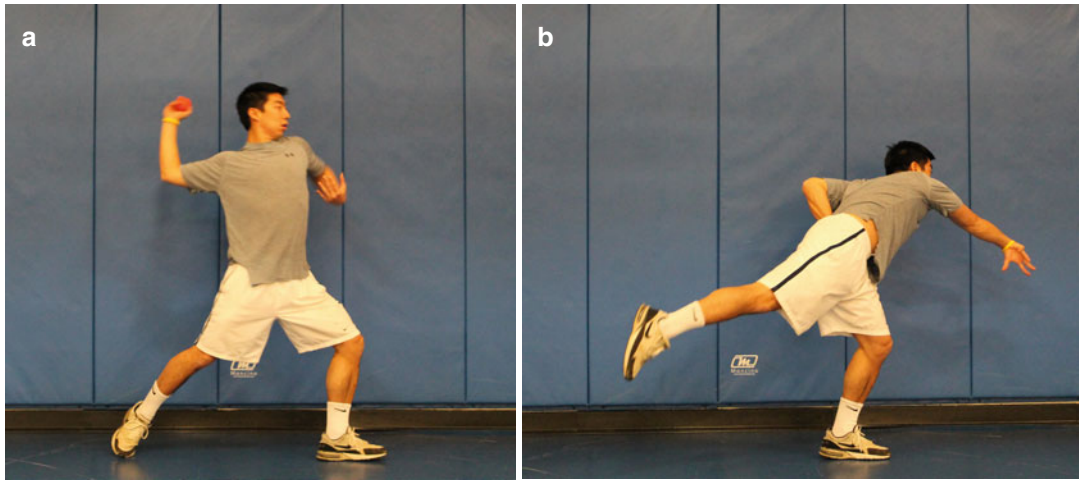

Fig. 12.4 (**a**, **b**) Sport-specific exercises with an emphasis on evaluation and correction of mechanics

- Overall conditioning addressing trunk and lower extremity demands along with continued endurance training.

12.7.4.5 Criteria for Discharge

- Isokinetic internal/external strength equal to unaffected side
- Independent, pain-free sport or activity-specific program

12.8 Outcomes and Experience in Treatment of Athletes

Outcomes following SLAP repairs have varied quite a bit depending on pathology treated, patient activity level, and technique and implant used. Most of the literature has focused on outcomes following Type II SLAP repairs. Morgan et al. and Kim et al. both reported greater than 90 % good to excellent results following Type II SLAP repair [12, 22]. Although initial studies have shown excellent results and high return to play rate, in our institution, overhead athletes anecdotally appeared to have slightly less satisfaction following a SLAP repair. Research to further analyze this subgroup was performed and has confirmed our thoughts.

Neuman et al. retrospectively analyzed 30 overhead athletes with Type II SLAP tears and noted that the ASES (American Shoulder and Elbow Society) and KJOC (Kerlan Jobe Orthopaedic Clinic) scores differed between baseball players and the rest of the overhead athletes, with 80 % of baseball players returning to previous level of play compared to 94 % for all other overhead athletes. The authors concluded that the KJOC score is a more specific scoring system for throwing athletes and perhaps can more accurately document an overhead athlete's difficulties, especially baseball players, in returning to sports [23].

Sayde et al. performed a systematic review of Type II SLAP repairs with a 2-year follow-up. They concluded that most athletes have good to excellent results (83 %), and overall, 73 % of the patients were able to return to their previous level of play. Upon further subanalysis, only 63 % of overhead athletes returned to their previous level of play [5].

Generally, patient satisfaction and return to play following Type II SLAP repair are favorable and high. However, one must be wary of the overhead athlete that may struggle to return to his or her previous level of play. The current data at this time is comprised of Level III and IV studies, and a large multicenter prospective trial is necessary to help enlighten physicians on a difficult problem in this special group of athletes.

Conclusion

SLAP lesions in the athlete present a challenging problem to the treating physician. A thorough physical examination and review of imaging studies as well as an understanding of the patient's activity level is imperative in making the correct diagnosis as well as choosing the best treatment. In our experience, patients undergoing a SLAP repair that follow a strict and regimented rehabilitation will generally have a high satisfaction and return to play rate. Special attention must be given to the overhead athlete, in particular baseball pitchers, as these patients may have a more difficult time in returning to their previous level of performance.

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SLAP Lesion: Part IV. Management of Concomitant Rotator Cuff Tear

13

Andrew J. Riff, Rachel M. Frank, and Brian Forysthe

13.1 Introduction

Rotator cuff tears (RCT) represent one of the most common causes of shoulder pain requiring surgical intervention. Surgical management is challenging given objectives of achieving structural healing and avoiding both postoperative stiffness (4.9 % in one series) [1] and anatomic retear (up to 94 % for large to massive tears) [2–4]. Further complicating things is the fact that rotator cuff tears are frequently associated with concomitant labral lesions that may contribute to symptomatology. Miller and Savoie demonstrated that 74 % of individuals with full-thickness rotator cuff tears had associated intra-articular lesions, with labral tears being the most frequently associated pathology [5]. Moreover, Snyder and colleagues determined that 40 % of patients with superior labrum anterior to posterior (SLAP) lesions have associated full- or partial-thickness rotator cuff tears [6]. The biomechanical role and appropriate treatment of SLAP lesions, in the setting of a rotator cuff tear, is controversial and is debated in the literature. In this chapter, we will discuss the clinical significance of the biceps-labral complex, the evolution of the classification of SLAP lesions, etiology of concurrent SLAP and rotator cuff lesions, clinical presentation of patients with coincident lesions, treatment options for patients with combined lesions, and our preferred treatment algorithm for patients with this challenging problem.

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13.2 Pathoanatomy, Biomechanics and Preferred Classification

The superior labrum is a triangular- or meniscoid-shaped structure composed of fibrous and fibrocartilaginous tissue that typically attaches medial to the articular margin of the superior glenoid. The long head of the biceps tendon takes root from both the superior labrum (40 % of its fibers) and from the glenoid at the supraglenoid tubercle (roughly 60 % of its fibers) [7]. The biceps root demonstrates significant anatomic variability, with a number of origins: (1) a central or more posterior origin (from the labrum itself), (2) an origin from the capsule superior or inferior to the supraspinatus, and (3) a bifid origin (between the labrum and supraglenoid tubercle) [8]. The anterosuperior labrum, between 12 and 3 O'clock, also demonstrates variable anatomy. Although the glenoid chondrolabral junction is typically confluent circumferentially, a "sublabral foramen" may be observed anterosuperiorly (9–18.5 % of cases) or the labrum may be entirely absent in the setting

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of a "Buford complex" (1.5–6.5 % of cases) [9–11]. While the variable anatomy of the biceps-labral complex has been extensively studied, the function of this structure is not entirely understood. Biomechanical studies have demonstrated that the biceps-labral complex provides translational and rotational stability to the glenohumeral joint [12]. Contraction of the long head of the biceps tendon depresses the humeral head and counteracts superior migration of the proximal humerus that arises from contraction of the short head of the biceps. When the shoulder is placed in the abducted and externally rotated position, tension of the biceps tendon improves the torsional rigidity of the joint. Rodosky and colleagues demonstrated that creation of a SLAP lesion in a cadaveric model decreases torsional rigidity in the overhead position [13]. Additionally, Pagnani and colleagues have suggested that translation of the humeral head increases with a SLAP lesion because the superior and middle glenohumeral ligaments originate from the superior labrum [14, 15].

Morgan and colleagues have postulated that in the patient with concomitant SLAP and rotator cuff lesions, the SLAP lesion typically represents the primary lesion which promotes evolution of a secondary rotator cuff tear [16]. The authors assert that the instability caused by a SLAP lesion results in repetitive humeral head translation that places increased stress upon the rotator cuff and ultimately leads to tearing. To support their hypothesis, they note that rotator cuff tears were observed in 31 % of patients with chronic SLAP tears; however, no rotator cuff pathology was witnessed in patients with acute SLAP tears.

A variety of injury mechanisms have been implicated in the pathogenesis of the SLAP lesion. These mechanisms can be divided into acute traumatic events or chronic repetitive injuries. For patients injured by acute trauma, the most common mechanisms include direct compressive loads or forceful traction to the arm. The most common cause of chronic repetitive injury is overhead throwing. A biomechanical study of impaction loading in cadavers demonstrated that the shoulder is at greatest risk for injuring the SLAP when in the abducted and forward flexed position [17]. Another biomechanical study of traction loading demonstrated that inferior subluxation of the humeral head most predictably created SLAP lesions [18]. Overhead athletes are particularly prone to these injuries due to a variety of anatomic and mechanical factors. Maximal external rotation of the shoulder in late cocking phase creates extreme torsional forces at the biceps root that produce a dynamic peel-back injury to the posterosuperior labrum. Additionally, it has been postulated by Burkhart and colleagues that the posterior capsular contracture that is common in the throwers' shoulder promotes increased posterosuperior migration of the humeral head [19]. Finally, repetitive contact of the posterosuperior labrum with the undersurface of the rotator cuff may result in degenerative fraying of the labrum. In the lead authors series of 34 patients with combined rotator cuff and SLAP lesions, 18 sustained traumatic tears (most commonly secondary to sports, falls, and motor vehicle collisions) while 16 experienced atraumatic tears [20].

The classification of SLAP lesions has gradually evolved since 1985 when Andrews and colleagues first proposed that tears of the superior labrum were related to forces imparted by the biceps tendon [21]. In 1990, Snyder et al. conceived the term "SLAP" lesion to describe injuries of the superior labrum including the biceps anchor and classified these injuries into four subtypes (Table 13.1) [22]. Type I lesions were categorized as superior labral fraying with localized degeneration with the superior labrum and biceps anchor still attached to the glenoid. Type II lesions are characterized by detachment of the biceps-labral complex from the glenoid with abnormal mobility of the complex. These injuries have been subclassified into anterior, posterior, and combined lesions. Type III lesions consist of a bucket-handle tear of the superior labrum with an intact biceps anchor. Type IV lesions have a bucket-handle tear of the superior labrum with extension into the biceps tendon splitting the biceps tendon (typically a significant portion of the biceps tendon remains attached). In 1995, Maffet et al. expanded upon Snyder's original classification adding injury types V–VII [23].

Table 13.1 Snyder classification of SLAP lesions (1990) initially consisted of types I–IV and was later expanded by Maffet et al. [23] to include types V–VII

Type	Finding	Percent (%) [6]
I	Labral and biceps fraying, anchor intact	21
II	Labral fraying with detached biceps tendon anchor	55
III	Bucket-handle tear, intact biceps tendon anchor (biceps separates from bucket-handle tear)	9
IV	Bucket-handle tear with detached biceps tendon anchor (remains attached to bucket-handle tear)	10
V	SLAP lesion and anterior labral tear (Bankart lesion)	
VI	Superior flap tear	
VII	SLAP lesion with capsular injury	

Maffet and colleagues attributed the additional injury subtypes to differences in injury mechanism noting that Snyder's cohort experienced a predominance of falls onto an outstretched extremity while Maffet's cohort had a greater proportion of traction related injuries. Type V lesions are SLAP lesions with an associated anteroinferior Bankart lesion. Type VI lesions involve a flap of the superior labrum resulting in biceps tendon separation from the glenoid. Finally, type VII lesions involve combined superior labrum and biceps tendon separation that extends anteriorly below the middle glenohumeral ligament. Among injury patterns, type II lesions are the most common clinically significant SLAP lesions accounting for up to 55 % of labral tears [6].

13.3 Clinical Presentation and Essential Physical Examination

A complete history and physical examination are critical to evaluating patients with suspected concomitant rotator cuff and SLAP pathology. These patients can typically be divided into two groups based on age: (1) younger patients with acute, traumatic etiologies and (2) older patients with chronic and/or acute-on-chronic presentations. At the initial evaluation, the mechanism of injury must be determined. Traumatic etiologies of RCT and/or SLAP lesions behave differently than insidious/atraumatic etiologies, and such differences may ultimately affect treatment strategies. Previous treatment, including both nonsurgical and/or surgical interventions, must be documented. Specifically, the patient must be asked if he/she experienced a traumatic dislocation versus a subluxation event. It is important to ascertain the position of the arm at the time of injury as well as the current and desired activity level of the patient. Treatment decisions are often made, regardless of the extent of the pathology, based on the desired activity level of the patient. It is imperative to differentiate between treatment options for high-level athletes/throwers versus weekend warriors and older, more sedentary patients, looking to improve their functionality with recreational sports and activities of daily living, respectively.

While certain features of the patient history may be suggestive of rotator cuff and/or SLAP lesions, patients may present with multiple coexisting pathologies. It is crucial to determine which is symptomatic and which is simply incidental or age appropriate. Perhaps some of the most difficult patients to evaluate are those with primary multidirectional instability (MDI). These patients will often complain of generalized, or deep, posterior shoulder pain, often accompanied with decreased athletic performance (often tennis, swimming, football linemen, etc), and loss of strength [24–26]. Similar complaints may be found in patients with chronic RCT/SLAP pathology, making evaluation of these patients challenging. The clinician must make every attempt to distinguish between such pathologies, as the treatments are vastly different. Patients with generalized ligamentous laxity and/or a history of multiple other joint subluxation events may lead more toward a diagnosis of MDI.

Patients with combined RCT/SLAP pathology may describe sharp shoulder pain resulting from an initial traumatic event, but more often these patients will complain of pain diffusely throughout the shoulder, and especially with overhead activities. Patients whose symptoms are predominately RCT

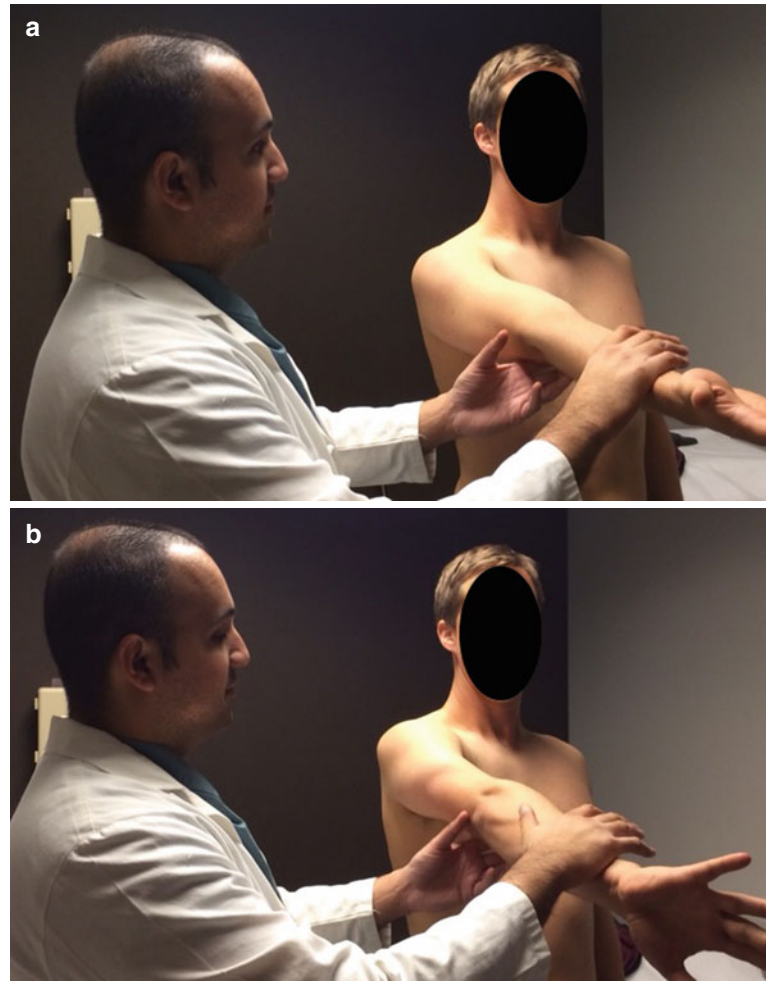

Fig. 13.1 (**a**, **b**) O'Brien's test for SLAP pathology is positive when the patient has increased pain with resisted forward elevation when the shoulder is in the adducted and internally rotated position

related may complain more of night pain and difficulty with overhead activities of daily living, while patients with predominantly SLAP-related complaints may complain more of symptoms during attempted overhead throwing or with repetitive activities requiring shoulder overhead motions.

On physical examination, the appearance, neurologic status, and stability of both shoulders should be carefully evaluated. Care should be taken to document atrophy, asymmetry, abnormal motion, muscle spasm, swelling, scapular winging, and abnormal glenohumeral tracking. The asymptomatic shoulder may be examined first to gain patient confidence and relaxation [27–29]. Scapulothoracic dyskinesis should be carefully evaluated and documented if present [30–32]. Any loss of motion, even subtle, should be documented, and every attempt should be made to improve motion with therapy preoperatively in order to avoid progressive loss of motion. Special tests for suspected SLAP pathology should then be performed, including O'Briens active compression test and dynamic labral shear (O'Driscoll's) test. The active compression test is performed with the shoulder in 90° of flexion and 30° of horizontal adduction. Downward pressure is placed onto the arm, and the patient is asked to resist the pressure both with the arm in maximum external (thumbs up) and internal (thumbs down) rotation. Labrum pathology should be suspected if the patient has pain during the internal rotation portion of the test that is decreased when the

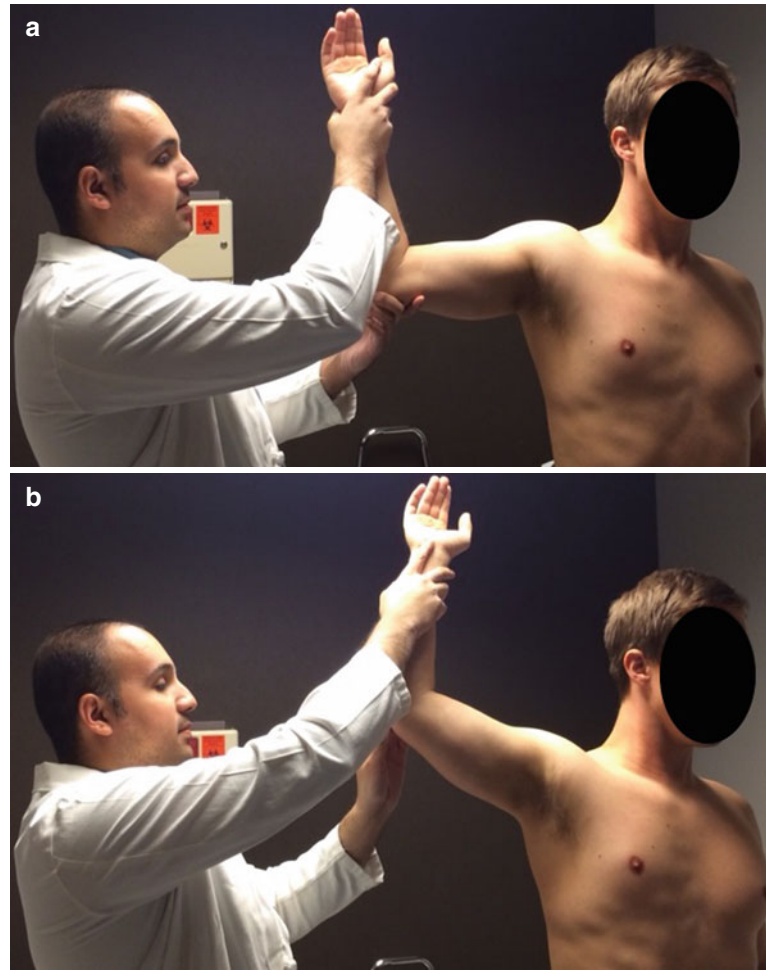

Fig. 13.2 (**a**, **b**) O'Driscoll's dynamic labral shear test is positive for SLAP pathology when the patient's pain is recreated with abduction of the arm from 90° to 120°

shoulder is placed into external rotation (Fig. 13.1a, b). The dynamic labral shear test is performed with the arm at the side and the elbow flexed to 90°. The shoulder is then externally rotated and abducted to 90°, followed by further abduction of the arm to 120° (with the elbow flexed). The test is considered positive for labrum pathology if pain is reproduced during the abduction from 90° to 120° (Fig. 13.2a, b). The test is considered equivocal if the patient has pain both within and outside this range.

Intraoperatively, examination with the patient under anesthesia includes evaluation of anterior and posterior glenohumeral translation, as well as the degree of inferior capsular laxity. Anteroposterior glenohumeral translation is graded as follows: grade I, humeral head translation up to but not over the glenoid rim; grade II, humeral head translation over the rim of the glenoid that spontaneously reduces; and grade III, dislocation of the glenohumeral joint. As described by Altchek and colleagues, the sulcus sign was graded as follows: grade 0, no inferior translation; grade I, inferior humeral head translation of up to 1 cm; grade II, inferior translation of 1–2 cm; and grade III, inferior translation of >2 cm [24]. The Gagey hyperabduction test is also performed to assess laxity of the inferior glenohumeral ligament. While stabilizing the scapulothoracic joint and passively abducting the arm (via the glenohumeral joint alone,), a 15° increase versus the contralateral side is suggestive of inferior capsular laxity (i.e. 105° vs. 90°) (Fig. 13.3a, b) [33].

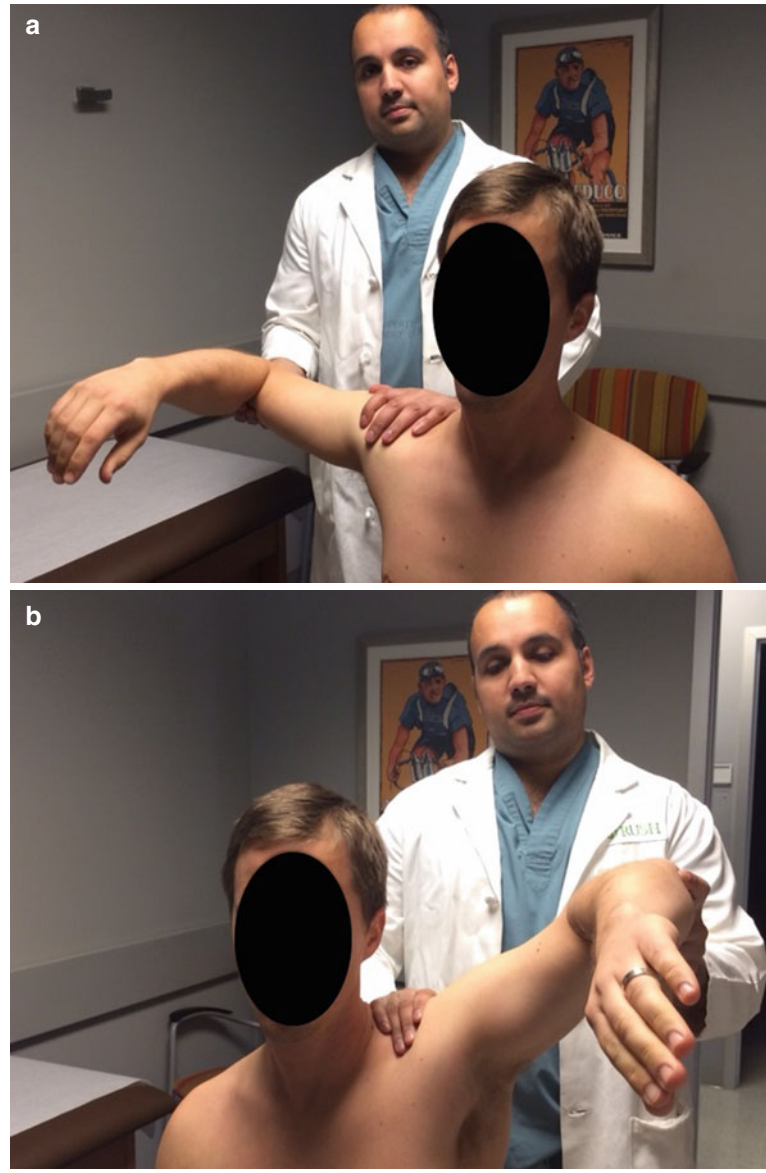

Fig. 13.3 (**a**, **b**) The Gagey hyperabduction test is suggestive of instability (more specifically IGHL laxity) if the involved shoulder demonstrates an increase of 15° of passive abduction relative to the contralateral

13.4 Essential Radiology

For initial evaluation of the shoulder, standard radiographs are obtained. In addition to the standard anterior-posterior (AP), scapular-Y, and axillary views, a shoulder instability series may also include a Grashey view, West Point axillary view, Stryker notch view, Garth view, or Bernageau view. The Grashey view aligns the glenoid such that it is perpendicular to the plane of the x-ray allowing for evaluation of the glenohumeral joint space. The West Point axillary view is a tangential view of the anteroinferior rim of the glenoid. The Stryker notch view is particularly useful in evaluating for the presence of a Hill-Sachs lesion. The Garth view, also known as the apical oblique view, is also used in patients with shoulder instability. Of note, the axillary view is often the best true lateral view of the shoulder. It allows for evaluation of anterior and posterior instability, glenoid fractures, and humeral head compression fractures. Note the

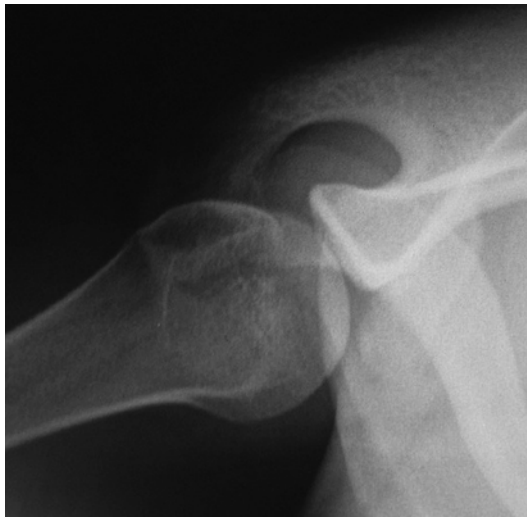

Fig. 13.4 The Bernageau glenoid profile view (normal in this example) is the most sensitive plain radiographic view for detecting the presence of a glenoid rim lesion

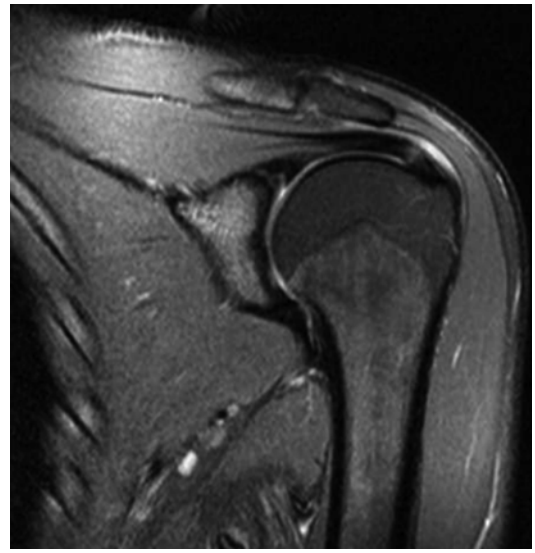

Fig. 13.5 Coronal T2-weighted MRI slice demonstrating a superior labral tear (evidenced by fluid tracking between the glenoid and superior labrum)

Bernageau view (Fig. 13.4) is an accurate and reproducible technique for measuring the presence of glenoid erosion, with similar results when compared to the 3D CT scan [34].

Magnetic resonance imaging (MRI) is useful in evaluating the rotator cuff, labrum, glenohumeral ligaments, cartilage, and capsule. This modality provides superior detail of soft-tissue pathology, while also detailing the bone marrow and articular cartilage integrity. MRI with the administration of intra-articular gadolinium (MRI-arthrogram, or MRA) results in improved sensitivity for detecting subtle pathology. Of note, indirect MRI-arthrogram is less invasive and better tolerated than the direct arthrogram. In this situation, intravenous gadopentetate dimeglumine is administered, resulting in homogeneous enhancement and improved delineation of soft-tissue structures. This modality is valuable in identifying inflammatory conditions and is indicated in diagnosing chronic Bankart lesions and their variants and SLAP lesions and in post-operative evaluation of the painful shoulder. Nevertheless, direct MRI-arthrogram is the diagnostic tool of choice for evaluation of the rotator cuff and labrum in young athletes. Direct MRI-arthrogram is indicated in rotator interval lesions, SLAP tears, anterior labrum periosteal sleeve avulsion (ALPSA) lesions, Bankart lesions, and its variants [9]. The utility of MRI without arthrography has been questioned by a recent study where a high level of sensitivity and low level of specificity was found with respect to diagnosis of SLAP lesions [35]. The investigation of choice is MRI-arthrogram with accuracies reported between ~75–90 %. Distinguishing between subtypes, however, can be difficult [36]. The characteristic finding is high signal (fluid on T2WI or contrast on T1WI) extending into the superior labrum and tracking into the labrum and sometimes into the biceps tendon (Fig. 13.5).

CT is also indicated in evaluating more chronic pathology in addition to grading the degree of muscle atrophy or fatty infiltration in the setting of concomitant rotator cuff pathology [9]. Patients with a history of previous instability surgery and patients with a midrange-of-motion instability should undergo CT scan to evaluate for anterior or posterior glenoid insufficiency, engaging Hill-Sachs or reverse Hill-Sachs lesions (Fig. 13.6). Intra-articular contrast may be of benefit in future defining bony anatomy in chronic cases.

Finally, ultrasound has become an important diagnostic tool in the evaluation of shoulder

complaints, especially for tendon pathology of the shoulder. In experienced hands, this modality is cost-effective and portable and has a high rate of acceptance from patients. In patients for whom MRI is not an option, ultrasound is the diagnostic tool of choice when evaluating the rotator cuff [9]. It also has been shown to have satisfactory sensitivities and specificities in the assessment of postoperative rotator cuffs. Ultrasound is also useful in imaging-guided shoulder interventions, such as the aspiration and injection of paralabral cysts [10–14].

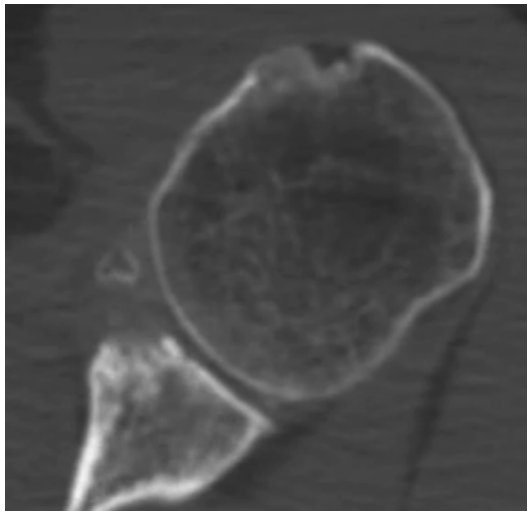

Fig. 13.6 Axial CT image demonstrating anterior glenoid bone loss in the setting of recurrent instability

13.5 Arthroscopic Pathology

While MRI is helpful in identifying lesions in the superior labrum, differentiating SLAP tears from normal anatomy is challenging because of the variable insertional anatomy of the anterosuperior labrum and the biceps anchor-labral complex. Therefore, arthroscopy remains the gold standard for diagnosing and classifying the lesion. During diagnostic arthroscopy, care should be taken to differentiate pathology from anatomical variants, i.e., sublabral foramen (Fig. 13.7) or a Buford complex. The shoulder should also be placed in abduction and external rotation to assess for the presence of a "peel-back" sign, which verifies the presence of instability (Fig. 13.8). The biceps-labral complex lesion is closely inspected and probed as its classification will direct the surgical strategy. Finally, the biceps tendon should be pulled into the joint with downward traction, with a blunt probe, to examine for fraying or tenosynovial inflammation of the tissue within the bicipital groove.

13.6 Treatment Options

The optimal management of patients with concurrent SLAP and rotator cuff lesions remains controversial. At a minimum, nearly all patients should undergo 3 months of conservative

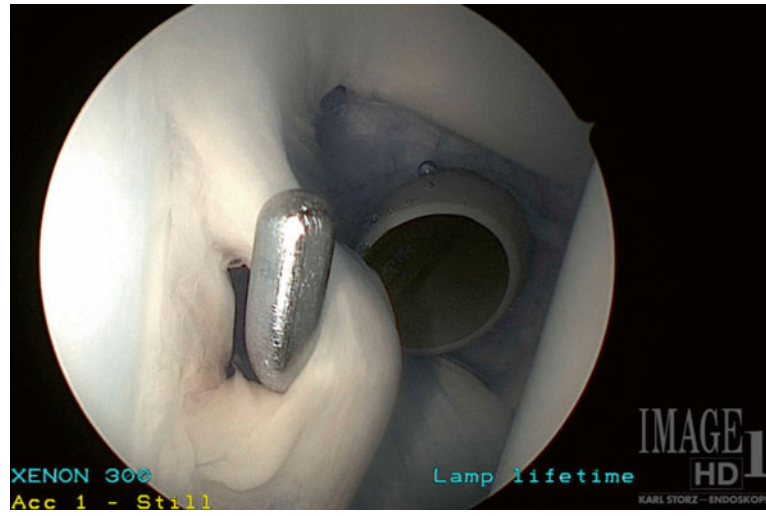

Fig. 13.7 Arthroscopic image of a sublabral foramen

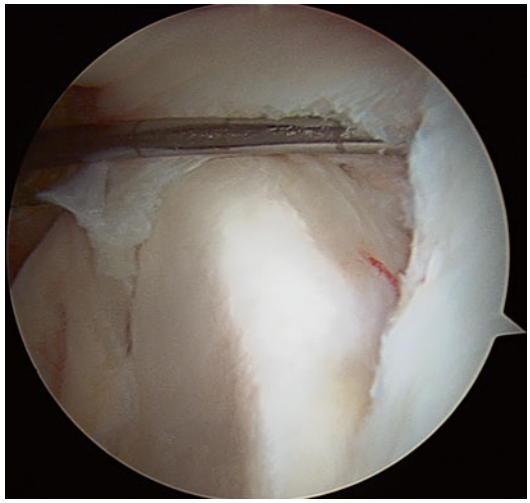

Fig. 13.8 Arthroscopic image demonstrating the peel-back sign

management before surgical intervention is even considered. Nonsurgical treatment begins with rest, activity modification, and oral anti-inflammatories. Patients should also be enrolled in physical therapy to focus on biceps stretching, strengthening of the rotator cuff, and ameliorating any scapulothoracic dyskinesis. In patients with persistence of pain or functional limitation after a trial of conservative treatment, shoulder arthroscopy is often warranted. In patients with combined lesions, surgical management of the rotator cuff is identical to that employed in patients with isolated rotator cuff pathology. Generally, partial-thickness tears <50 % width are debrided and partial-thickness tears greater than 50 % width and full-thickness tears are repaired [37]. Symptomatic bursal-sided tears greater than 3 mm in thickness are generally repaired. Similarly, type I, III, and IV SLAP lesions are also managed as they would be if they were stand-alone lesions. The principal dilemma when treating concomitant rotator cuff and SLAP lesions involves management of patients with type II SLAP lesions. While surgeons broadly agree that surgical repair renders superior outcomes for isolated type II SLAP lesions, there is reluctance among many surgeons to perform concurrent rotator cuff and SLAP repair due to concern over prolonged postoperative immobilization, stiffness, and poor clinical outcomes. Therefore, some surgeons advocate biceps-labral complex debridement, others advocate biceps anchor tenotomy or tenodesis, and others still advocate surgical repair of the biceps-labral complex.

Abbot and colleagues randomized 48 patients older than 45 years of age with rotator cuff tear and concomitant type II SLAP lesion to receive either SLAP repair or labral debridement in addition to rotator cuff repair [38]. They demonstrated that at 2-year follow-up, the debridement cohort had significantly better UCLA scores (34 vs. 31), function (5.5 vs. 3.8), and pain relief than the SLAP repair cohort. The authors therefore concluded that rotator cuff repair with SLAP debridement may optimize patient satisfaction and functional outcome in patients over 45 years of age with a minimally retracted rotator cuff tear and associated SLAP lesion.

Franceschi and colleagues randomized 63 patients older than 50 years of age with concurrent lesions undergoing rotator cuff repair to receive either SLAP repair or biceps tenotomy [39]. At minimum 2.9-year follow-up, the tenotomy cohort experienced significantly better UCLA scores, range of motion, and patient satisfaction. Similarly, Kim and colleagues evaluated 42 patients who underwent arthroscopic rotator cuff repair and either SLAP repair or biceps tenotomy for large to massive rotator cuff tears and concomitant type II SLAP lesions, respectively [40]. At minimum 2-year follow-up, patients who underwent biceps tenotomy demonstrated significantly better pain relief, range of motion, simple shoulder test, ASES, and UCLA scores.

In spite of the results of the aforementioned comparative trials, a number of authors support management of the patient type II SLAP lesions and concurrent rotator cuff tear with combined SLAP and rotator cuff repair. As discussed earlier, Morgan and colleagues have theorized that patients with concurrent lesions initially develop a SLAP lesion, which promotes posterosuperior instability that ultimately may contribute to rotator cuff damage. For this reason, they recommend surgical repair of type II SLAP lesions to remove

the pathological entity that precipitated (or aggravated) the rotator cuff tear in the first place [16]. Levy and colleagues evaluated patients younger than 50 years (mean 33) and compared results of those who underwent isolated repair of a type II SLAP lesion with those who underwent repair of a type II SLAP lesion and either rotator cuff repair or debridement [41]. They found that both groups had equivalent outcomes with regard to UCLA shoulder score and, therefore, concluded that management of a rotator cuff lesion did not have a negative effect on results of patients undergoing repair of a type II SLAP lesion. Forsythe and colleagues retrospectively compared 34 patients who underwent concomitant rotator cuff and SLAP repair with a control group of 28 patients who underwent isolated rotator cuff repair [20]. Although patients with concurrent lesions had significantly worse preoperative ASES scores (22.6 vs. 34.3), postoperatively there was no significant difference between groups with regard to range of motion or ASES score. Therefore, the authors conclude that simultaneous SLAP repair and rotator cuff repair provides results comparable to isolated rotator cuff repair.

13.7 Author's Preferred Treatment

For patients with concurrent lesions who have failed a minimum course of 3 months of conservative measures, the decision is often made to perform shoulder arthroscopy. After induction of general anesthesia or administration of a regional block, the patient is placed in the beach chair position. Examination under anesthesia is performed to assess range of motion and the extent of translation anteriorly, posteriorly, and inferiorly.

A posterior portal is established 2 cm inferior and 1 cm medial to the posterolateral corner of the acromion, and a standard anterior portal is established immediately superior to the subscapularis. Diagnostic arthroscopy is conducted in a systematic fashion to confirm the diagnosis of SLAP tear, to assess the quality of the biceps tendon, and to classify the lesion. If the biceps tendon is partially torn (>30 %), a mini-open subpectoral tenodesis is considered in patients who have active lifestyles and participate in sports, have medium or heavy demand jobs, have an aversion to cosmetic deformity (pop-eye deformity), or are workers' compensation cases. However, if the biceps tendon appears healthy, lesions are treated according to SLAP tear classification. Type I lesions may be treated with debridement or left alone if deemed clinically insignificant. Unstable type II lesions are typically repaired unless the patient is low demand or has significant risk factors for postoperative stiffness (i.e., adhesive capsulitis, glenohumeral osteoarthritis, or a large or massive rotator cuff tear requiring prolonged postoperative immobilization). Type III lesions are treated with resection of the unstable labral fragment and repair of the middle glenohumeral ligament. Type IV lesions are treated with either debridement of the involved labrum and biceps tendon (<30 % of tendon involved), biceps tenodesis and labral repair (>30 % of tendon involved in a young or active patient), or bicep tenotomy versus tenodesis and labral debridement (>30 % of tendon involved in an older, sedentary patient). Overall, if the tendon is in good condition and the biceps-labral complex is unstable, it is our preference to perform suture anchor stabilization as follows.

A second working portal is established utilizing another 7 mm cannula. This portal is placed anterosuperolaterally within the rotator interval just posterior to the biceps tendon. This position is favored as it facilitates anchor placement and increases the working interval between both cannulae. In patients with full-thickness rotator cuff tear, the accessory portal may be placed through the tear instead of through the rotator interval.

The superior aspect of the glenoid at the biceps root is subsequently prepared using a 4.5 mm shaver to debride any fibrous debris and fibrillated portions of the superior labrum (Fig. 13.9a–c). An arthroscopic rasp, shaver, and/or SLAP burr is used to abrade the superior aspect of the glenoid and supraglenoid tubercle to bony, cortical bleeding (Fig. 13.10a, b). A fishmouth drill guide is

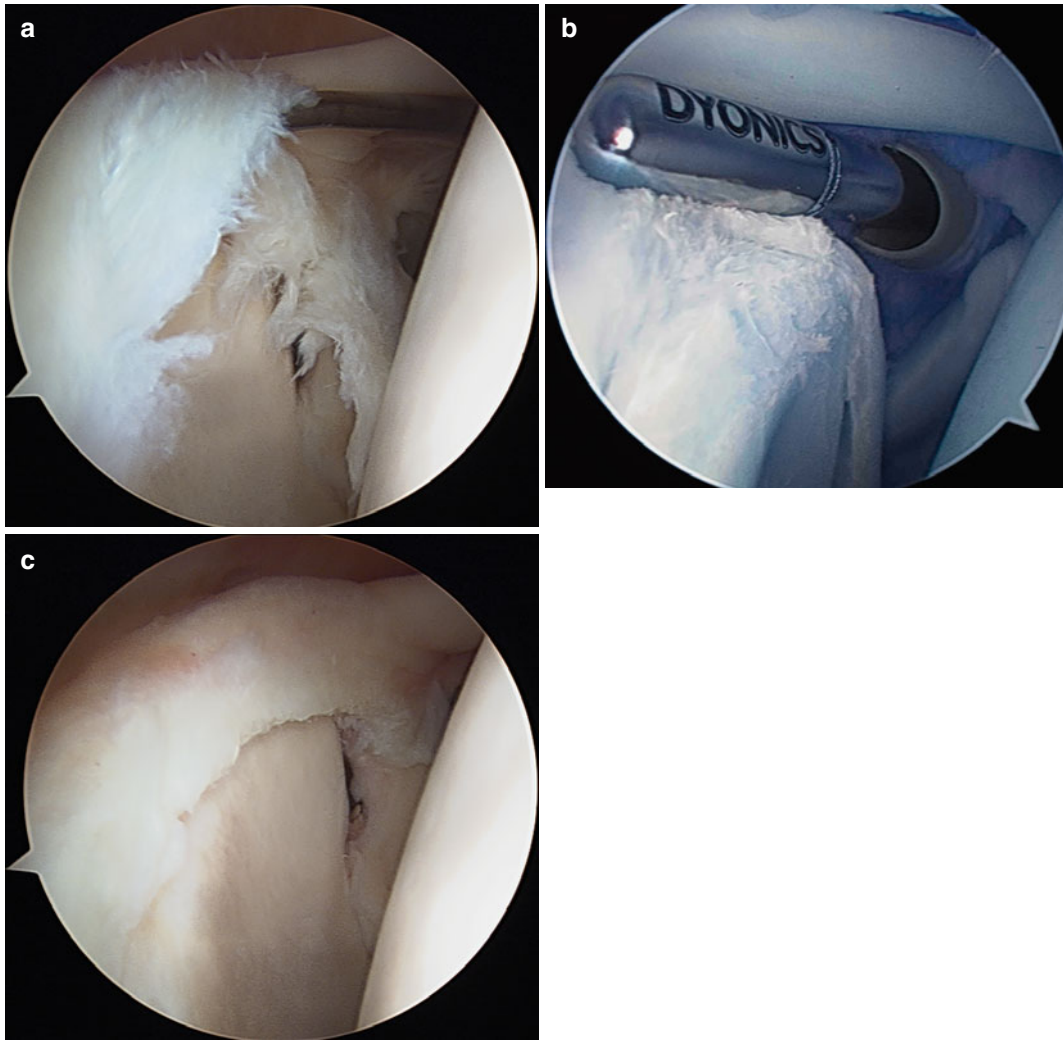

Fig. 13.9 (**a**–**c**) Prior to fixation of a type II SLAP lesion, labral fraying is carefully debrided with a shaver

placed through the anterosuperolateral portal onto the superior glenoid rim at a 45° angle (Fig. 13.11). After drilling the pilot hole, a double-loaded 3 mm anchor is placed at the midpoint of the biceps anchor (Fig. 13.12).

A loop retriever is used to retrieve two limbs of the same suture through the anterior portal, while the remaining two limbs of the other suture are left within the superior portal. A 90° suture lasso (Fig. 13.13) is passed through the superior portal and underneath the biceps-labral complex to shuttle a limb of the posterior suture under the posterior superior labrum resulting in paired limbs of the posterior suture lying on either side of the labrum posterior to the biceps root (Fig. 13.14). This process is repeated through the anterior portal such that the limbs of the anterior suture straddle the labrum anterior to the biceps root. The limbs on the posterior side of the biceps-labral complex are tied first using a sliding arthroscopic Weston knot followed by three alternating half hitches. The limbs of the anterior suture are tied in the same fashion (Fig. 13.15). If necessary, additional anchors may be placed through the superior portal to repair any residual labral tearing. Labral tears that extend further posteriorly may necessitate additional posterosuperior and posteroinferior portals. The posterior

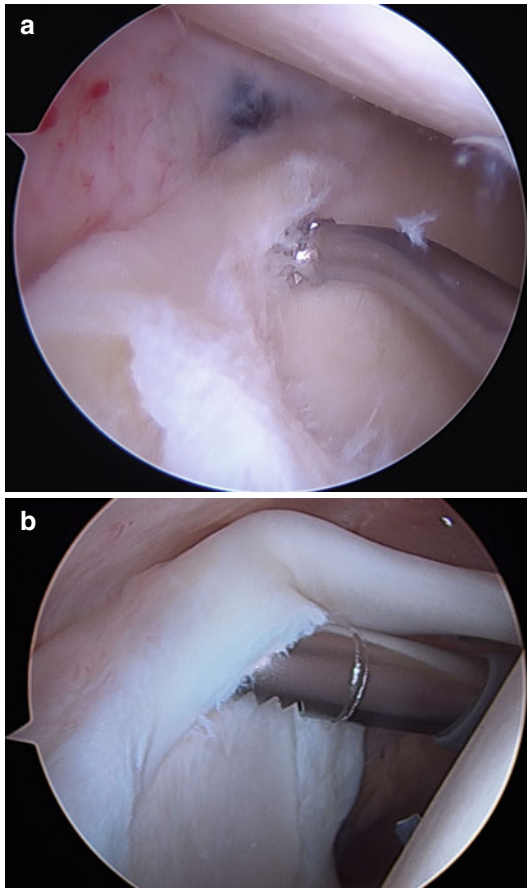

Fig. 13.10 (**a**, **b**) The cortical rim of superior glenoid is gently abraded with an arthroscopic rasp, shaver, and/or SLAP burr to promote a bony bleeding response

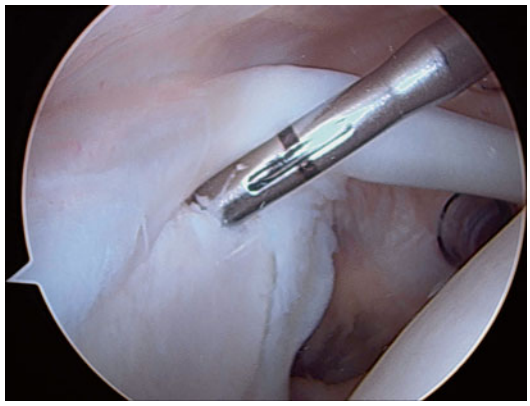

Fig. 13.11 The drill guide is placed at the articular margin at a dead man's angle of 45° relative to the glenoid

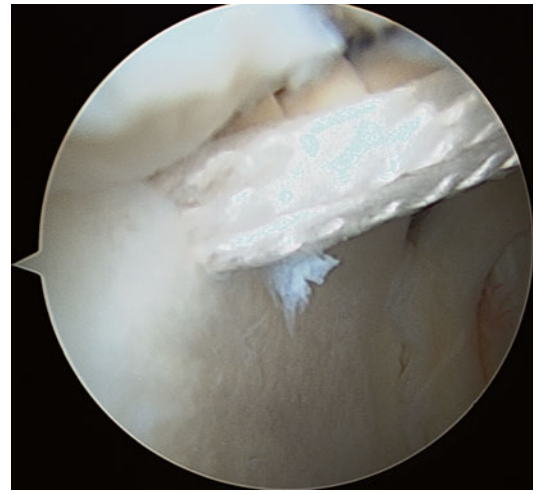

Fig. 13.12 Arthroscopic image demonstrating insertion of a double-loaded suture anchor at the apex of the glenoid

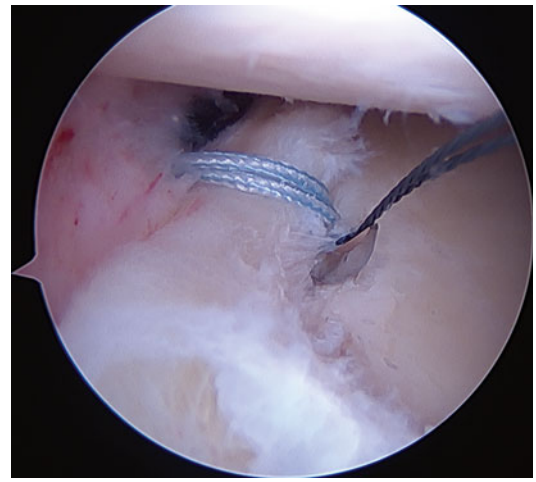

Fig. 13.13 A suture lasso placed under the superior labrum sequentially shuttle one strand of each suture medial to the labrum (of note this image demonstrates a single-loaded anchor)

viewing portal may also provide a satisfactory approach angle for anchor placement posterosuperiorly (between 9 and 11 O'clock).

From a technical standpoint, our preference is for knotless fixation of SLAP lesions in overhead athletes to prevent abrasion of the articular side of the rotator cuff with the arm in abduction and maximal external rotation (–internal impingement during cocking position.) Biomechanically,

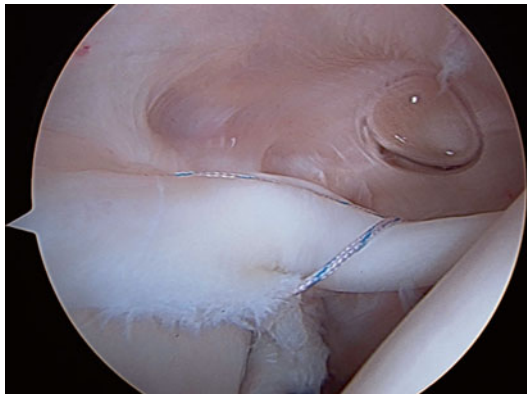

Fig. 13.14 Once sutures have been shuttled, the two strands of each suture will straddle the superior labrum (of note this image demonstrates a single-loaded anchor)

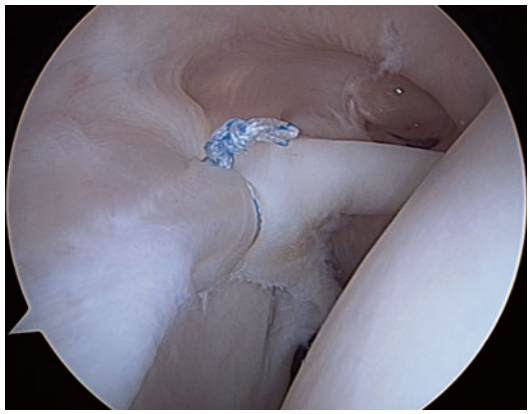

Fig. 13.15 Postoperative arthroscopic image demonstrating knotted fixation of a SLAP lesion anterior and posterior to the biceps root

this approach is similar in strength to suture anchor repair techniques [42], and it can be employed with simple or horizontal mattress suture configurations to optimize footprint contact area and biceps anchor-labral complex morphology. In the overhead athlete, we avoid placement of anchors anterior to the biceps tendon insertion to prevent overtightening of the capsulolabral tissues in external rotation [43].

Upon completion of the SLAP repair, attention is directed to repair of the rotator cuff. In preparation, a subacromial decompression is performed (if indicated for bursal-sided RTC tearing or type II, III, and IV acromion morphology), and bursectomy is carried into the anterior and lateral gutters. Care is taken to preserve the coracoacromial ligament. If possible, a transosseous equivalent double-row rotator cuff repair is performed in patients with adequate tendon tissue. The footprint is prepared with a 4.5 mm shaver to remove fibrous debris and to promote a bony cortical bleeding response. Two double-loaded 4.5 mm Smith and Nephew (Andover, MA) PEEK suture anchors are placed along the medial row at the articular margin at a "dead man's angle" of 45°. Sutures are passed with a combination of instruments, Arthrex Scorpion FastPass (Naples, Fl) suture lassos (Arthrex, Fl) and Spectrum suture passers (ConMed Linvatec, Largo, Fl), and are tied in a horizontal mattress configuration, providing a total of 4 medial row fixation points. Next, two bone sockets are created in line with the medial anchors roughly 7–8 mm lateral to the lateral edge of the great tuberosity to accommodate two 4.75 mm PEEK SwiveLock anchors. One suture strand from each medial horizontal knot is retrieved through the lateral portal and threaded through the SwiveLock anchor which is then advanced into one of the lateral bone sockets with concurrent tensioning of sutures (–typically 4 sutures with a 2 cm anteroposterior sized tear.) Sutures are cut flush with the anchor, and the remaining #2 FiberWire suture loaded within the SwiveLock is utilized to stabilize any redundant flaps of RTC tissue (dog-ears) or to reinforce the construct on an as-needed basis. These steps are then repeated utilizing the remaining four suture strands, from the medial row, with a second SwiveLock.

13.8 Rehabilitation

Postoperatively, the shoulder is immobilized in a sling and abduction pillow in 15° of abduction. Pendulum exercises are initiated on the first postoperative day. The patient is limited to active range of motion of the elbow, wrist, and hand. The patient may remove the sling and abduction pillow for tabletop activities (e.g., typing and eating) as long as the elbow remains adducted to the side. The sling and abduction pillow are maintained for 4–6 weeks for sleeping and activities

away from home. Passive shoulder range of motion and shoulder isometric exercises are initiated 2 weeks postoperatively; however, smaller tears in younger patients may begin therapy at 1 week and larger or massive tears may begin therapy at 3–4 weeks. Motion is gradually increased, with the goal of achieving full range of motion by 8 weeks. Strengthening exercises begin at 10 weeks postoperatively. Light activities below shoulder level are allowed at 6 weeks, and activities above shoulder level are allowed at 3 months. Unrestricted activities are initiated at 6 months, and overhead athletes may conduct a throwing program between 9 and 12 months post-op.

13.9 Advantages and Pitfalls, Complications

For patients with a concomitant rotator cuff tear and type II SLAP lesion, we advocate concurrent repair of both the SLAP and rotator cuff lesions. However, a higher threshold for repair applies to overhead throwers who will not tolerate subtle losses in range of motion. An advantage of this approach is that it restores stability to the glenohumeral joint, which may reduce stresses that contribute to rotator cuff retear. The primary drawback associated with this approach is the risk of increased postoperative stiffness that has been witnessed by authors comparing SLAP repair with labral debridement, biceps tenotomy, and biceps tenodesis. While postoperative stiffness has not been a complication that we have routinely seen in our patients, there are a couple of technical strategies to keep in mind to avoid postoperative stiffness. Surgeons should avoid incorporating a normal sublabral foramen or Buford complex into the repair as this will result in a loss of shoulder external rotation. Care should be taken to retrieve suture from the same side of the biceps tendon following passage with the suture lasso. Crossing suture may result in entrapment of the biceps tendon and diminished range of motion. Early gentle passive and active range of motion is recommended, with the arm in adduction, to prevent stiffness in external rotation.

Patients with glenohumeral arthritis (Outerbridge grade III or IV), massive rotator cuff tear, or adhesive capsulitis should not undergo simultaneous SLAP and rotator cuff repair as they are at increased risk of postoperative stiffness. Any patient with a structural biceps tendon lesion should not undergo SLAP repair, as they are at risk for ongoing postoperative pain. Finally, low-demand patients with large or massive rotator cuff tears with an associated large SLAP tear may be best managed with a biceps tenodesis or tenotomy.

Patients with failed SLAP repairs may be effectively treated with a biceps tenodesis in a safe and predictable manner [44]. However, biceps tenodesis should be considered with caution as the primary treatment of SLAP lesions in overhead throwing athletes secondary to its inability to completely restore translational stability [45].

13.10 Experience in Treatment of Athletes

Treatment of SLAP tears in overhead athletes differs in that more extensive nonoperative management is pursued as surgical repair has had limited success. In a recent study of Major League Baseball players, the return-to-play rate for 27 pitchers who underwent 30 procedures for SLAP lesions was 48 %, and the return-to-previous-performance rate was only 7 % [46]. The initial focus is on correcting scapular dyskinesis and posterior capsular contracture associated with glenohumeral internal rotation deficit (GIRD). Pain-free return to throwing is then progressed. Overhead athletes should fail two cycles of nonoperative treatment before considering surgical intervention.

Throwing athletes with partial-thickness articular-sided tears are initially treated with debridement alone in conjunction with treatment of concomitant pathology, i.e., SLAP lesions. The threshold for surgical repair of a partial-thickness tear is classically greater than 50 % of the tendon width; however, some authors and some noted surgeons with experience in manag-

ing elite overhead throwers have more recently advocated a higher threshold of tendon involvement before considering formal RTC repair [47, 48]. If a full-thickness tear is indicated for repair, we recommend a single-row repair to the lateral aspect of the footprint to prevent restriction of end range of motion in abduction and external rotation and to allow for physiologic contact between the articular side of the RTC insertion and the posterosuperior labrum. It should be noted that full-thickness tears in elite overhead athletes have had dismal outcomes after surgical repair.

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Scapular Dyskinesis: Part I. Overhead Athletes

14

Aaron Sciascia and W. Ben Kibler

14.1 Introduction

Dynamic upper extremity-dominant tasks such as throwing, hitting, and serving occur as the result of integrated, multi-segmented, sequential joint motion and muscle activation. In order for the tasks to be effective and efficient, optimal muscle flexibility, strength, proprioception, and endurance must exist as well as the ability to perform the task consistently on a repetitive basis. When a deficit exists within one or more of these functional components, increased load and stress may occur on the shoulder which can lead to pain or injury.

The scapula is one of many links within the kinetic chain. It has been recognized that the scapula serves many roles in shoulder function including serving as a stable base for muscle activation, precise concavity/compression ball and socket kinematics throughout the arm motion, optimal force and energy transfer from the core to the hand, and efficient work through the system of the shoulder, arm, and hand. The most effective scapular position to achieve these goals is retraction. Control of internal/external rotation, not allowing excessive internal rotation, and anterior/posterior tilting, not allowing anterior tilt, facilitate the control of retraction. The loss of retraction control can be equated to having a faulty link in the system, i.e., weak link in the chain because the inability to obtain or maintain scapular retraction decreases the ability of the arm to optimally function. The loss of retraction can be caused by anatomical disruption (tissue derangement), anatomical impairment (tissue inflexibility, strength imbalance), or kinetic chain impairment (lower extremity inflexibility or weakness).

The focus for clinicians is to identify the cause(s) which led or contributed to the impairment. The clinician must then implement injury rehabilitation and prevention programs which will initially eliminate physical deficits followed by a focus on increasing an athlete's longevity while simultaneously decreasing the risk of injury. The purpose of this chapter is to describe normal scapular function, both in an isolated manner as well as in the kinetic chain of function, and the consequences of scapular dysfunction relative to the shoulder; provide a description of a routine scapular examination; and present a rehabilitation regimen for combating scapular dysfunction and restoring arm function.

14.2 Pathoanatomy/ Biomechanics/Preferred Classification

A kinetic chain is a coordinated sequencing of activation, mobilization, and stabilization of body segments to produce a dynamic activity [1].

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Many kinetic chains exhibit both closed- and open-chain activities. The concept of sequential force development throughout the segments of a kinetic chain is the best framework to understand the dynamics involved in upper extremity-dominant athletic activities. Optimal athletic function is the result of physiological motor activations creating specific biomechanical motions and positions using intact anatomical structures to generate forces and actions. Sports-specific function occurs when the activations, motions, and resultant forces are specific and efficient for the needs of that sport. For example, overhead athletic tasks such as throwing or striking a projectile require sequential muscle activation from both the upper and lower extremities, i.e., the links in the chain. The majority of force required to propel the projectile forward is developed in the larger muscles of the legs and trunk in a closed-chain fashion, is funneled through the smaller muscles of the scapulohumeral complex using closed-chain biomechanics, and is eventually transferred to the arm.

The scapula serves as a critical link in kinetic chain function because it serves as the bridge between the energy-producing muscles of the legs and trunk and the energy delivery muscles of the arm. Thus, scapular stability and optimized kinematics are an essential part of proper kinetic chain function.

The scapula performs rotary motion around three distinct axes and translations in two directions as part of the normal scapulohumeral rhythm [2]. The rotary motions are upward/downward rotation, which occur around an anterior/posterior axis perpendicular to the scapula, internal/external rotation around a vertical superior to inferior axis along the medial border, and anterior/posterior tilt around a horizontal medial to lateral axis along the scapular spine. The translations are upward/downward along the thorax and medial to lateral around the ellipsoid thorax. Scapular movement is complex and multidimensional, with the scapula rarely moving in only one of the motions and translations when accomplishing most scapular roles. However, loss of control of specific motions seems to alter glenohumeral kinematics and function more than others. Loss of control of posterior tilting, allowing more anterior tilt, and loss of control of external rotation, allowing more internal rotation, appear to be most commonly associated with altered function or injury. Normal scapular resting position and active motion can be altered in overhead athletes due to the repetitive motions, with increases in posterior tilt and upward rotation being common alterations.

These alterations can be collectively termed scapular dyskinesis (dys = alteration of, kinesis = motion). Scapular dyskinesis refers to altered scapular motion and position that can be associated with shoulder symptoms. Because of the important but minimal bony stabilization of the scapula by the clavicle, dynamic muscle function is the major method by which the scapula is stabilized and purposefully moved to accomplish its roles. The predominant clinical finding demonstrating altered scapular motion or position is observation of prominence of the medial border of the scapula at rest or during motion. Scapular dyskinesis appears to be a nonspecific response to a painful condition in the shoulder rather than a specific response to certain glenohumeral pathology [3, 4]. This leads to excessive protraction of the scapula and depression of the acromioclavicular joint in all phases of motion which leads to increased symptoms of impingement. Scapular dyskinesis has multiple causative factors, both proximally (muscle weakness/imbalance, nerve injury) and distally (AC joint injury, superior labral tears, rotator cuff injury) based. The medial border prominence appears to be the result of abnormal muscle activations, either directly due to muscle involvement, such as inflexibility, weakness, and fatigue, or due to nerve injury, and is usually treated by rehabilitation.

The upper and lower trapezius muscles, which usually are activated independently, and the serratus anterior muscle contribute the most to scapular stability and mobility. Coupling of activation of these two muscles initiates upward rotation and posterior tilt. This force couple is especially active at the beginning of arm elevation and with arm elevation below 90°. As the arm elevation exceeds 90°, the lower trapezius is optimally

positioned to increase and maintain upward rotation through a direct line of pull. In this arm position, the serratus anterior works to stabilize the medial border against the thorax, acting as a regulator of scapular internal rotation. Lower trapezius activation is also important in the descent from maximum elevation, being activated eccentrically to control excessive anterior tilt. Other intrinsic muscles, the rhomboids and pectoralis minor, play important but not primary roles. Extrinsic muscles, mainly the latissimus dorsi and pectoralis major, create scapular motion through their effect as prime movers of the arm. Together the local and global muscles work together to provide concurrent stability and minimally constrained mobility. All muscles operate within the complex kinetic chain system where individual body segments or links have influence on multiple surrounding structures.

Tissue derangement both at or around the scapula and elsewhere within the kinetic chain can have deleterious effects on functional performance. Scapular dyskinesis has been found in association with almost every pathologic injury in the shoulder and arm in overhead athletes, including labral injury, impingement, instability, rotator cuff disease, acromioclavicular joint separations, and elbow MCL injury. The incidence varies, but dyskinesis can be identified in between 50 and 100 % of throwers with injuries. The dyskinesis is thought to be due to muscle inhibition caused by pain from the tissue derangement. It has also been hypothesized that lower extremity injury such as a history of ankle or knee injury can decrease shoulder function. It is suspected that alterations in the proximal links of the kinetic chain do not allow an athlete to achieve adequate stability prior to initiating force generation resulting in a reduction in the amount of energy being generated and transferred through the scapula and arm. Injury to any of the static restraints within the body may negatively affect arm function suggesting that surgical restoration of damaged tissue may need to be performed in order for scapular function to adequately return. Observed alterations in either the proximal or distal kinetic chain links should be initially treated conservatively with rehabilitation. However, if the alterations are addressed but dysfunction continues to exist, the most appropriate surgical intervention should be considered as a viable treatment option.

Impairments at or around the scapula such as muscle tightness and/or weakness can produce increased loads on local structures creating injury and can also negatively impact the desired biomechanical output during athletic tasks. This has been illustrated in overhead athletics, specifically baseball and tennis. Biomechanical assessments tend to show that individuals with altered scapular position and changes in glenohumeral range of motion (ROM) also exhibit altered muscle performance. These results show that a thrower's dominant shoulder that is positioned more anterior, in addition to having forward scapular position, displays decreased lower trapezius and serratus anterior muscle performance. Additionally those with increased external rotation ROM display decreased posterior rotator cuff and lower trapezius muscle performance. This suggests that alterations in resting position and available ROM are strongly related to muscle performance. It is therefore recommended that any observed alterations be treated only if they are found in association with injury.

From a biomechanical perspective, dysfunction of a particular segment in the chain can result in either altered performance or injury to a more distal segment. For example, the muscles of the shoulder girdle are not capable of generating the substantial angular velocities seen at the shoulder during throwing; the force is largely generated by the more proximal segments of the lower extremities and trunk. The substantial forces that are transferred to and subsequently reabsorbed by the distal segments at the shoulder and arm during throwing leave these segments vulnerable to injury. In a closed system such as the kinetic chain, alteration in one area creates changes throughout the entire system. This is known as the "catch-up" phenomenon where the changes in the interactive moments alter the forces in the distal segments. The increased forces place extra stress on the distal segments such as the scapula or shoulder, which often result in the sensation of pain or actual anatomic injury.

14.3 Clinical Presentation and Essential P/E

Most scapular-related problems in throwing athletes can be traced to loss of control of normal resting scapular position and dynamic scapular motion, resulting in alterations in the position or motion that produce a position and motion of excessive protraction. This position and motion, in the face of functional demands of the throwing or overhead motion, can create inefficiencies and deficits in the kinematics of the shoulder which can decrease performance and increase injury risk.

The history is an important part of the evaluation. Specific questions should be asked regarding past or present trauma to the scapula, clavicle, or AC joint, chronic or acute spinal symptoms, recent or remote hip or leg injuries, or any surgical procedures. It is also important to establish if the patients have had physical therapy for any of these conditions or for a scapular condition, to document the exact extent of the therapy, and to document the results. Therapy that emphasizes modalities, early open-chain rotator cuff exercises with resistance, shoulder shrugs, and shoulder protraction exercises has not been found to be effective for scapular dyskinesis. The poor outcomes are thought to occur because (1) only the symptoms (pain, irritation) are being addressed; (2) the scapular positions known to be related with dysfunction are encouraged prior to reestablishing scapular control, i.e., protraction before retraction; or (3) the exercises are too demanding for the non-stabilized scapula (long-lever maneuvers such as traditional rotator cuff exercises being implemented before short-lever maneuvers have been mastered).

The goals of the physical exam of the scapula are to establish the presence or absence of scapular dyskinesis; to evaluate joint, muscle, and bone causative factors; and to employ dynamic corrective maneuvers to assess the effect of correction of dyskinesis on symptoms. The results of the exam will aid in establishing the complete diagnosis of all the elements of the dysfunction and will help guide treatment and rehabilitation.

Dynamic examination of scapular motion can be reliably performed by clinical observation of the motion as the arm elevates and descends. This motion requires activation of the muscles to maintain the closed-chain mechanism of scapulohumeral rhythm. Failure to maintain this rhythm can result in increased scapular internal rotation, with consequent medial border prominence. Clinical observation of medial border prominence in symptomatic patients has been correlated with biomechanically determined dyskinesis, and this method is clinically reliable enough to be used as the basis for determination of the presence or absence of dyskinesis [5–7]. The examination is conducted by having the patients raise the arms in forward flexion to maximum elevation and then lower them three to five times with a 3–5 lb weight in each hand. Prominence of any aspect of the medial scapular border on the symptomatic side is recorded as "yes" (prominence detected) or "no" (prominence not detected).

The scapular assistance test (SAT) and scapular retraction test (SRT) are corrective maneuvers that can alter the injury symptoms and provide information about the role of scapular dyskinesis in the total picture of dysfunction that accompanies shoulder injury and needs to be restored [8, 9]. The SAT helps evaluate scapular contributions to impingement and rotator cuff strength, and the SRT evaluates contributions to rotator cuff strength and labral symptoms. In the SAT, the examiner applies gentle pressure to assist scapular upward rotation and posterior tilt as the patient elevates the arm. The major biomechanical effect of the SAT is increasing scapular posterior tilt by 7–10° throughout the entire arc of arm elevation. This test has shown "acceptable" inter-rater reliability. A positive result occurs when the painful arc of impingement is relieved and the arc of motion is increased which would be expected with increased scapular upward rotation. In the SRT, the examiner first grades the supraspinatus muscle strength following standard manual muscle testing procedures. The examiner then places and manually stabilizes the scapula in a retracted position. The biomechanical effects are a combination of increased external rotation and posterior tilt. A positive test occurs when the demonstrated supraspinatus strength is increased or the symptoms of internal impingement are relieved in the retracted position.

Although these tests are not capable of diagnosing a specific form of shoulder pathology, a positive SAT or SRT shows that scapular dyskinesis is directly involved in producing the symptoms and indicates the need for inclusion of early scapular rehabilitation exercises to improve scapular control [1, 2].

Coracoid-based inflexibility can be assessed by palpation of the pectoralis minor and the short head of the biceps brachii at their insertion on the coracoid tip. The muscles will usually be tender to palpation, even if they are not symptomatic in use, can be traced to their insertions on the ribs as taut bands, and will create symptoms of soreness and stiffness when the scapulae are manually maximally retracted and the arm is slightly abducted to approximately 40–50°.

A major portion of the scapular exam is the evaluation of the proximal kinetic chain and distal glenohumeral joint structures that affect scapular position and motion. Kinetic chain screening can be accomplished by the one-leg stability series—a combination of a standing balance test which assesses static control and a single-leg squat test which assesses dynamic control of the body over the planted leg. In the standing balance test, the patient is asked to place their hands over their chest and stand on one leg with no other verbal cue. Deviations such as a Trendelenburg posture or internally or externally rotating the weight-bearing limb indicates inability to control the posture and has been found to correlate with proximal core weakness especially in the gluteus medius. The single-leg squat is the next progressive evaluation. Assuming the same starting point as the standing balance test, the patient is asked to do repetitive partial half squats going down and returning to the standing position with no other verbal cues. Similar deviations in the quality of the movement are assessed as in the standing balance test. A Trendelenburg posture which may not be noted on standing balance may be brought out with a single-leg squat. The patient may also use their arms for balance or may go into an exaggerated flexed or rotated posture—"corkscrewing"—in order to put the gluteal or short rotator muscles on greater tension to compensate for muscular weakness.

14.4 Disease-Specific Clinical and Arthroscopic Pathology

The majority of scapular dyskinesis cases have root causes related to altered muscle function, i.e., strength imbalances, lack of flexibility, or altered muscle activation patterns. The kinematic alterations seen with scapular dyskinesis can also be due to neurological issues such as long thoracic or accessory nerve palsy which can be confirmed with diagnostic nerve conduction studies. Bony trauma such as a scapular fracture may be present which can be evaluated with standard radiographic imaging. Soft tissue injury involving the disruption of the rhomboids and/or lower trapezius from the scapular attachment sites has been recently described and labeled a scapular muscle detachment. Patients with this injury often have debilitating pain along the medial border of the scapula both at rest and during arm movement, limited use of arm function in forward elevation especially overhead motion, and pronounced scapular dysfunction similar to patients with neurological involvement. While tissue disruption is rare, it should be ruled out prior to making any treatment decisions.

More commonly, scapular alterations are due to inhibition of activation driven by pain from glenohumeral joint injury, strength imbalance among the scapular stabilizers, fatigue of muscle activation, or change in activation pattern. The serratus anterior and lower trapezius are often weak and display less activation intensity and increased latency, while the upper trapezius displays increased activation and decreased latency. This results in kinematic alterations of less posterior tilt, less external rotation, and less upward rotation motions, but increased elevation translation. These results have been found in athletes with impingement, instability, and labral tears.

Dyskinesis can also result from muscle or capsular stiffness caused by coracoid-based muscle inflexibility, i.e., pectoralis minor and short head of the biceps brachii. Tightness of these muscles decreases scapular posterior tilt, upward rotation, and external rotation. Similarly, pectoralis major and latissimus dorsi tightness can create dyskine-

sis through their action on the humerus. Glenohumeral internal rotation deficit, which is related to posterior muscle stiffness and capsular tightness, creates dyskinesis by producing a "windup" of the scapula into protraction as the arm rotates into follow-through.

Bony disruption such as clavicle fractures and high-grade acromioclavicular joint injuries may produce dyskinesis if the anatomy is not completely restored. Shortened malunions or nonunions decrease the length of the strut and alter the scapular position toward internal rotation and anterior tilt. In addition to changes in length, changes in clavicle curvature or rotation will affect scapular position or motion. Angulated fractures result in functional shortening and loss of rotation. The distal fragment in midshaft fractures often externally rotates, decreasing the obligatory clavicle posterior rotation and scapular posterior tilt during arm elevation. Acromioclavicular separations disrupt the strut function and allow a "third translation," in which the scapula translates inferior to the clavicle and medial on the thorax.

14.5 Treatment Options

Since it is most frequently an alteration of muscle activation, scapular dyskinesis is traditionally treated with conservative efforts focusing primarily on restoration of muscle flexibility, strength, and restoration of activation patterns. Any surgically treatable conditions must be fixed as a precondition for scapular rehabilitation. Examples are scapular muscle detachment; high-grade acromioclavicular separations; shortened, rotated, or non-united clavicle fractures; acromioclavicular joint arthrosis; and intraarticular glenohumeral pathology, such as labral or biceps injury, or rotator cuff pathology. All of these problems create anatomic or physiologic limitations to normal scapular function. Treatment of scapular dyskinesis starts with optimized anatomy, locally around the scapula, distally in the glenohumeral and acromioclavicular joints and clavicle, and proximally in the kinetic chain. These alterations will have been discovered in the evaluation process. Proximally, kinetic chain and core stability are key to optimal scapular kinematics. Most commonly, kinetic chain/core exercises are the first stage of scapular rehabilitation, followed by the restoration of scapular control, and end with strengthening of the rotator cuff and the larger muscles of the arm.

14.6 Rehabilitation

Functional tasks involving the scapula and shoulder most frequently are dependent upon appropriate functioning of the kinetic chain as a unit. This requires optimization of the individual kinetic chain segments and appropriate coordination of the individual segments. A typical progression to follow in order to assure each segment is optimized is (1) acquire flexibility of all segments involved, (2) establish core strength and stability, (3) facilitate critical kinetic chain links via sequential activation, (4) utilize a closed- to open-chain sequence of exercise, and (5) work in multiple planes. This progression has recently been described in detail [10]. A protocol template summarizing the appropriate exercises for each component is listed in Appendix A.

14.6.1 Acquire Flexibility

Flexibility of both the upper and lower extremity can be increased using various techniques and approaches with standard static, dynamic, and/or ballistic stretching being some of the options available to clinicians. Based on previous findings regarding flexibility deficits in upper extremity-dominant athletes, the hip extensor, flexor, and rotary muscle groups in addition to the knee flexor groups should be targeted for the lower extremity. Improving lower extremity muscle flexibility has been linked to improving lower body movement patterns and improving overall athletic performance. The muscles responsible for scapular stabilization and arm rotation specifically the pectoralis minor, latissimus dorsi, and posterior shoulder muscles should be the point of focus for the upper extremity.

14.6.2 Establish Core Strength

The local and global stabilizers of the trunk together provide optimal core stability. The larger global muscles (abdominal muscles, erector spinae, and hip abductors) are designed for power generation but also provide stability for upper extremity function. Core strengthening regimens have assisted clinicians in gaining strength of the pelvis and trunk muscles in patients with common ailments such as low back pain and shoulder impingement. In order to create a stable base, the rehabilitation protocols should focus on the local muscles (transverse abdominus, multifidi, abdominal obliques, and quadratus lumborum) which are responsible for segmental spinal stability and alignment. The core is the critical link between the development of and transfer of energy; therefore, an early focus on strength and stability is necessary for later stages of rehabilitation to be successful.

14.6.3 Facilitate Scapular Motion

Peri-scapular muscles such as the serratus anterior and lower trapezius should be a point of focus in early training and rehabilitation. Early training should incorporate the trunk and hip in order to facilitate proximal to distal sequencing of muscle activation. It is important to remember that scapular rotation is accessory in nature whereas scapular translation is physiologic or voluntary. Therefore, implementing exercises which attempt to isolate scapular rotation is not functional and should be discouraged. Utilizing the lower extremity in order to encourage scapular motion is ideal in that it mimics kinetic chain sequencing. Minimal stress is placed on the glenohumeral joint during hip and trunk extension which facilitates scapular retraction (Figs. 14.1 and 14.2). All exercises are started with the feet on the ground and involve hip extension and pel-

Fig. 14.1 Sternal lift. This maneuver is performed standing with the trunk and knees slightly flexed (**a**). The patient is instructed to extend the hips and trunk and retract the scapulae without moving the glenohumeral joint (**b**)

Fig. 14.2 Robbery exercise. The robbery maneuver begins with the knees and trunk flexed and the arms held away from the body (**a**). The patient is instructed to extend the hips and trunk and to "place the elbows in the back pockets," holding this final position for 5 s (**b**)

vic control. The patterns of activation are both ipsilateral and contralateral [11]. Diagonal motions involving trunk rotation around a stable leg simulate the normal pattern of throwing (Fig. 14.3). As the shoulder heals and is ready for motion and loading in the intermediate or recovery stage of rehabilitation, the patterns can include arm movement as the final part of the exercise.

Excessive scapular protraction does not allow optimal rotator cuff activation to occur. Rotator cuff strength can increase when the scapula is stabilized and retracted. The muscles responsible for performing scapular retraction can help control scapular protraction through eccentric control. When optimized, these muscles can properly maintain scapular stability thus decreasing excessive protraction with arm movement. For this reason, the early phases of training should focus on scapular strengthening in an attempt to restore normal scapular kinematics rather than placing an early emphasis on rotator cuff strengthening as performed in more traditional rehabilitation protocols.

14.6.4 Early Closed-Chain Implementation

Kinetic chain-based rehabilitation activities have been grouped into open and closed chain. Typically, when soft tissue is pathologic, closed-chain exercises are implemented early in the rehabilitation process. There are three components which make usage of closed kinetic chain exercise advantageous in early rehabilitation. First, the exercise environment can be controlled. This allows the focus to be taken away from the arm as an integrated unit with high dynamic demands and place it in a stable, axially loaded, static setting. Second, closed-chain exercise is ideal for working "at" specific ranges of motion compared to working "through" a range of motion. Finally, closed-chain exercise allows the rotator cuff and scapular musculature to be unloaded by decreasing the amount of force generated and stress applied to the involved soft tissue. These types of exercises are best suited for reestablishing the proximal stability and control in the links of the kinetic chain such as the pelvis

Fig. 14.3 Lawn mower exercise. The lawn mower begins with the hips and trunk flexed and the arm slightly forward elevated (**a**). The patient is instructed to extend the hips and trunk, followed by rotation of the trunk to facilitate scapular retraction (**b**)

and trunk. Open-chain exercises, which generate greater loads in comparison to closed-chain activities, should be utilized later in rehabilitation programs due to their increased demand on the soft tissue due to the longer arm levers these exercises require.

The rationale behind the closed-chain framework is to maximize the ability of the inhibited muscles to activate. This involves placing the extremity in a closed-chain position, emphasizing normal activation patterns, and focusing on the muscle of interest by deemphasizing compensatory muscle activation. For example, if a patient presents with shrugging during arm elevation, then it can be assumed that the lower trapezius and/or serratus anterior is not working effectively enough during the dynamic task. A closed-chain exercise such as the low row should be utilized because the short-lever positioning in conjunction with the pelvis and trunk acting as the driver facilitates lower trapezius and serratus anterior co-activation which decreases the activation of the upper trapezius (Fig. 14.4). This is the normal muscle activation pattern for scapular retraction and depression. Once the normal activation pattern has been restored, then more challenging isolated exercises can be employed.

14.6.5 Work in Multiple Planes

Strengthening and stabilization should begin by emphasizing work in successful planes and then progress to deficient planes. Clinicians should avoid the use of single planar exercises which isolate specific muscles or specific joints. Greater

Fig. 14.4 Low row exercise. The patient is positioned standing with the hand of the involved arm against the side of a firm surface and legs slightly flexed (**a**). The patient should be instructed to extend the hips and trunk to facilitate scapular retraction and hold the contraction for 5 s (**b**)

isolation should be utilized in the later stages of the rehabilitation protocol. During the early phases, emphasis should be placed on achieving successful positions, motions, and muscle activation sequences. In this manner, normal physiologic activations are restored, which lead to restoration of normal biomechanical motions.

Most activities, whether they are sports-related or normal daily movements, occur in the transverse plane. Therefore, the transverse plane should be exploited particularly in the early phases of rehabilitation. The protocol should progress to more unilateral planes as normal scapulohumeral kinematics are restored. Exploitation of the transverse plane and exaggeration of trunk motion help accentuate both scapular retraction and protraction. By forcing proximal stability, the hip and trunk muscle activations, which have been demonstrated to precede arm motion, will be more effective during a specified task (Fig. 14.5). In addition to generating and transferring energy to the distal segments, this component of rehabilitation allows the utilization of the stable base for arm motion and forces the lower extremity and core to drive the arm (Figs. 14.6 and 14.7). Rehabilitation programs should attempt to encourage stimulation of proper proprioceptive feedback as well, so the patient can return to their desired level of function.

14.7 Advantages/Pitfalls/Complications

The primary advantage of considering scapular function as a component of overall shoulder function is that scapular considerations allow the clinician to employ a comprehensive approach of assessment and treatment. Such an approach helps to eliminate placing sole focus on the site of symptoms and instead direct attention toward potential causes of symptoms and/or dysfunction. Adhering to the kinetic chain model of function is also helpful in appreciating the influence the scapula can have on arm function because of the understanding of segmental motion and energy transfer.

Fig. 14.5 (**a**, **b**) Lawn mower with lateral step. By adding the step, the patient is forced to utilize the hip abductors making the exercise more functional

However, dyskinesis is nonspecific and as a result can be either overlooked as a potential impairment or mistakenly thought to be a pathologic entity. It is challenging to reestablish activation patterns and strength. To effectively re-create optimal muscle activation patterns, clinicians must have an intimate understanding of adequate scapular kinematics as well as an appreciation of kinetic chain function. This necessary knowledge has been absent for some time but has just recently become available through published consensus statements developed by experts in scapular function [3, 9]. Prior to these publications, clinical expertise in evaluating and managing the scapula had been lacking. In order to restore scapular stability, the serratus anterior and lower trapezius muscles should be addressed during rehabilitation. Strengthening these muscles will allow maximal rotator cuff strength to be achieved off a stabilized, retracted scapula. However, contrary to traditional rehabilitation philosophies, rotator cuff emphasis in rehabilitation should be after scapular control is achieved. Increase in impingement pain when doing open-chain rotator cuff exercises indicates the wrong emphasis at the wrong stage of the rehabilitation protocol. Additionally, muscle strength is typically developed over the span of 4–6 weeks, so adequate time must be allotted for regaining the necessary strength for functional scapular stabilization.

Fig. 14.6 (**a**, **b**) Robbery exercise with posterior step. Utilizing the step allows the lower extremity to drive the upper extremity

14.8 Experience in Treatment of Athletes

Athletes tend to have scapular alterations often as a result of sports-specific demands. This has been shown repeatedly in overhead athletes involved in baseball, softball, tennis, and swimming. In some cases, these alterations can be considered positive adaptations such as the bony remodeling of the humerus in skeletally immature leading to increased external rotation in cocking which results in greater development of velocity. However, not all adaptations lead to positive results. Scapular dyskinesis can develop as a result of focusing exclusively on larger muscles during strengthening programs rather than comprehensive regimens. Additionally, the repetitious nature of overhead athletics has short- and long-term effects on the soft tissue of the shoulder with acute decreases in glenohumeral internal rotation, horizontal adduction, and total range of motion. Loss of motion in one or more of these directions can affect scapular function and lead to future injury. Routine implementation of stretching and strengthening maneuvers which target the surrounding soft tissue of the scapula and glenohumeral joint, both preemptively and post-activity, can be effective at reducing the occurrence of injury.

Another consideration is that muscle endurance can be lacking in overhead athletes. General arm pain not generated by disrupted anatomy or kinetic chain deficit suggests that the extremity is not conditioned to handle the required repetitive tasks, is being used too often, or is being used incorrectly. Implementing conditioning programs which are designed to build muscle endurance, i.e., low-load, high-repetition programs, may help increase the necessary muscle endurance.

Fig. 14.7 (**a**, **b**) Low row with posterior step. The posterior step back requires hip and trunk extension which facilitates scapular retraction and depression

However, even when muscle physiology has been optimized, excessive use without appropriate recovery time can lead to muscular fatigue which in turn decreases muscular activity and force production, subsequently causing biomechanical abnormalities (decreased cocking, dropped elbow), all of which can result in pain or soreness. Adequate rest and recovery should be allotted in order for muscular function to be less affected by the stress of physical activity. Finally, kinetic chain function should be integrated throughout all rehabilitation and conditioning programs. From the early phases of rehabilitation through the functional phases of sports-specific conditioning, utilization of the kinetic chain encourages adequate muscle activation, proper motor pattern development, and optimal performance output.

The scapula plays multiple key roles in normal scapulohumeral rhythm and shoulder function. Alterations of scapular resting position and dynamic motion, collectively termed scapular dyskinesis, are associated frequently with many shoulder injuries in throwing athletes. The clinical exam for presence or absence of scapular dyskinesis as well as physical impairments within the kinetic chain is best achieved through observational assessments. If scapular dyskinesis is present, corrective maneuvers may be used to determine the effect of dyskinesis on shoulder symptoms. If deficits within other segments of the kinetic chain exist, they should also be addressed as part of the comprehensive rehabilitation regimen for treating scapular dysfunction. Scapular control in a position of retraction, external rotation, and posterior tilt should be a key detriment of return-to-play status and should therefore be optimized prior to releasing athletes to their respective activities.

14.9 Appendix A: Kinetic Chain-Based Scapular Strengthening Guidelines

14.9.1 A.14.1 Phases of Rehabilitation

14.9.1.1 A.14.1.1 Phase I: Acute Phase (Weeks 1–2)

Pearls

- Acquire flexibility
  - Upper extremity
    - Sleeper stretch (posterior shoulder muscles)
    - Open book stretch (pectoralis muscles)
    - Corner stretch (pectoralis muscles)
    - Door frame stretch (latissimus dorsi)
  - Lower extremity
    - Hip rotation
    - Hip extension
    - Hip flexors
    - Other maneuvers as needed
  - Manual joint mobilizations permitted as allowed by tissue integrity
- Establish core strength and stability
  - Lower extremity strengthening focusing on hip abduction and extension recommended
    - Lateral step
    - Step downs
    - Lunge progression
    - Physioball exercises

Goals

- No limitations of muscle or capsular tightness
- Establish trunk/hip motion and strength for quality scapular motion later

14.9.1.2 A.14.1.2 Phase II: Recovery Phase (Weeks 3–5)

Pearls

- Facilitate critical kinetic chain links
  - Facilitate retraction
    - Lawn mower (Fig. 14.3)
    - Robbery (Fig. 14.2)
- Utilize closed- to open-chain exercise
  - Closed chain
    - Table slides (for integrated motion)
    - Low row (Fig. 14.4)
    - Inferior glide (Fig. 14.8)

Fig. 14.8 Inferior glide. This maneuver encourages co-contraction of local and global muscles resulting in depression of the humeral head

  - Open chain
    - Low row with step back (Fig. 14.7)
    - Lawn mower with step back (Fig. 14.5)
    - Robbery with step back (Fig. 14.6)

Goals

- Lower extremity driving upper extremity motion
- Full active range of motion
- Adequate scapular control to progress to longer-lever exercise maneuvers

14.9.1.3 A.14.1.3 Phase III: Functional Activity Phase (Weeks 5±)

Pearls

- Work in multiple planes
  - Integrated motion
    - Punching
    - Power position (Fig. 14.9)
    - Power position with step back (Fig. 14.10)
  - Traditional rotator cuff exercises
    - Scaption
    - Horizontal abduction
    - Internal and external rotation

Goals

- Fine-tune scapular motion to alleviate all dyskinesis
- Increase strength and endurance of rotator cuff and scapular stabilizing muscles

Fig. 14.9 Power position. The athlete is positioned standing with dominant arm in 90/90 position and forearm pronated (**a**). The athlete is instructed to rotate the trunk without moving the feet while maintaining the 90/90 position of the arm (**b**). The forearm should be allowed to supinate to imitate the act of the overhead throwing

Fig. 14.10 (**a**, **b**) Power position with step back. This maneuver requires stability of the lower extremity in order for the upper extremity positioning to be achieved

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Scapular Dyskinesis: Part II. A New Diagnostic Modality – Three-Dimensional Wing CT

15

Jin-Young PARK and Jung-Taek Hwang

15.1 Introduction

Scapular dyskinesis is defined as observable alterations in the position of the scapula and the patterns of scapula motion in relation to the thoracic cage [1–4]. Scapular dyskinesis most frequently occurs as a result of alteration of muscle activation or coordination. It has been thought that scapular dyskinesis affects normal scapulohumeral rhythm (SHR) and shoulder arthrokinematics and, therefore, plays a role in producing the dysfunctions associated with some shoulder and elbow pathologies [2–13]. It is important that a proper rehabilitation is performed according to the type of scapular dyskinesis, especially for elite athletes. There are several systems for assessing scapular dyskinesis. One is the standard observational typing system by Kibler [1, 2, 14]. And another is an assessment using 3-D wing CT with a high inter-rater reliability (IRR) [3, 4]. The other systems include fringe projection technique [15], double calibration methods [16], infrared cameras [17], and 3-D tracking [2, 17]. 3-D scapular motion studies have some inherent problems like the overlying skin movement, choice of body landmarks, identification of these landmarks by palpation, and definition of rotation angles. But the assessment of scapular dyskinesis using 3-D wing CT can exclude these problems.

15.2 Observational Types of Scapular Dyskinesis

To assess scapular dyskinesis accurately, Kibler introduced the observational typing method, which is considered as the gold standard [1, 2, 14]. A type 1 scapular dyskinesis is characterized by prominence of the inferior medial scapular angle and would be associated with excessive anterior tilting of the scapula. A type 2 scapular dyskinesis is characterized by prominence of the entire medial border and would be associated with excessive scapular internal rotation. A type 3 scapular dyskinesis is characterized by prominence of the superior scapular border and would be associated with excessive superior translation of the scapula. A type 4 scapular dyskinesis is characterized as normal, with no asymmetries identified and no prominence of the medial or superior border observed [1–3, 14] (Table 15.1, Fig 15.1). Another observational assessment method is the "yes/no" method, which collapses three dyskinesis categories (types

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Table 15.1 Scapular dyskinesis system used to categorize abnormal scapular motion

Pattern	Definitions
Type 1	The prominence of the inferior medial scapular angle. It would be associated with excessive anterior tilting of the scapula
Type 2	The prominence of the entire medial border. It would be associated with excessive scapular internal rotation
Type 3	The prominence of the superior scapular border. It would be associated with excessive superior translation of the scapula
Type 4	It is normal, with no asymmetries identified and no prominence of the medial or superior border observed

Kibler et al. [1, 29]

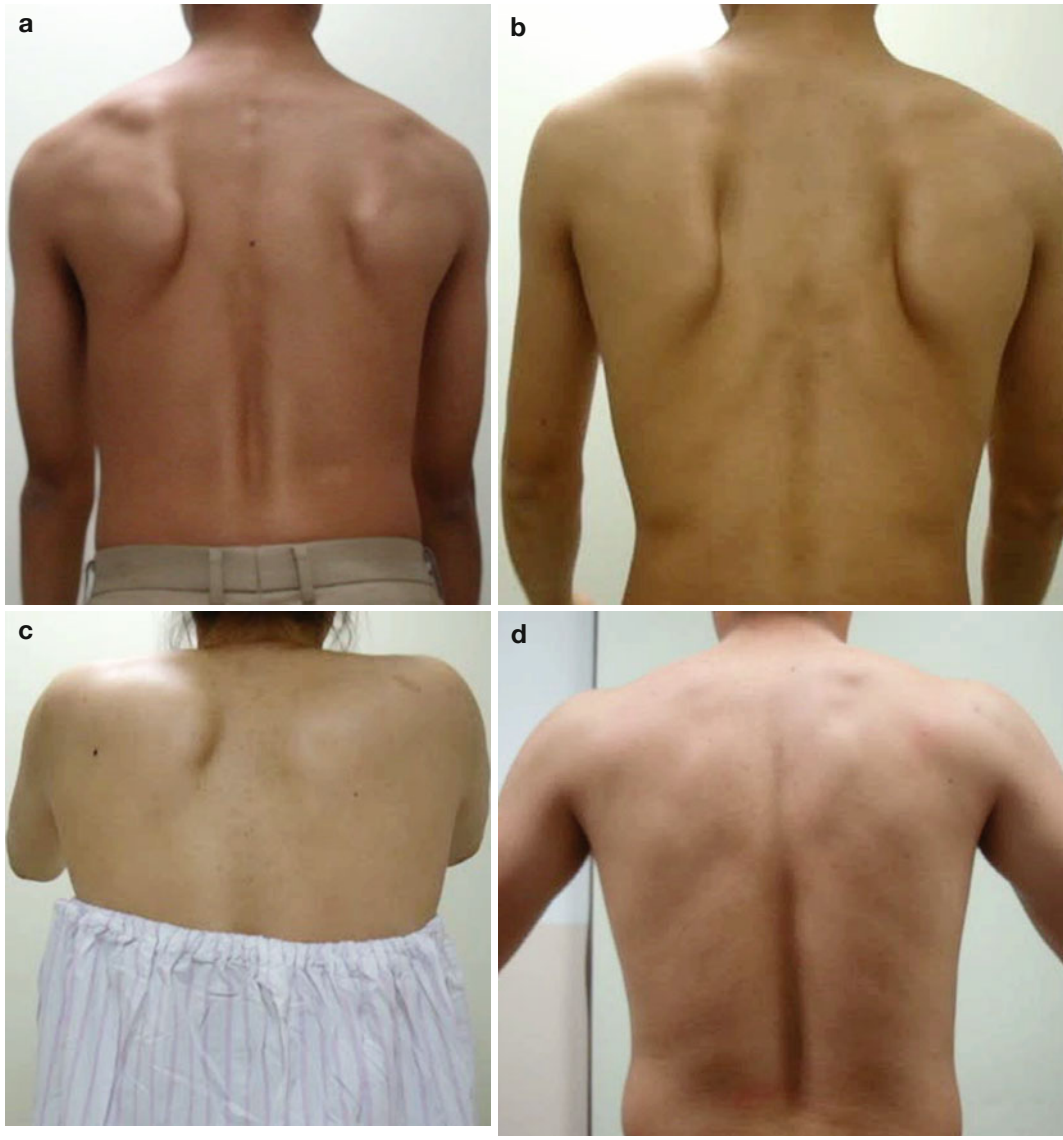

Fig. 15.1 Scapular dyskinesis. (**a**) Type 1 dyskinesis, with inferior medial border prominence (both scapulae, but left is more definite.). (**b**) Type 2 dyskinesis, with prominence of the entire medial border (both scapulae, but left is more definite). (**c**) Type 3 dyskinesis, with prominence of the superior medial border (left scapula). (**d**) Type 4 dyskinesis, with normal scapular motion and position (both scapulae)

1–3) into a single category of "yes" (an abnormal pattern was observed) and designates the normal ones as "no" [2, 11, 18].

15.3 Assessment of Scapular Dyskinesis Using 3-D Wing CT

There are two studies using 3-D wing CT in assessing scapular dyskinesis. In these studies, the 3-D wing CT images were acquired using a 16- or 64-slice multidetector CT (MDCT) scanner (LightSpeed Pro16 or LightSpeed VCT, GE Healthcare, Little Chalfont, Bucks, UK). Each subject was examined in a supine position or prone position with the arms at the side of the body and the palms toward the body. The 3-D wing CT images depicted the thoracic region, consisting of bilateral scapula, bilateral clavicle, and the spine from the C7 to T7 vertebra [3, 4].

15.3.1 Five Motions of Scapula and Measurement of Five Angles of Scapular Position in 3-D Wing CT

15.3.1.1 Five Motions of Scapula

Scapular movement is a composite of three motions – upward/downward rotation around a horizontal axis perpendicular to the plane of the scapula, internal/external rotation around a vertical axis through the plane of the scapula, and anterior/posterior tilt around a horizontal axis in the plane of the scapula. The clavicle acts as a strut for the shoulder complex, connecting the scapula to the central portion of the body. This allows two translations to occur – upward/downward translation on the thoracic wall and retraction/protraction around the rounded thorax [18–20] (Fig. 15.2).

We adopted upward rotation (UR), superior translation (ST), anterior tilting (AT), protraction (PRO), and internal rotation (IR) as the representative five motions of scapula for simplification. Upward rotation is outward movement of inferior angle of the scapula as the arm is elevated. Superior translation is superior migration of the scapula with no medial or lateral movement. Anterior tilting of the scapula means that the acromion moves anteriorly and the inferior angle of the scapula moves posteriorly. Protraction is lateral translation of the medial border of the scapula without superior movement or rotation. Internal rotation means that the scapula rotates internally around a vertical axis. These are the three rotational movements and two translations of the scapula [3, 4, 18–20].

15.3.1.2 Measurement of Five Angles of Scapular Position in 3-D Wing CT

Because the acromioclavicular (AC) joint undergoes no translational movement during scapular rotation, the three bony landmarks defined were the inferomedial angle (IMA) of the scapula, the AC joint, and the root of the scapular spine (RSS). The measurement criteria included five movements of the scapula, consisting of three rotations and two translations: upward rotation, internal rotation, anterior tilting, superior translation, and protraction. Angular denominations were chosen for measurements to avoid false impressions due to anatomic variations, such as scapular size, scoliosis, and different acromion shapes [3, 4].

To avoid discrepancies in the measurement criteria, the midpoint of the AC joint was designated as the AC joint, the most caudal point of scapula as the IMA, and the most medial point of the scapular spine as the RSS. The five angles of scapular movement were measured, as below, with reference from several previous reports. There were a few modifications in measuring angles of scapula because of the 3-D wing CT setting [3, 4].

UR Angle

The UR angle is measured as the angle between the extension of line from the AC joint to RSS and the vertebral axis (C7–T7) on a posterior-coronal view [3, 4, 17, 18, 21] (Fig. 15.3a).

IR Angle

It is the angle between the line joining the two AC joints and the line from the corresponding AC joint to RSS on a superior-axial view [3, 4, 17, 21, 22] (Fig. 15.3b).

AT Angle

It is the angle between the line from IMA parallel to the medial border of scapula and the line joining the anterior tips of C7 and T7 vertebrae on a lateral-sagittal view [3, 4, 17, 21] (Fig. 15.3c).

ST Angle

It is the angle between the line from the AC joint to the midpoint of the spinous process of the C7 vertebra and the vertebra axis (C7–T7) on a posterior-coronal view [3, 4, 18, 19] (Fig. 15.3d).

PRO Angle

It is the angle between the line parallel to the vertebral axis (C7–T7) and the line from the corresponding AC joint to the center of the C7 vertebral body on a superior-axial view [3, 4, 17, 21] (Fig. 15.3e).

15.3.2 Statistical Analysis of Correlation Between the Five Angles of Scapular Position on 3-D Wing CT and the Observational Types of Scapular Dyskinesis

There are two studies about assessment of scapular dyskinesis using 3-D wing CT. In an earlier study of them, 89 athletes (178 shoulders) were videotaped and 7 blinded observers categorized scapular dyskinesis into 4 types, which was

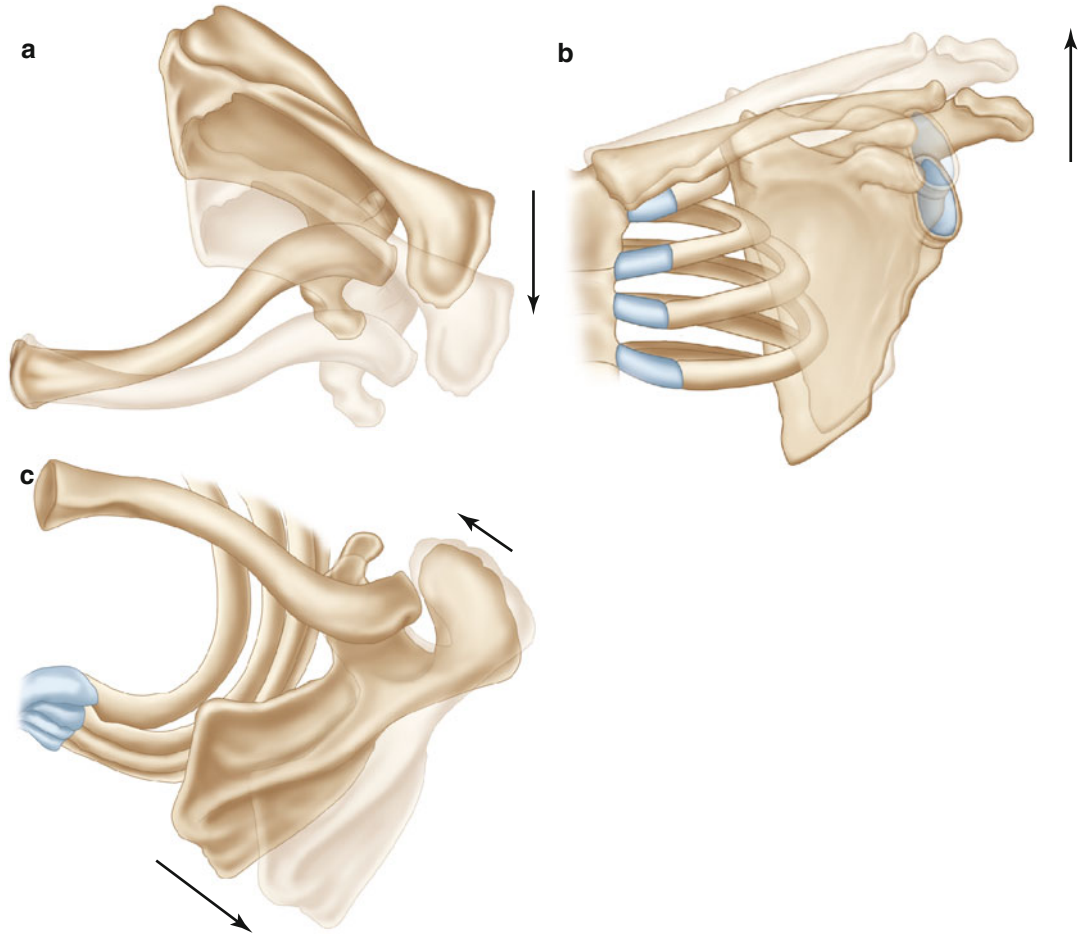

Fig. 15.2 Motions of the scapula. (**a**) Protraction. (**b**) Superior translation. (**c**) Internal rotation. (**d**) Upward rotation. (**e**) Anterior tilting

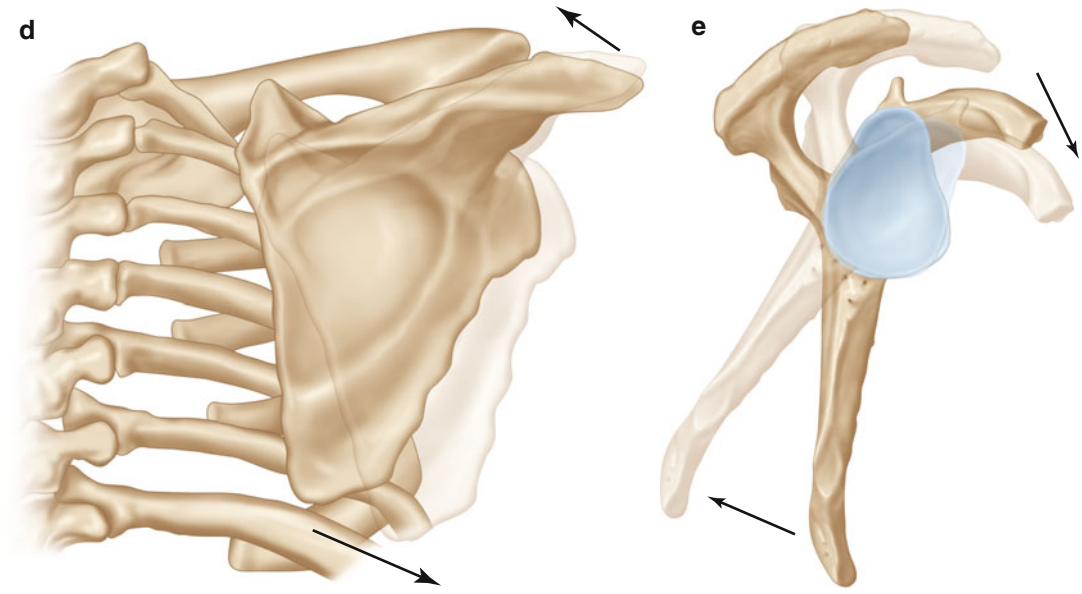

Fig. 15.2 (continued)

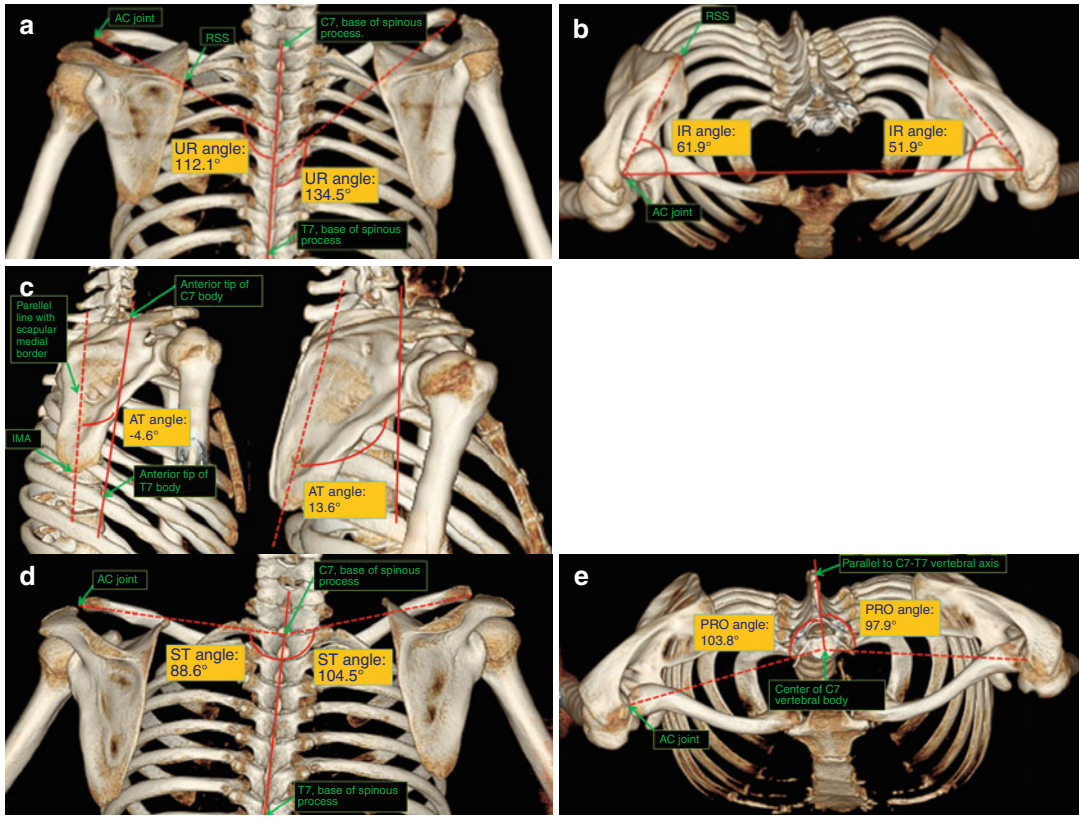

Fig. 15.3 The measurement of 5 angles on 3-D wing CT. (**a**) Upward rotation. (**b**) Internal rotation. (**c**) Anterior tilting. (**d**) Superior translation. (**e**) Protraction (Park et al. [4]). *AC joint* acromioclavicular joint, *IMA* inferomedial angle, *RSS* root of scapular spine

followed by 3-D wing CT in supine position. Four blinded examiners evaluated five angles [UR, IR, AT, ST, and PRO angles]. The inter-rater reliability (IRR) with observational assessment of scapular dyskinesis was good. The 3-D wing CT analysis had a very high IRR. There was a statistically significant correlation between observational assessment and 3-D wing CT analysis. The UR angle as well as the ST angle in type 3 scapular dyskinesis and the AT angle in type 1 scapular dyskinesis were increased as compared with those in the other types of scapular dyskinesis, and the difference was statistically significant. The IR angle in type 2 scapular dyskinesis was increased as compared with that in normal scapular motion, and the difference was statistically significant. The PRO angle in type 1 scapular dyskinesis was increased as compared with that in normal scapular motion, and the difference was also statistically significant [3].

In a more recent study of them, the 330 scapular movements of 165 patients were classified into 4 types by 7 blinded observers. Then, 3-D wing CT was performed with patients prone, and four blinded observers measured the above five angles [4]. UR and ST angles were significantly larger in type 3 more than in the other types, and the AT angle showed a similar pattern in type 1. The PRO angle was significantly larger in types 1, 2, and 3 more than in type 4, and the IR angle was significantly larger in type 2 more than in the other types [4]. The CT measurement in the more recent study showed a similar pattern with the previous study, but with some differences. First, the IR angle in type 2 increased significantly compared with those in other types, whereas the UR angle and ST in type 3 and the AT angle in type 1 are similar to our previous study. Second, the PRO angle increased significantly in types 1, 2, and 3 compared with that in type 4, whereas the IR angle increased only in type 1 in our previous study. Third, the cutoff values were determined for each angle to classify scapular dyskinesis according to the five angles. Fourth, the correlation among the classification according to the five angles and concomitant diseases was analyzed statistically [3, 4].

15.3.3 Cutoff Values of the Five Angles of Scapula in 3-D Wing CT in Prone Position According to a Significant Correlation with Each Observational Type

The cutoff values were determined for each angle among the types showing significant differences using the ROC curve (Table 15.2). The cutoff values of the five angles were UR, 117°; ST, 90°; AT, 8°; PRO, 99°; and IR, 51° [4].

15.3.4 Correlation Between Concomitant Diseases and the Five Angles of Scapular Dyskinesis in 3-D Wing CT According to the Cutoff Values

The statistical analysis showed a correlation between a classification using the cutoff value and the concomitant diseases of the affected side. The UR angle showed a correlation with osteo-

Table 15.2 Cutoff values by receiver-operating characteristic (ROC) curve (Park et al. [4])

Angle	Group vs. group	Cutoff value by ROC curve °	Sensitivity (%)	Specificity (%)
UR angle	Type 3 vs. types 1, 2, 4	117	84.6	74.1
ST angle	Type 3 vs. types 1, 2, 4	90	80.8	63.0
AT angle	Type 1 vs. types 2, 3, 4	8	94.6	84.5
PRO angle	Types 1, 2, 3 vs. type 4	99	70.0	74.0
IR angle	Type 2 vs. types 1, 3, 4	51	75.5	75.0

UR upward rotation, *ST* superior translation, *AT* anterior tilting, *PRO* protraction, *IR* internal rotation

chondritis dissecans (OCD) of the elbow and glenohumeral internal rotation deficit (GIRD), and the AT and IR angles showed a correlation with multidirectional instability (MDI) of the shoulder. In addition, a relation between IR angle and a Bennett lesion of the shoulder showed a borderline significance. Among them, the UR angle showed a more significant correlation with GIRD and IR angle with MDI [4].

The significant correlations among the measured angles and concomitant diseases in this study correspond to previous studies. A study in 2010 presented that collegiate baseball players had more GIRD than high school players and that the former had less scapular upward rotation than the latter [23]. This result suggests that players who have more GIRD show a lesser scapular upward rotation. Another study reported that individuals with MDI demonstrated a significant decrease in scapular upward rotation in scapular plane abduction and a significant increase in scapular internal rotation during scapular plane abduction compared with asymptomatic controls [24].

15.3.5 Rehabilitation Treatment of Scapular Dyskinesis Using 3-D Wing CT Assessment

It is important to assess scapular dyskinesis precisely for the enhancement of sports performance, the treatment of concomitant disorders, and the prevention of sports injury [25–27]. And based on the measured angles on the 3-D wing CT and the cutoff values, the accurate rehabilitation can be performed. Because a more accurate quantification of improvement during rehabilitation can be obtained using 3-D wing CT, it is convenient to determine the type, intensity, and duration of rehabilitation. Rehabilitation of scapular dyskinesis in our clinic is based on the routine proximal to distal protocol [1, 20] (see Chap. 33). At the initial visit, observational typing was performed for suspicious scapular dyskinesis patient on the history taking and routine physical examination. 3-D wing CT was checked for the patient strongly suspected with scapular dyskinesis on the result of the observational typing. According to the 3-D wing CT assessment, the rehabilitation of scapular dyskinesis was determined. After 3–4 months of the routine rehabilitation, observational and 3-D wing CT assessments were rechecked and the degree of improvement was evaluated based on the measured five angles, the cutoff value, and the relief of symptom.

15.4 Advantages and Disadvantages

The assessment of scapular dyskinesis using 3-D wing CT showed a higher IRR [3, 4] (0.972–0.981) than the other observational typing methods [3, 28, 29] (IRR: 0.186–0.780). The observational assessment of scapular dyskinesis has several problems that result in a low IRR [11]. First, the overlying muscles and soft tissues are obstacles to the assessment [30, 31]. Second, assessment methods should consider three rotational movements and two translations of the scapula, but clinical observational assessment should use static measures to evaluate the scapula in one plane or, at most, two planes [2, 30].

In particular, the assessment of scapular dyskinesis using the 3-D wing CT in prone position has several advantages. First, the 3-D wing CT image in the prone position showed an unblocked increase in the IR and PRO angles, especially in type 2, compared with that in the supine position. Second, the new classification was made using the cutoff values, which can be used for the diagnosis, treatment, and rehabilitation of patients with scapular dyskinesis. Third, correlations of scapular dyskinesis with concomitant diseases were partly found, and these correlations can be used for searching the pathology and treatment of concomitant diseases.

On the other hand, the assessment of scapular dyskinesis using the 3-D wing CT in prone position has several disadvantages. First, 3-D wing CT imaging in the prone position was beneficial in eliminating the effect of gravity, but gravity could exaggerate the IR and PRO angles in type 2. Second, the only participants of this

study were symptomatic athletes. There was no non-symptomatic control group. Third, there was an exposure to radiation. To perform 3-D wing CT imaging, the scapula, spine, breast, and other tissues should be scanned and, therefore, are exposed to radiation. A radiation dose of advantage CT scan was about 1,000 millirems, and the international standard annual dose limit is 5,000 millirems. Therefore, the CT scan can be performed two to three times per year with an interval of more than 3 months. Fourth, the 3-D wing CT method is more expensive than the observational assessment. However, 3-D wing CT analysis can be cost-effective, owing to a higher IRR than the observational method. Fifth, 3-D wing CT methods assess static motion rather than dynamic motion. However, the two studies about the 3-D wing CT analysis of scapular dyskinesis proved the statistically significant correlation between the static position and the dynamic motion of the scapula. Finally, the 3-D wing CT imaging in this study was performed only with patients in prone position with arms at their sides. There are several important positions of the scapula, such as standing with arms at the side or with arms abducted, supine with arms abducted, and prone with arms abducted [4].

15.5 Experience in Treatment of Athlete

A 31-year-old male professional archer visited our clinic complaining of right shoulder pain and click sound in full draw phase lasting 3 months. On routine physical examination, X-ray, and MR arthrography, he was diagnosed with posterior labral fraying and tendinitis of biceps long head, which were treated conservatively. On the observational assessment of scapular dyskinesis, type 1, 2 scapular dyskinesis was suspected on the affected shoulder and 3-D wing CT in prone position was performed. On the 3-D wing CT analysis, the five angles of both shoulders were as follows: right/left, UR, 130°/116°; IR, 59°/53°; AT, 1°/4°; ST, 100°/98°; and PRO, 103°/98°. The routine rehabilitation was performed including periscapular exercise to make a symmetric scapular motion. On the follow-up of 3 months, observational and 3-D wing CT assessments were rechecked. On the observational assessment, there was no scapular dyskinesis. On the 3-D wing CT analysis, the five angles of both shoulders were as follows: right/left, UR, 114°/106°; IR, 45°/44°; AT, 8°/7°; ST, 89°/88°; and PRO, 96°/97°. UR, IR, ST, and PRO angles of the affected shoulder improved after the routine rehabilitation [4] (Fig. 15.4).

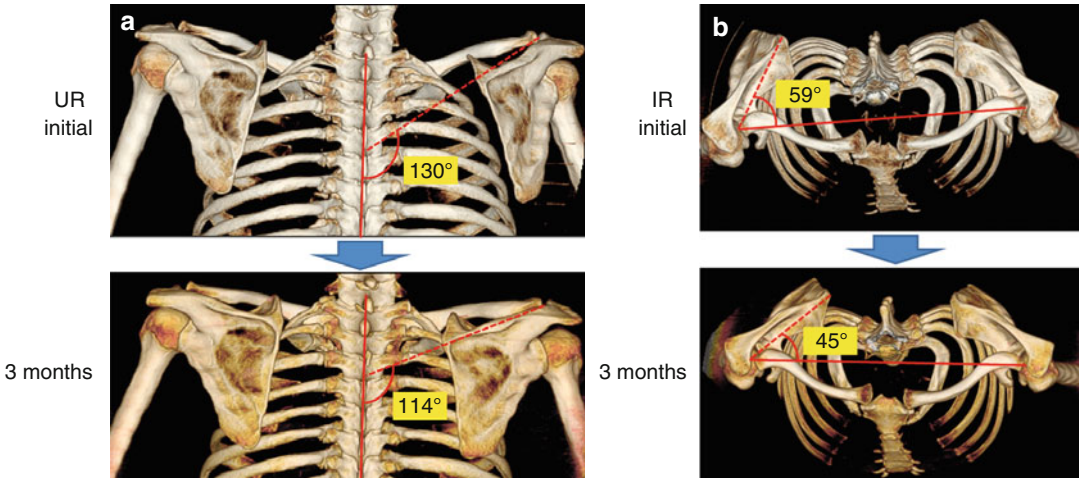

Fig. 15.4 3-D wing CT follow-up of a professional archer with right-sided scapular dyskinesis. (**a**) The upward rotation (*UR*) angle improved from 130° to 114°. (**b**) The internal rotation (*IR*) angle improved from 59° to 45°. (**c**) The superior translation (*ST*) angle improved from 100° to 89°. (**d**) The protraction (*PRO*) angle improved from 103° to 96°

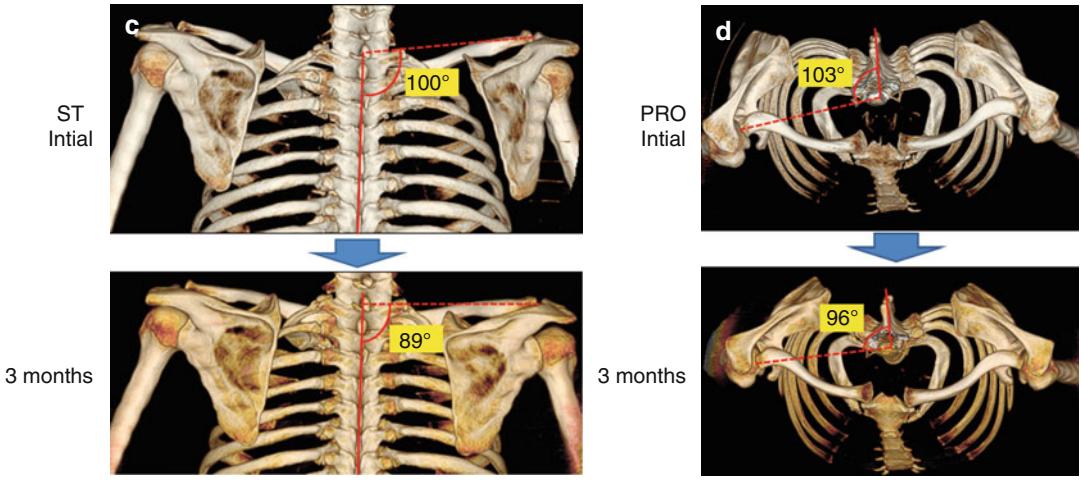

Fig. 15.4 (continued)

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16 Posterosuperior and Anterosuperior Impingement in Overhead Athletes

Eugene W. Brabston, Balazs Galdi, and Christopher S. Ahmad

16.1 Introduction

Repetitive overhead-throwing maneuvers not only place the shoulder in the extreme ranges of motion but also expose the shoulder to supraphysiologic amounts of stress. In fact, professional baseball pitchers generate up to 92 Nm of humeral rotation torque, which is greater than the torsional failure limit in human cadaveric shoulders [52]. Not surprisingly, throwing athletes may often develop significant shoulder pathology. Over the past century, there has been an increased awareness and understanding of the etiology of shoulder pain in overhead-throwing athletes. In 1959, Bennett hypothesized that baseball pitchers were prone to posterior shoulder pain secondary to repetitive traction on the triceps tendon insertion site which led to posterior capsular inflammation [3]. Several years later, while operating on patients with shoulder pain during the late cocking stages of throwing, Lombardo and colleagues noted ossification and excess fibrous tissue in the posterior aspect of the capsule [38]. In 1985, Andrews and colleagues noted that pitchers were prone to partial tears of the supraspinatus tendon from repetitive overuse but did not describe a specific mechanism leading to this [1].

In 1989, Jobe and colleagues reported an association between anterior instability and rotator cuff impingement and found that subacromial decompression has very limited success rate in overhead athletes [30]. In 1991, Walch and colleagues reported impingement between the deep side of the supraspinatus tendon and the posterosuperior edge of the glenoid cavity in a young sports thrower when the arm was in abduction-retropulsion and in forced lateral rotation [61]. However, it was not until a year later that the first clinical evidence to support internal impingement with a specific mechanism was described [60]. In their series, seventeen athletes presenting with unexplained shoulder pain on throwing underwent arthroscopic examination. With the arm placed in full external rotation and 90° abduction (the throwing position), impingement was found between the posterosuperior border of the glenoid and the undersurface of the tendinous insertions of supraspinatus and infraspinatus.

16.2 Pathoanatomy, Biomechanics, and Preferred Classification

Internal impingement occurs when the rotator cuff becomes entrapped between the humeral head glenoid labrum. Depending on the

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pathologic changes that have occurred, this impingement may occur at numerous locations. Posterosuperior impingement is much more common and occurs when the supraspinatus and infraspinatus become entrapped between the greater tuberosity and posterosuperior aspect of the glenoid labrum with the arm in 90° of abduction and full external rotation. On the contrary, anterosuperior impingement involves impingement of the subscapularis tendon between the anterior humeral head and anterior glenoid labrum with the arm in forward flexion, adduction, and internal rotation. Because these are separate entities occurring in different sets of patients, we will describe them each individually.

16.2.1 Posterosuperior Impingement

The dominant shoulder of a throwing athlete often exhibits increased external rotation and decreased internal rotation compared to the contralateral side [5]. While the total arc is often maintained, it is shifted by 10° into external rotation [42]. Both soft tissue and bone adaptations secondary to repetitive overhead throwing lead to such changes. With repetitive overhead throwing, the anterior capsule and glenohumeral ligaments become stretched, while the posterior capsule and glenohumeral ligaments are contracted [19]. In addition, during normal childhood development, humeral retroversion decreases from 78° in utero to 30° at skeletal maturity [18]. However, repetitive throwing prior to adulthood restricts the physiologic derotation process of the humeral head during growth [67].

The most prevalent injuries found in overhead-throwing athletes include posterosuperior labral tears and undersurface rotator cuff tears of the supraspinatus and infraspinatus tendon. In one series, 100 % of 36 competitive athletes with partial-thickness rotator cuff tears had concomitant posterosuperior labral tears [1]. While there is no debate regarding the most prevalent injuries found in this group of patients, there are various theories on the etiology of these findings. These include the posterior capsular contracture theory, the scapulothoracic dysfunction theory, and the internal impingement theory.

Burkhart and colleagues noted that the posterior capsule must withstand 750 N during the deceleration and follow through phases of throwing, with the main restraint to this force being the infraspinatus and posterior capsule, namely, the posterior band of the inferior glenohumeral ligament [7]. Over time, these structures undergo hypertrophic changes and stiffen, shifting the glenohumeral center of rotation to a more posterosuperior position [23]. This altered position subsequently leads to posterosuperior instability with the arm in abduction and external rotation [24], causing increased shear in the infraspinatus. Furthermore, a torsional force known as the peel-back phenomenon is applied to the biceps anchor during the late cocking phase of throwing, leading to the development of a superior labral anterior posterior (SLAP) lesion [9].

The scapula plays a very important role in transmitting energy from the trunk to the humerus during overhead throwing, and similar to other structures, it also undergoes adaptations to repetitive movements. The scapula on the throwing side demonstrates increased abduction, protraction, and inferior translation [48]. During late cocking, upward scapular rotation helps maintain glenohumeral joint symmetry [46]. However, weakness of the periscapular musculature may disrupt this normal relationship and alter the forces transmitted to the shoulder girdle, leading to scapular dyskinesia and the SICK (scapular malposition, inferior medial border prominence, coracoid pain, and dyskinesis of scapular movement) scapula [8]. With the scapula in a protracted and upwardly tilted position, the posterior glenoid lies in proximity to the humerus, predisposing the posterosuperior labrum and rotator cuff to impingement and injury. Furthermore, with a SICK scapula glenohumeral angulation is increased, which exacerbates the biceps peel-back effect and may predispose to SLAP lesions [34].

Although physiologic contact between the posterosuperior labrum and rotator cuff does exist in asymptomatic individuals [51], repetitive movements may lead to pathologic changes.

With the shoulder in 90° of abduction and 90° of external rotation, the supraspinatus and infraspinatus becomes entrapped between the greater tuberosity and glenoid labrum [62]. With forceful and repeated contact between the articular side of the rotator cuff and superior labrum, partial-thickness cuff tears and superior labral tears may arise [14]. With the onset of pathologic changes, a downward spiral may ensue, in which the athletes compensate for the symptoms felt while throwing with improper mechanics, which subsequently makes them prone to further injury.

16.2.2 Anterosuperior Impingement

As previously mentioned, anterosuperior impingement is the far less commonly encountered form of internal impingement. It involves entrapment of the subscapularis tendon between the anterior aspect of the humeral head and the anterior glenoid labrum leading to undersurface tearing of the subscapularis. Gerber and colleagues detailed the lesion arthroscopically and noted that it occurred with the shoulder in a flexed, adducted, and internally rotated position [20]. Lesions of the long head of the biceps and bicipital pulley have also been indicated in this disorder, as authors have shown that 50 % of cases of biceps subluxation were associated with degenerative changes of the anterosuperior labrum [22]. While often found in overhead athletes, it has also been described in paraplegic patients who use their upper extremities to drive the wheelchair.

The exact cause of undersurface tearing is unclear but is believed to be related to the relatively poor vascularization of the deep portion of the tendon, making it vulnerable to injury [2]. Subcoracoid impingement has been described as a cause of anterior shoulder pain and may also contribute to undersurface tearing. However, a relationship exists between the chronicity of full-thickness supraspinatus tears and the severity of subscapularis tendon abnormalities [4]. This may suggest that anterior instability may be a more important contributing factor to subscapularis tendon abnormalities than static subcoracoid impingement in the setting of a full-thickness supraspinatus tendon tear.

16.3 Clinical Presentation and Essential Physical Exam

A thorough history and physical examination is essential in the diagnosis of internal impingement. Most cases of posterosuperior impingement involve patients under the age of 30 who participate in sports that require repetitive overhead throwing. While the first major series described by Walch and colleagues involved volleyball and tennis players [60], it has most consistently been described in baseball pitchers [41]. However, non-athletes who perform repetitive overhead maneuvers with the shoulder abducted and externally rotated may also develop this condition.

Athletes with posterosuperior impingement often complain of shoulder pain during overhead throwing, namely, during the late cocking phase of throwing (Fig. 16.1). The shoulder pain is typically localized to the posterior aspect of the shoulder, but may also be more generalized. In fact, Burkhart and colleagues demonstrated an 80 % rate of anterior coracoid pain in their series of 96 athletes with a disabled throwing shoulder [8]. While patients may note a generalized dull aching pain at rest, it is with provocative maneuvers that they feel sharp stabbing pain as the supraspinatus impinges upon the glenoid labrum. They will often not be able to recall a particular inciting event, but instead note that their symptoms developed over time. In addition, they will often state that is particularly difficult for them to warm up or "get loose." Pitchers in particular will note a decrease in throwing velocity along with a loss of control and accuracy. They may also present with symptoms similar to rotator cuff disease, such as a "dead arm," shoulder weakness after throwing, a sense of "slipping" in the shoulder, frank subluxation, or apprehension with abduction and external rotation [10].

In contrast to posterosuperior impingement, patients with anterosuperior impingement are

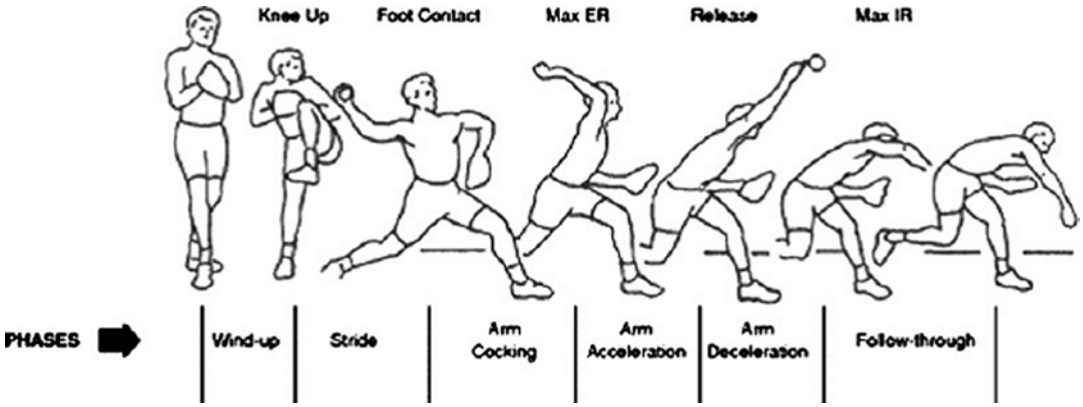

Fig. 16.1 Patients with internal impingement will complain of pain during the arm cocking and arm acceleration phases of throwing (Reprinted with permission from Digiovine et al. [15])

slightly older. In a series of sixteen patients with this entity, the average age was 45.3 years [20]. In that series, the majority of patients were not athletes, but rather engaged in manual professions that required regular overhead activity, such as masonry and carpentry. Symptoms are often worsened with overhead movements that involve flexion, adduction, and internal rotation of the shoulder. Patients will often note a gradual insidious onset of pain in the anterior aspect of the shoulder and may also note pain in the region of the bicipital groove if associated pathology of long head of the biceps tendon exists. Furthermore, popping, locking, and snapping may occur with unstable labral tears.

A stepwise and systematic physical examination should be performed to assess the lower back, hip, and knee. The clinician may assess functional movements with single leg squats for hip and trunk control, muscle imbalance, and inflexibilities. Examining the patient from the back, scapular winging and muscular atrophy should be noted. The glenohumeral joint line, acromioclavicular joint, the long head of the biceps, and the coracoid process should be evaluated for tenderness. Tenderness over the coracoid process is suggestive of pectoralis minor tendinitis, which can be correlated with scapular dyskinesis [8]. Other signs of scapular dyskinesis include a prominent inferior medial border of the scapula and the appearance of an inferiorly drooped throwing shoulder [35].

Overhead-throwing athletes often display asymmetry between the dominant and nondominant shoulder due to relative muscular hypertrophy of their dominant side. A thorough assessment of range of motion of both shoulders should be performed in both adduction and 90° of abduction. For many years, it has been the general belief that affected shoulder has 10–15° more external rotation at the expense of 10–15° of internal rotation, with the overall arc of motion being comparable to the contralateral side [47]. However, it has recently been demonstrated that patients with symptomatic internal impingement have a decrease in the total arc of motion as well. In a study on collegiate level baseball players, the dominant shoulders had a mean arc of 136.2° compared with 145.8° in the nondominant group, for a side-to-side difference of 9.6°.

While patients with internal impingement often have physiologic laxity, this must be distinguished from pathologic instability. Even without any dislocation or subluxation events, patients may have increased translation that alters their throwing mechanics. The amount of anterior and posterior translation should be noted and compared to the contralateral extremity. The posterior impingement test can also be used to assess for apprehension and pathologic instability. With the patient in the supine position, the shoulder is placed in 90° of abduction, 10° of forward flexion, and maximum external rotation. A positive test is constituted by the reproduction of pain in

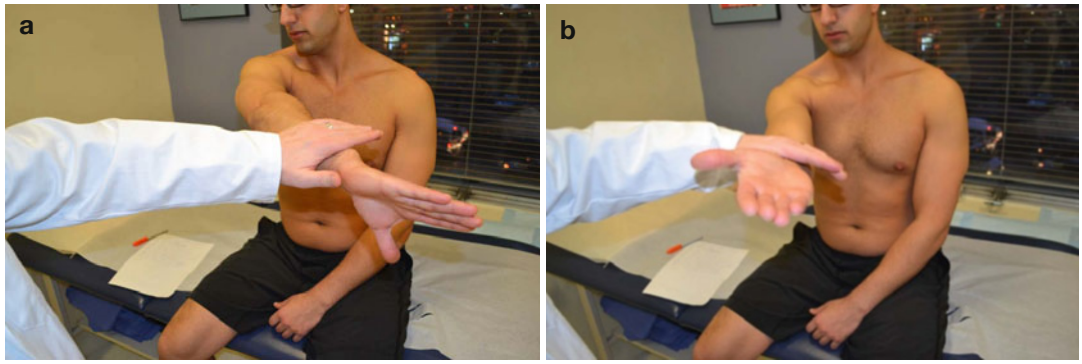

Fig. 16.2 (**a**, **b**) O'Brien's Test – the patient's arm is forward elevated to 90°, adducted 15° across the midline, and brought into maximum internal rotation. The patient is told to maintain this position while the examiner stands behind the patient and provides a downward force to the arm. Subsequently, with the palm fully supinated, the examiner once again provides downward pressure. The test is considered positive when then patient notes a significantly greater amount of pain with the arm in pronation and relief of pain with supination

the posterior aspect of the shoulder. The Jobe's relocation test is also a useful maneuver to test for internal impingement. The patient is once again placed supine; the arm is brought into 90° of abduction and 10° of forward flexion, and the shoulder is forced anteriorly. With pathologic laxity, patients will report pain with maneuver; however, the pain subsequently subsides with a posteriorly directed force [28].

Since internal impingement is often associated with SLAP tears, it must be examined for during the physical examination. To perform O'Brien's active compression test, the most sensitive and specific exam for SLAP lesions, the patient's arm is forward elevated to 90°, adducted 15° across the midline, and brought into maximum internal rotation so that the thumb is pointing downward. The patient is told to maintain this position while the examiner stands behind the patient and provides a downward force to the arm. The patient is asked to quantify the amount of pain felt with this maneuver. Subsequently, the patient's arm is brought into full external rotation while maintaining the other positions of the shoulder. With the palm fully supinated, the examiner once again provides downward pressure which the patient is told to resist (Fig. 16.2a, b). O'Brien's test is considered positive when then patient notes a significantly greater amount of pain with the arm internally rotated, along with a decrease or resolution of symptoms in full supination. For optimum results, the examiner should not be resisting the patient's attempt at forward elevating past 90°, but rather the patient should actively resist the clinician's downward force [49].

16.4 Essential Radiology

Radiographic evaluation begins with a standard shoulder series, including the anteroposterior, axillary, and outlet views to assess for overall alignment and geography. Specific for internal impingement, Bennett and colleagues described an exostosis of the posteroinferior glenoid rim secondary to repetitive triceps traction in baseball players and coined it the Bennett lesion [3]. The greater tuberosity must also be examined for sclerotic and cystic changes, as these are found in nearly half of pitchers with internal impingement [66]. Furthermore, rounding or remodeling of the posterior glenoid rim may also be noted.

Magnetic resonance imaging is the most utilized imaging modality to diagnose pathologic conditions of the shoulder. Of note, many asymptomatic patients may also have positive MRI findings; it is therefore imperative to compare radiographic findings to the physical examination. MRI has an advantage over even arthroscopy in that in can not only detect articular or bursal-sided rotator cuff tears but also diagnose

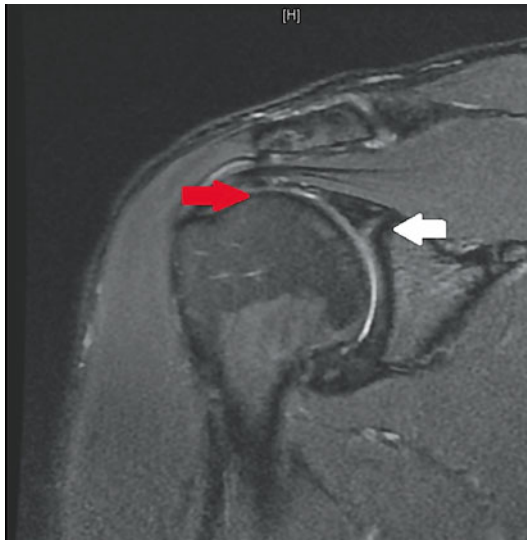

Fig. 16.3 Coronal oblique image of MRI arthrogram showing a superior labral tear (*white arrow*) with dye tracking into the space between glenoid and labrum and a partial-thickness rotator cuff tear (*red arrow*)

intrasubstance degeneration, which may be difficult to directly visualize. Articular-sided rotator cuff tears are often found in patients with internal impingement. In fact, up to 40 % of professional baseball pitchers have completely asymptomatic partial articular-sided supraspinatus tendon avulsion (PASTA) lesions [12]. Furthermore, MRI can also be enhanced with gadolinium contrast to increase the diagnostic value; it has sensitivity, specificity, and accuracy for diagnosing labral tears of approximately 90 % [16] (Fig. 16.3). Some experts have recommended performed magnetic resonance imaging with the shoulder in both the abducted and abducted and externally rotated position. A recent study showed that these sequences appear to improve the diagnostic accuracy of soft tissue anterior and posterior labral tears, SLAP tears, and significant bony glenoid lesions [45].

The most common constellation of MRI findings in patients with internal impingement includes undersurface tears of the supraspinatus or infraspinatus tendon and cystic changes in the posterior aspect of the humeral head associated with posterosuperior labral pathology [21]. Additional findings may include mature periosteal bone formation at the posterior aspect of the capsule (Bennett lesion) and posterior capsular contracture at the level of the posterior band of the inferior glenohumeral ligament complex [17]. There may also be remodeling of the glenoid with narrowing of the spinoglenoid notch from chronic pressure transmitted to the posterosuperior aspect of the glenoid from the shoulder being placed in repetitive abduction and external rotation.

16.5 Disease-Specific Clinical and Arthroscopic Pathology

Prior to a diagnostic arthroscopy, a thorough examination under anesthesia is performed to obtain a true assessment of the patient's range of motion and laxity. The clinician should record the patient's forward elevation, external rotation at neutral and 90° of abduction, and internal rotation. The shoulder should also be examined for the degree of anterior and posterior translation. Grade 1 represents mild translation, grade 2 is translation to the glenoid rim, grade 3 translation causes a dislocation that spontaneously reduces, and grade 4 is a fixed dislocation. Both range of motion and degree of translation should be compared to the contralateral extremity.

A comprehensive diagnostic arthroscopy is performed visualizing the entirety of the joint with a probe. Depending on the nature of the expected pathology, the patient is placed into the beach chair position for the rotator cuff or the lateral decubitus position for labral work and capsular laxity. Two portals are typically used, the posterior and rotator interval portal, with further portals dependent on the visualized pathology. Arthroscopic evaluation begins with examination of the glenoid and humeral head for chondral wear. The superior glenoid is then evaluated for SLAP lesions; viewing from both anterior and posterior is mandatory to fully appreciate these lesions. The glenoid articular cartilage generally extends medially over the superior corner of the glenoid; absence of cartilage in this area indicates labral detachment. The arthroscopic peel back was described by Burkhart and colleagues. With the arm placed in the throwing position, in the presence of a tear, the labrum will peel away from the glenoid [6]. The biceps

tendon is next evaluated from its insertion on the supraglenoid tubercle distally toward the intertubercular groove. A probe is used to pull the tendon into the joint which allows for circumferential examination for tendinosis or synovitis, as well as to test for the stability of the biceps anchor.

Following inspection of the biceps, the superior and anterior labrum is evaluated from 12 to 6 o'clock, assessing for the integrity of the anterior glenohumeral ligaments as well as the degree of capsular laxity or tears. The subscapularis should also be examined for tearing as anterosuperior impingement can cause undersurface tears. Next, examination of the axillary pouch is performed, assessing the volume of the pouch and viewing for synovitis or hemosiderin deposits. From the axillary pouch, visualization proceeds posteriorly where the posterior labrum can be assessed. However, visualization of this area can be enhanced by placing the arthroscope in the anterior portal.

The rotator cuff should then be inspected, with particular attention given to the undersurface of the cuff at the junction between the supraspinatus and infraspinatus. Viewing from posterior, the arm may be brought into 90° of abduction and full external rotation to assess for abnormal impingement between the rotator cuff and posterosuperior labrum. Lastly, the subacromial space should be examined for bursitis and evidence of external impingement, such as fraying or ossification of the coracoacromial ligament.

16.6 Management of Internal Impingement

Following a physical examination and appropriate imaging of the affected shoulder, the clinician can make recommendations for treatment. Both nonoperative and operative treatment may be recommended by the treating provider based on several principles. The clinical evaluation must correspond with imaging to make the diagnosis of internal impingement. The diagnosis of internal impingement without supporting physical exam findings or radiographic confirmation is unlikely to have fruitful recovery following an undirected course of therapy or arthroscopy. The level of function of the athlete is an important consideration when making a choice as well. The time frame of return to sport can inform the provider to make certain recommendations as well as the length of time necessary for expected recovery from both nonoperative management and in the postoperative course. Lastly, other pathological states including rotator cuff tears, SLAP tears, or labral tears should be evaluated and treatment of these conditions should be considered as corresponding diagnoses with different treatment options and recovery time frames.

16.6.1 Nonoperative Treatment

Nonoperative treatment should always be the first line of treatment for patients with internal impingement of the shoulder. Athletes with internal impingement should be notified that the majority of patients improve with cessation of throwing activities and focused therapy modalities. Patients with new-onset impingement symptoms should be initially treated with throwing modification, initiation of routine NSAID use, and enrollment into a formal throwing therapy program. Attenuation of throwing activity can be an initial period of 2–3 weeks to allow for the initial phase of inflammation to subside while using NSAIDs and undergoing a focused regimen of thrower-specific therapy.

A focused throwing regimen should contain three key components: application of kinetic chain exercises, shoulder mobility, and shoulder strengthening [33]. The authors recommend a complete therapy regimen that focused on these three principles in an effort to maximize treatment outcomes.

Kinetic chain exercises are initiated with the primary reason of making the throwing motion a more dynamic and mechanically sound action. These exercises focus on proximal core strengthening, hip mobility and strengthening, and leg drive and strength. It is known that about 50 % of throwing velocity comes from step and rotation of the trunk [57]. It is important to have flexibility and a fluid kinetic chain of motion allowing for transfer of energy from the lower extremity and

core muscles to the throwing arm. In an effort to encourage muscular training and control during the kinetic chain, therapy should focus on core and lower extremity flexibility, balance, and strength.

Keys behind shoulder mobility include scapular stabilization, restoring normal external and internal rotation deficits, and addressing any mechanical or dynamic restraints within the spine and shoulder. Muscle balance and symmetry is the goal behind this second key principle of rehabilitation in the throwing shoulder [33]. However, before symmetrical and mechanically sound kinematics may be restored, any pathological motion must be minimized. This is done through selective stretching. Selective stretching has not only been found to alleviate impingement as well as rotation deficits, it also has been noted to be protective from future injury [54, 63].

Contractures of posterior capsule, posterior rotator cuff, pectoralis minor, and the short head of the biceps can lead to glenohumeral internal rotation deficit. Several different exercises have focused on improving compliance of posteroinferior contracture through selective stretching. The cross-body stretch was described by McClure et al. as an exercise that was shown to increase internal rotation of the affected side when compared to the contralateral side [40]. An additional exercise is the sleeper stretch which has further been shown to be effective in stretching the posterior contractures as well as pectoralis minor which has additionally been implicated in pathological scapular motion [36]. The pectoralis minor may also be stretched by placing a rolled towel between the athlete's shoulder blades while supine and applying a posterior directed force on the shoulder. Two additional stretches include the internal rotation stretch and the horizontal adduction stretch. The internal rotation stretch involves the arm being placed in the throwing (cocked) position. The arm is then internally rotated to stretch the posterior rotator cuff. The horizontal adduction stretch may be done with the arm horizontally adducted while the scapula is stabilized.

All of these stretches have a goal of minimizing internal rotation deficit to within 18° (range 13–20°) compared to the contralateral side [64]. In an article by Tyler et al., the authors noted the clinical outcomes of a stretching program on symptomatic throwers [58]. In that article, the authors found that there was a greater improvement in posterior shoulder tightness in those patients with complete resolution of symptoms compared to patients with residual symptoms (35° vs 18°). The authors further noted that improvements in glenohumeral internal rotation deficit and external rotation loss were not different between patients with and without residual symptoms of pain.

The last tenet of nonoperative treatment of internal impingement of the shoulder is shoulder-specific strengthening through restoration of scapular motor control, initiation of a scapular feedback program, and promotion of eccentric control of the shoulder and elbow through an increased number of repetition throwing cycles. Several authors have pointed out that function in the throwing shoulder is heavily dependent on scapular muscle strength, endurance, and also neuromuscular control [31]. Specific exercises focusing on these principles often beginning with closed chain exercises progressing to open chain scapular muscle training.

As an impingement that occurs later in the throwing shoulder pathological process, anterior impingement remains a described yet mostly unknown entity. It is understood that anterosuperior impingement is likely due to chronic micro-instability of the anterior capsulolabral structures with injury to the deep portion of the capsule and subscapularis tendon. This impingement is thought to occur during the follow-through phase of throwing. Although there is not a specific regimen for therapy for anterosuperior impingement, the same neuromuscular control and muscle recruitment for a mechanically balanced shoulder may be assumed to be of benefit. Furthermore, emphasis on proper throwing mechanics with kinetic chain exercises can teach proper muscular control from the wind up to the follow-through of the pitch.

After initiation of the initial phases of therapy, more advanced phases are undertaken with a goal of increasing power and endurance and a gradual reintroduction of throwing activities. The various

portions of therapy have been described in phases with phase 1, the acute phase, focusing on scapular and glenohumeral control and activation [33]. Phase 2 is focused on core exercises, the kinetic chain, and isotonic strengthening. This is the recovery phase. The last phase, the functional phase, involves reintroduction of throwing with emphasis on control, velocity, and endurance. This last portion of therapy may prove to be the most problematic as the reintroduction of throwing activities is very dependent on patient response to pain. Such an interval throwing program requires that the throwing athlete remain asymptomatic before advancement of the throwing to normal velocity and number of throws performed. The expectation is that the patient be able to return to full throwing velocity over 3 months time. If the patient fails to meet this criterion in a 3–6-month range and cannot perform at a level necessary for competition, then the patient is considered for surgical intervention.

There is a certain subset of patients that can be appropriately treated with nonoperative management after the diagnosis of internal impingement is made. It is the responsibility of the provider to be able to recognize those patients who would make the most significant gains after a directed course of physical training and therapy. Several features elicited in the physical examination have been found to predict success with nonoperative management [32]. Those factors include pain with resisted abduction, pain with forward flexion, and the positioning of the scapula prior to therapy. These authors further found that sustained rotator cuff strength at 90° of abduction was predictive of success in those patients not having surgery.

16.6.2 Operative Treatment

Operative treatment of internal impingement is focused on sites of pathology as identified by physical examination and advanced imaging. Because internal impingement represents a spectrum of injury, the orthopedic provider must be willing to address multiple sites of injury including the anterior and posterior labrum, the superior labrum, the biceps tendon, the capsule, and the rotator cuff specifically the supraspinatus and anterior portion of the infraspinatus. Jobe has offered that there are five main anatomical sites of pathology: the posterosuperior labrum, the articular portion of the rotator cuff, the greater tuberosity, the inferior glenohumeral ligament, and the posterosuperior glenoid [29]. Treatment of these sites of injury is focused on restoring the kinematics and anatomy necessary for a functioning high-level throwing athlete. Although surgical repair cannot make the anatomy "normal," it can remove sites of physical aberration and provide a more structurally sound labral and rotator cuff complex.

16.6.2.1 Operative Treatment of Labral Pathology Associated with Internal Impingement

The labrum serves many purposes in the shoulder and does more than simply provide a mechanical bumper for stability. This fact has been noted in that with labral resection, the amount of glenohumeral translation increases only approximately 10–20 % [37]. It has been noted to enhance the concavity/compression mechanics of the glenohumeral joint; serve as a site for attachment for the biceps, glenohumeral ligaments, and capsule; and lastly serve as a proprioceptive feedback sensor [50, 59]. Surgical intervention should be focused on restoring these key characteristics of the labrum by enhancing stability of the labral tissue without restricting the compliance of the soft tissue.

Surgical intervention of the labrum begins with an arthroscopic evaluation and mechanically testing the labrum under direct visualization. The surgeon will need to view the labrum attachment to the glenoid and will need to pay specific attention to the superior labral attachment in the SLAP region. Diagnosis and treatment are based primarily on arthroscopic evaluation of the SLAP region [44]. There is some difficulty in diagnosing a true type II SLAP tear as a separate entity from one of the many normal anatomic variant in the patient undergoing surgery. Normal variants in the superior and anterior labrum include sublabral foramen, a

Buford complex with a cord-like MGHL, and discoid variant superior labral tissue. There is great variation in the anterior superior labrum and the presence of a cord-like middle glenohumeral ligament is more often seen in isolation than combined with a deficient anterior labrum [26]. The quality of the labral tissue, amount of inflammation around the labrum, and the amount of exposed glenoid can be assessed and used to direct a surgical decision. Performing dynamic testing to recreate the "peel-back" mechanism can further demonstrate loss of labral integrity [6]. The "peel-back" maneuver is done by placing the arm in a throwing position with external rotation and abduction of the shoulder. The labrum is seen to "peel back" from the posterosuperior glenoid in a positive test. The arthroscopic evaluation will also need to focus on the amount of chondral injury more specifically examining cartilage damage on the superior rim of the glenoid as a sign for an acute SLAP injury [53]. Further chrondral damage may be assessed in the case of instability and early degenerative changes. The glenohumeral ligaments and capsular attachments will also need to be examined by viewing the capacious nature of the joint as demonstrated through a "drive-through sign." Insufficiency of the glenohumeral ligaments can also be noted through dynamic abduction and rotation testing under visualization. The posterior labrum and posterior capsular tissue will also need to be noted for any adhesions or decreased compliance as noted in the GIRD phenomenon [9].

16.6.2.2 Operative Treatment of Rotator Cuff Injury Associated with Internal Impingement

Rotator cuff injuries are included in the pathological process of internal impingement. The rotator cuff tears that are found are almost always partial undersurface tears that are approximately 2–5 mm from the actual insertion site [32] (Fig. 16.4). This undersurface tearing may in fact be a normal result of the throwing motion in high-level athletes which has been noted to be present in 40 % of asymptomatic pitchers [12]. In symptomatic patients, a partial-thickness tear of the articular side of the rotator cuff was noted in 80 % of professional overhead athletes with a diagnosis of internal impingement [51]. The proposed mechanism for these partial-sided rotator cuff tears is significant horizontal abduction with increased contact stresses at the posterior cuff/labral interface that is a result of compensatory external rotation in throwers. The typical appearance of these impingement-type rotator cuff tears is fraying in the infraspinatus that does not extend into the medial tendon. If there is delamination that extends medially into the supraspinatus or the infraspinatus tendon, several authors have recommended transtendinous repair [25]. Furthermore, many authors agree that if there is greater than 50 % involvement of the insertion, results tend to be less reliable with just a simple debridement of partial articular-sided tear. This has led several authors to propose stabilization of impingement-type tears if they are greater than 50 % of the width of the insertion [65]. Conway et al. examined 14 baseball pitchers with an average age of 16 who were treated with a repair of intratendinous rotator cuff tears and concurrent pathology including labral tears and SLAP tears [13]. They found that 89 % percent of patients undergoing intratendinous repair were able to return at the same or higher level at 16-month follow-up. An additional study by Ide

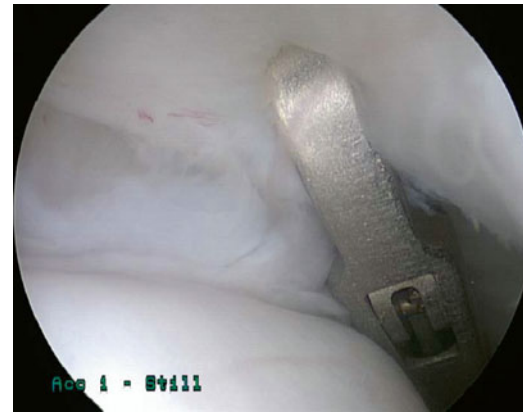

Fig. 16.4 Partial-thickness articular-sided tear pattern often seen with internal impingement. The partial articular-sided tears found within the spectrum of internal impingement are often posterior supraspinatus and infraspinatus tears

et al. reported the results of an arthroscopic transtendon repair in patients with >6 mm partial-thickness articular-sided tears of the supraspinatus tendon [27]. The authors found good to excellent results in 16/17 patients. However, of six overhead-throwing athletes, only two were able to return to their previous sport at the same level. Many techniques for repair have been recommended including transtendinous reattachment of the tendon to bone using bone tunnels as well as anchors [43]. Although there is no evidence that one fixation technique is more mechanically sound than the others, the surgeon will need to use proper rotator cuff repair techniques to ensure firm fixation. Some authors also recommend completion of the partial-thickness tear in an effort to perform a more formal repair technique. [39] The authors recommend completion of the tear only if there is a greater than 75 % involvement of the insertion site.

The subscapularis tendon will also need to be evaluated in cases of anterosuperior impingement. Any fraying should be debrided and if there is a loss of integrity of the subscapularis tendon, the tendon should be repaired.

16.6.2.3 Operative Treatment of Posterior Capsular Contracture

Surgical treatment of patients with GIRD is strictly limited to those patients who have failed to improve with an aggressive internal rotation stretching program. After confirmation of internal rotation deficit, the posteroinferior capsule is examined for tightness intraoperatively. The results of selective posterior release have shown good results for patients who have failed nonsurgical treatment in the setting of GIRD. In one study, Morgan et al. found that there was an average of 62° (55–68°) increase in internal rotation a capsulotomy was performed [8]. In an additional study, Yoneda and colleagues performed posterior capsular releases on 16 patients [68]. They reported that 11 of the 16 patients, including all 4 who had no other concomitant lesions, returned to their preinjury level of competition.

Several studies have highlighted the importance of avoiding a concomitant subacromial decompression in the setting of internal impingement [56]. In this study, Tibone reported that after acromioplasty in the setting of internal impingement, only 22 % of overhead athletes returned to presurgical level of competition. This has led some authors to recommend against concomitant subacromial decompression with internal impingement especially in the setting of a transtendon repair or significant labral fixation.

16.7 Authors' Preferred Treatment

After appropriate history and physical of the patient with confirmatory imaging, the authors recommend the use of regional anesthesia with conscious sedation. The positioning of the patient is extremely important. The patient is placed in lateral decubitus position if there is concomitant instability requiring either a labral repair or capsular plication. A beanbag is used with all bony prominences padded. With adequate abduction, forward flexion, and traction applied to the affected extremity through use of the spider, a small bump is placed in the axilla to enhance glenohumeral distraction. Additionally, the patient is placed in 20° of reverse Trendelenburg with the torso tilted slightly posteriorly. A beach chair position is used if the patient has a concomitant SLAP tear or rotator cuff tear. A posterior viewing portal is made inferior and at the lateral edge of the posterior acromion. The arthroscope is introduced and diagnostic arthroscopy is performed. A "drive-through" sign may be assessed at this point. The anterior portal is made under direct visualization often using a spinal needle through the rotator interval placed at the correct trajectory so that instruments may be introduced parallel or superior to the level of the superior glenoid if a SLAP repair is expected. The portals are placed based on expected areas of fixation with more laterally based portals being done if anterior or posterior labral work is required allowing for more tangential positioning of instrumentation. After establishing the anterior portal, a probe is introduced to access peel back and the labral tissue

quality in addition to the biceps anchor and the rotator cuff. At this point, glenohumeral ligament tensioning and capsular tissue quality can also be examined.

With symptomatic unstable type II or greater SLAP tears, the author prefers surgical fixation utilizing suture anchor fixation. After debriding the labrum and creating a bony bed for the superior labrum, the anchor is introduced through a percutaneous approach without cannula placement through the accessory portal of Wilmington. We prefer to place these anchors percutaneously as larger portals with cannula placement through the portal of Wilmington have been associated with postsurgical pain and rotator cuff dysfunction [11, 55]. The author typically will use 2.0 suture anchors and a suture lasso to pass the sutures. The author prefers to place smaller single-loaded suture anchors for multiple points of fixation as opposed to a larger 2.4 double-loaded suture anchor. After passing the suture around the labrum using a lasso, the sutures are tied with the more medial strand acting as the post for tying. The suture is cut using an open suture cutter in an effort to minimize intra-articular suture burden. After placement of the labral sutures, the peel-back phenomenon again is assessed. We prefer to minimize anchor placement on the anterior superior labrum just inferior to the biceps in an effort of minimizing limitation of normal biceps motion.

Following assessment and fixation of the SLAP region, the rotator cuff is then examined for amount of involvement (Fig. 16.5a–c). If there is a rotator cuff tear that is noted to be more than 50 % of the width of the tendon, surgical fixation is again planned and executed. Otherwise, debridement of the partial articular-sided tear is performed. If there is greater than 75 % involvement, then the remaining intact tendon is debrided to convert to full thickness for repair. Debridement of the exposed footprint is performed to assess the amount of involvement. Knowing the size of the shaver (often a 3.5 or 4.5 shaver) can be beneficial in using the width of the shaver to measure the involvement of the tear. Prior to placement of anchors and passing suture for tendon repair, a subacromial bursectomy is performed to allow for ease of suture passage and visualization. The arthroscope is again introduced into the glenohumeral joint in the case of a retained portion of intact tendon and a spinal needle is used to position the anchor in the desired trajectory and a percutaneous incision is made. The drill guide pierces the tendon and is placed with the proper angle on the greater tuberosity. Care should be taken to allow for a small amount of bone between the anchor placement and the edge of the articular margin. Only the anterior supraspinatus attaches next to the articular surface with the posterior supraspinatus and infraspinatus attach several millimeters from the margin allowing for a normal anatomic "bare area." If the surgeon inadvertently does not recreate the bare area in the posterior portion, there will be resulting limitation of range of motion and functional deficit in the overhead athlete. For this reason, we prefer to avoid anchor placement in overhead athletes unless absolutely necessary. Intralaminar tears may be repaired using a similar shuttling technique without anchor placement. Once the anchor is placed, suture passing is then performed. Any number of devices may be used to pass the suture, but we prefer a shuttling technique using an 18 gauge spinal needle. A needle meniscal repair device with a wire shuttle may also be used to pass the suture. This is done in an effort to minimize trauma to the intact rotator cuff. One of the suture limbs is retrieved and a spinal needle is then used to penetrate the intact bursal cuff. A monofilament suture is placed through the needle and used to shuttle the anchor suture. After passage of sutures, the camera is reintroduced in the subacromial space and the sutures are tied. Lastly, the glenohumeral joint is revisualized once more to inspect the integrity of the repair.

After addressing the rotator cuff, we then examine the posterior capsular tissue and the posterior cuff for thickening and lack of compliance. In those patients who have failed to improve with internal stretching exercises, a selective posterior inferior capsular release may be performed. An arthroscopic posteroinferior quadrant capsulotomy is performed from 6 to 9 o'clock position on the right shoulder and 3 to 6 o'clock on the left

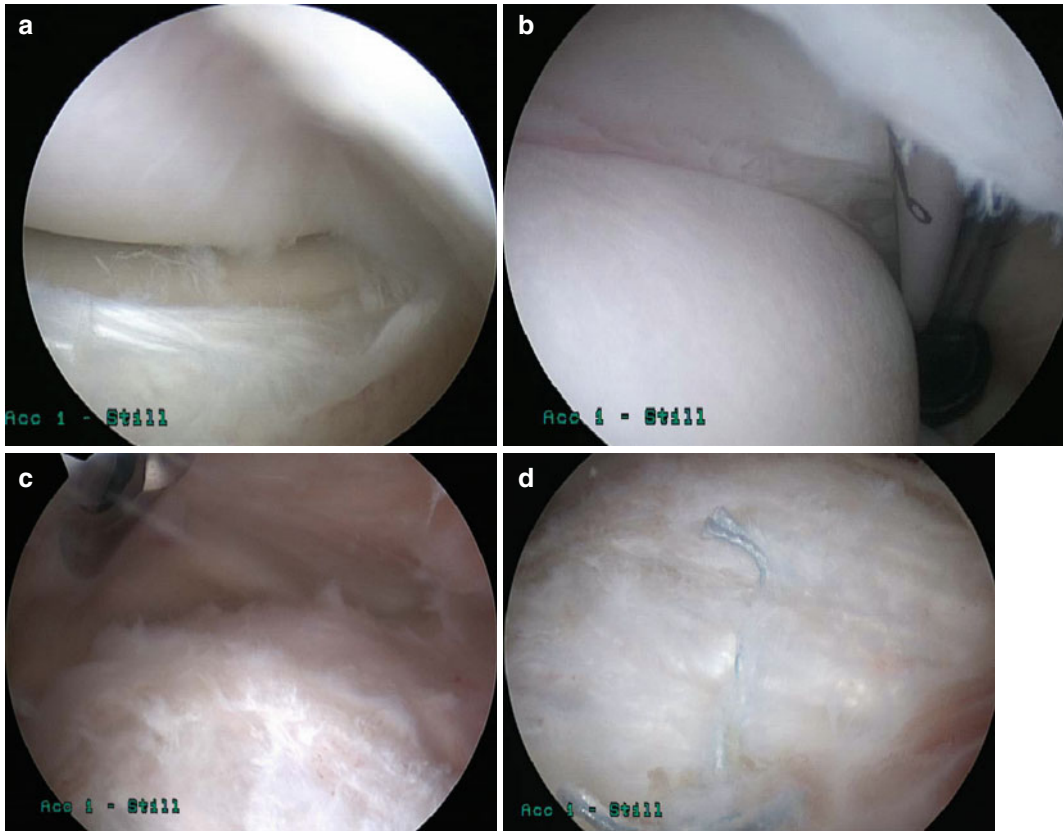

Fig. 16.5 Intraoperative assessment of internal impingement in the lateral position. (**a**) The partial articular-sided posterior rotator cuff tear may be appreciated on the superior portion of the image and the posterosuperior labrum is noted with some degeneration in the inferior portion. (**b**) Debridement of partial-thickness articular-sided rotator cuff tear with intralaminar repair. The footprint is debrided using a shaver to encourage healing. (**c**) The camera is then introduced into the subacromial space to perform a bursectomy before being reintroduced into the glenohumeral space (**b**). The anterior and posterior portions of the intralaminar tear are identified and an 18 gauge needle is passed percutaneously through each limb. A PDS suture is passed through each needle to act as a shuttle for the suture limb. Each PDS is retrieved through an anterior portal and is tied to each end of a single #0 FiberWire (Arthrex, Naples, FL). Each shuttle is pulled and the camera is again placed into the subacromial space. Each limb of the FiberWire is retrieved through a lateral cannula and tied. (**d**) The camera is again introduced into the subacromial space to tie the suture limbs. A subacromial view of the repair is noted in (**c**)

shoulder. Visualization of the muscular portion of the posterior cuff should confirm appropriate capsular release.

16.8 Postoperative Rehabilitation in the Throwing Athlete with Internal Impingement

The goals of rehabilitation are to protect the surgical repair and allow the tissues to heal while preventing any significant stiffness. There is a delicate balance between allowing the acute inflammatory phases of healing to resolve by limiting use and encouraging early motion in an effort to minimize stiffness. The postoperative course may change dependent on the degree of fixation. The postoperative rehabilitation protocol after both transtendon and full-thickness rotator cuff repairs is very similar. After completion of surgery, the arm is placed in a sling with a small abduction pillow. The sling is worn continuously for 6 weeks with elbow flexion and extension being encouraged immediately after

surgery. Patients begin passive external rotation exercises immediately after surgery. We prefer avoiding overhead stretching until 6 weeks after surgery. Rehabilitation of the rotator cuff, deltoid, and scapular stabilizers begins approximately 8–10 weeks after surgery with focus on isotonic strengthening. The patient is started in a throwing protocol as discussed above typically within the 3–4-month range with progressive activities incorporated as strength allows. Typically, athletes can return to competitive throwing between 6 and 12 months of postoperative therapy.

16.9 Pearls of Treatment, Pitfalls, and Complications

The treatment of internal impingement in the throwing athlete can be a challenge for the orthopedic provider. The challenge exists for several reasons. First, the pain associated with internal impingement is poorly understood. Although there are several sites of pathology in the clinical scenario of internal impingement, it is not clearly understood how some aberrations can cause pain and others do not. Pain can be localized to a certain area of the shoulder or can be more diffuse. This factor has led some authors to recommend certain treatments based on pain patterns [17]. Second, the diagnosis of internal impingement is not always understood by the provider assuming care of the patient. The provider must understand that impingement is in fact a spectrum of pathology with multiple sites of injury that need to be addressed. By completely assessing all of the sites of injury in internal impingement, the treating provider can make more appropriate recommendations for treatment and ultimately can offer a more accurate treatment time and return to competition. A third factor that makes treatment of internal impingement difficult is the level of function of the athlete being treated. High-level throwing athletes have high demands with extremes in physiological range of motion, velocity, and repetitive stresses that must be maintained for the athlete to excel in a competitive setting. Small changes in range of motion and velocity, for instance, can have a profound impact on the level of expectation for an overhead athlete when compared to a laborer or noncompetitive athlete.

One of the biggest obstacles to achieving an excellent outcome in treating internal impingement is the balance of stability and stiffness. Because the injury pattern is multifactorial, both nonoperative and operative treatments are focused on maintaining a large range of motion that is required for overhead performance. Nonoperative treatment is most often able to achieve resolution of pain with continuation of high-level throwing through use of a selective throwing and stretching program [63]. If there is lack of improvement with nonoperative treatment, there must be certainty with clinical diagnosis and proficiency with surgical skills in the treatment of internal impingement on behalf of the treating provider. The provider must discuss with the patient that although surgical treatment can provide improvement, there can be residual symptoms including continued pain and loss of range of motion. The surgeon must fully understand principles of surgical treatment of impingement in an effort of minimizing postoperative stiffness and pain. Key principles include proper positioning of anchors, appropriate repair of the rotator cuff and labral tissue, minimizing biceps tethering, selectively releasing the posteroinferior capsule, and avoiding a concomitant aggressive subacromial decompression. The patient's willingness to accept a small risk of postoperative stiffness often informs the overall result of both nonsurgical and surgical treatment. Another key principle that encourages a positive outcome is to have a group approach to treating the throwing athlete. Physiatrists, therapists, administrative assistants, and athletic trainers should all be in constant discussion regarding the plan and progression of the athlete with treatment. It is useful to have a well-documented protocol that all team members can use as a common reference point. The athlete should also be given the protocol so that expectations of advancement of therapy can be addressed.

Conclusion

The treatment of the overhead athlete is a complex endeavor. The diagnosis of internal impingement must be made based on clinical and radiographic findings by an informed orthopedic provider. Internal impingement represents a spectrum or cascade of aberration that can lead to multiple pain generators that can ultimately lead to dysfunction of the throwing shoulder. Treatment is focused on maintaining a high level of range of motion and strength while promoting resolution of pain. Most symptoms will resolve with use of a designated throwing shoulder program focusing on kinetic chain exercises, shoulder mobility, and shoulder strengthening. When nonoperative measures fail, surgical treatment attempts to restore functional anatomy.

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Traumatic Anterior Shoulder Instability: Part I. General Concepts and Proper Management

17

Alexandre Lädermann, Samy Benchouk, and Patrick J. Denard

17.1 Introduction

The glenohumeral joint has six degrees of freedom with minimal bony constraint that provides a large functional range of motion. It thus renders this diarthrodial joint particularly vulnerable to instability. The glenohumeral joint is the most commonly dislocated large joint of the body, affecting approximately 1.7 % of the general population [1]. In greater than 90 % of cases, the instability is anterior, has a traumatic origin, and occurs in young athletes involved in contact sports [2, 3]. Ongoing sports participation in this population is associated with a high recurrence rate [4]. The risk for progressive irreversible intra-articular injuries with recurrent instability episodes may negatively affect subsequent surgical success rates and thus provides a rationale for early surgical stabilization in this population. Effectively, recurrent instability can lead to increasing bone loss [5], cartilage lesions [6], and more soft tissue damage (Fig. 17.1). Subsequently, these may result in chronic pain, functional impairment, prolonged time away from work and sports [7], a negative impact on quality of life [8], or eventual early-onset arthropathy [9]. The management of anterior shoulder dislocation has thus evolved with time and depends on variables such as patient age, sports participation, and physical and radiological characteristics. The goal, whether nonoperative treatment or surgical stabilization is selected, is to achieve a stable, functional shoulder, with full painless range of motion.

Arthroscopic shoulder techniques have conferred significant advantages on open techniques. Visualization is improved, allowing easier identification and treatment of associated intra-articular pathology, such as humeral avulsion of the glenohumeral ligament (HAGL) or superior labrum anteroposterior (SLAP) lesions. Moreover, an arthroscopic approach reduces surgical trauma, particularly the need to detach subscapularis tendon. With these advantages, arthroscopic stabilization has become a viable and frequently used procedure to address anterior instability [10–13].

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Chapters 1, 2, and 3 of this book have addressed shoulder anatomy, biomechanics, epidemiology, and physical examination, respectively. This chapter will therefore focus on the pathology, classification, and natural history and treatment of primary traumatic anterior shoulder instability. We subsequently present our current therapeutic techniques and provide a stepwise approach to this condition that is frequently encountered by the orthopedic surgeon.

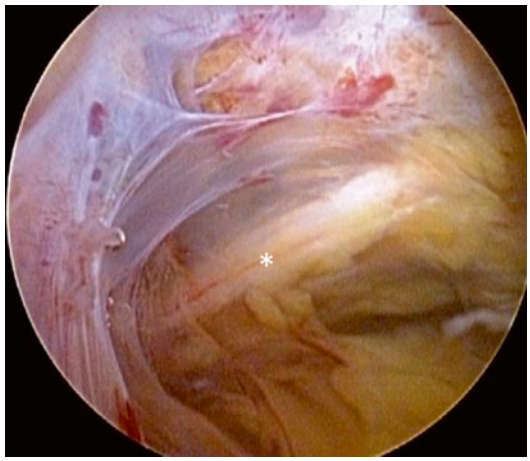

Fig. 17.1 Arthroscopic view of a left shoulder through a posterior portal. This patient has sustained more than 50 subluxations. The axillary nerve is clearly identifiable (*white asterisk*). There is no more capsule or inferior glenohumeral ligament, and the subscapularis muscle is hardly recognizable

17.2 Pathoanatomy, Biomechanics, and Preferred Classification

Traumatic anterior shoulder instability in the athlete usually occurs with an anteriorly directed force applied to an abducted and externally rotated arm, or from a direct blow. During a traumatic anterior dislocation, many of the passive and active stabilizers may be damaged. The glenoid labrum, the glenohumeral ligaments, and the glenohumeral joint capsule representing the soft tissue passive stabilizers will be injured; an avulsion of the anterior labrum, the classic Bankart lesion (Fig. 17.2) or its variations (glenolabral articular disruption (GLAD), Perthes, anterior labroligamentous periosteal sleeve avulsion (ALPSA)), is almost invariably present [14–16] although it does not produce instability in isolation [17]. The anteroinferior glenohumeral ligaments and the capsule can be detached from the glenoid rim, and a plastic deformation of the glenohumeral ligaments and an HAGL lesion [18] (Fig. 17.3) are other common features. The plastic deformation of these structures becomes progressively more severe with subsequent episodes [19–21]. The middle glenohumeral ligament functions to limit both anterior and posterior translation of the arm at 45° of abduction and 45° of external rotation, whereas the inferior glenohumeral ligament resists translation of the arm in greater degrees of abduction [22].

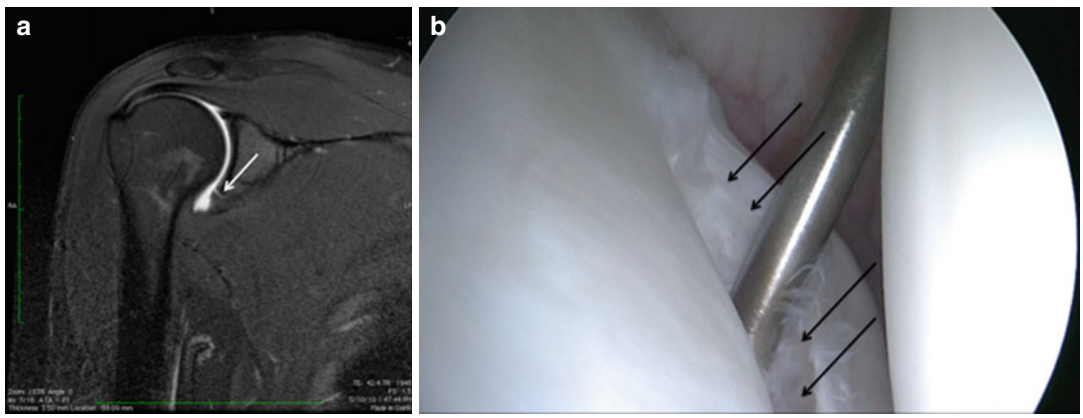

Fig. 17.2 (**a**) Coronal MRI of a right shoulder showing disruption of the anteroinferior glenoid labrum (*white arrow*). (**b**) Arthroscopic image confirming discontinuity of the glenoid surface and redundant anterior labrum (*black arrows*)

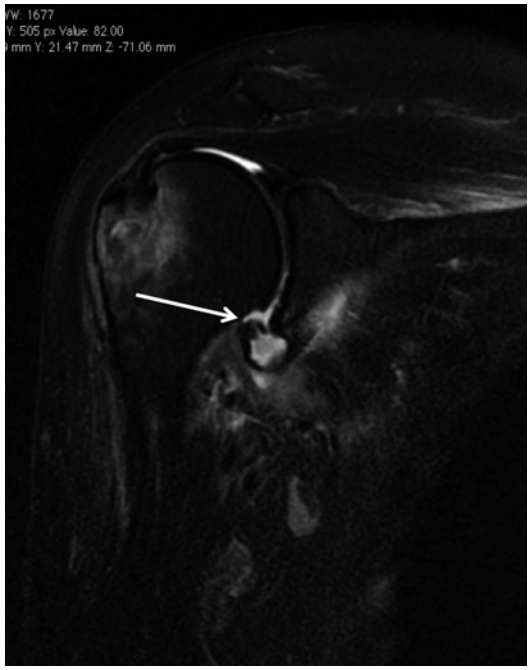

Fig. 17.3 Coronal T2 MRI of a right shoulder. The *white arrow* highlights an HAGL lesion. This lesion must be recognized as an isolated retensioning of the inferior glenohumeral ligament will lead to recurrent instability

In addition to progressive soft tissue injury, recurrent dislocations can facilitate bony injury [23, 24]. Bony lesions are frequent in recurrent cases and may include defects of the glenoid (bony Bankart or bevelling of the anterior glenoid resulting in loss of glenoid concavity), impaction of the posterolateral humeral head (Hill-Sachs lesion), or even coracoid or proximal humerus fractures (Fig. 17.4) [5]. Given that the average glenoid diameter is about 24 mm, a 6-mm-wide or larger fragment of the glenoid will typically equate to a 25 % or more of the articular surface and is considered a large bony fragment [25, 26]. Such significant glenoid bone loss can be viewed arthroscopically as an inverted-pear configuration. All Hill-Sachs lesions are by definition engaging lesions (since it has engaged at least once). Thus the notion of "engaging" versus "non-engaging" can lead to significant confusion. Some have proposed that the important lesions are those that engage in the 90-90 position. A more recent proposal by DiGiacomo et al. is to describe the lesions as "on-track" or "off-track" to determine the need for remplissage in addition to arthroscopic Bankart repair in the setting of glenoid bone loss of less than 25 % [27]. This view is discussed in more detail in subsequent sections.

Finally, the active restraint, mainly a lesion of the rotator cuff above the age of 40, will complete this complex situation [28, 29]. Since rotator cuff tears associated with instability usually occur in the nonathletic population, this situation is not discussed in detail in this chapter.

The degree, nature, and combination of the injury in athletes are highly variable. It would appear, therefore, that reducing the incidence of recurrent instability from a pathoanatomical viewpoint would be desirable. All the damage to the osseous and soft tissue stabilizers of the shoulder, as well as vascular and neurological impairment, must be detected and analyzed in order to offer to the patient the best treatment option.

Instability can be classified as primary or recurrent. The latter can be further classified as dislocation, subluxation, apprehension, or an unstable painful shoulder. In frank *dislocation*, the articular surfaces of the joint are completely separated. *Subluxation* is defined as symptomatic translation of the humeral head on the glenoid without complete separation of the articular surfaces. *Apprehension* is classically defined by fear of imminent dislocation in the 90-90 position. This could correspond to an instability phenomena or a persistent fear after a successful glenohumeral stabilization [30]. The *unstable painful shoulder* presents as pain only (as opposed to a sense of instability) during an apprehension maneuver at clinical examination [31, 32]. The majority of these patients have a history of trauma, but simply do not report a clear history of trauma. Careful preoperative and/or arthroscopic examination will show that the majority of these patients also have evidence of instability (i.e., labral tear, glenoid fracture, or Hill-Sachs lesion).

Five types of traumatic anterior dislocation have been described. The subcoracoid dislocation has an

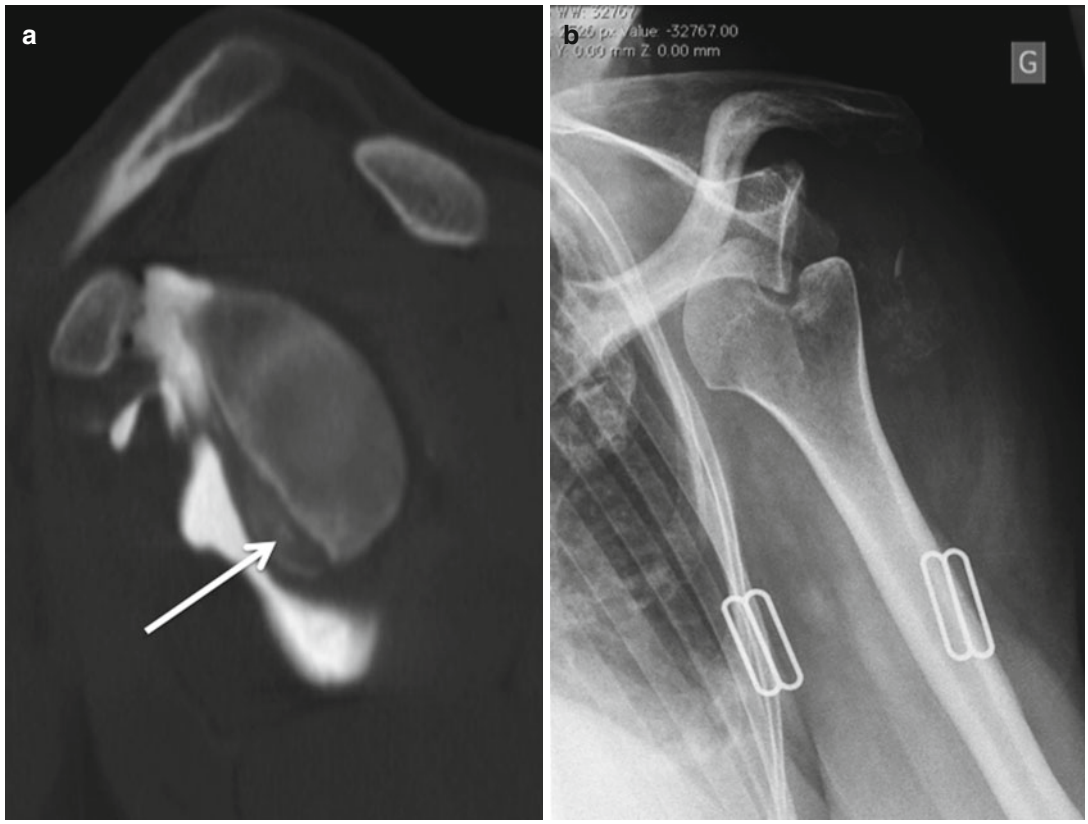

Fig. 17.4 (**a**) Sagittal view of a CT arthrogram of a left shoulder demonstrates a significant Bankart fracture (*white arrow*) that produces an "inverted-pear" glenoid. (**b**) Plain anteroposterior radiograph reveals an anteroinferior glenohumeral dislocation with an "engaged" Hill-Sachs lesion of the humerus

anteroinferior direction and is the most common. Other types including subglenoid, subclavicular, retroperitoneal, and intrathoracic are rare and usually associated with severe trauma [33, 34].

17.3 Clinical Presentation and Essential Physical Examination

The history should document age, hand dominance, occupation, participation in sporting activities, initial mechanism of the injury, the position of the arm (extension, abduction, and external rotation favors anterior dislocation), how long the shoulder stays out, the method of reduction, the number of recurrences (frank dislocation vs. subluxation), and the effectiveness of a previous nonoperative or operative treatment. The diagnosis of recurrent traumatic anterior glenohumeral instability is usually made easily on the basis of the history, radiographs, and a positive apprehension sign. However, when collision athletes are seen, care should be taken because they may not experience clear dislocation or subluxation and only complain of pain or weakness as discussed previously.

A comprehensive physical examination is essential. The aim is to define the direction of instability and the presence of an associated pathologic hyperlaxity and to exclude neurological and rotator cuff impairment. Passive and active glenohumeral range of motion should be assessed. Rotator cuff examination includes strength tests such as belly-press [35], bear hug [36], and Jobe [37, 38] tests and strength in external rotation against resistance. The authors do not

systematically perform tests for anterior and superior labral lesions as they have a poor sensitivity and specificity [39]. The neurovascular status of the upper extremity is assessed, particularly with regard to the axillary nerve since there is a high incidence of injury to this nerve with traumatic instability.

Laxity is a normal, physiologic, and asymptomatic finding that corresponds to translation of the humeral head in any direction to the glenoid [40]. Laxity is assessed with the sulcus sign, anteroposterior drawer, hyperabduction tests, and external rotation elbow at side. The two former tests are only qualitative and are not routinely performed by the authors. Hyperlaxity is constitutional, multidirectional, bilateral, and asymptomatic. Hyperlaxity of the shoulder is probably best defined as external rotation elbow at the side equal or greater than 85 [41]. This non-pathological finding is a risk factor for instability but does not by itself demand treatment unless there is clear pathological laxity. Pathological laxity of the inferior glenohumeral ligament is observed when passive abduction in neutral rotation in the glenohumeral joint is above 105°, when there is apprehension above 90° of abduction, or if a difference of more than 20° between the two shoulders is noted [42]. For apprehension the patient is initially invited to demonstrate his or her functional problem to the examiner (no-touch examination). This examination alone, coupled with a good history, often provides the information needed. However, if the direction of the instability remains unclear, the apprehension (crank) test, an abducted and externally rotated position suggestive of anterior instability, is performed. Fear of dislocation or a feeling of anterior pain is considered positive for damage to the anterior capsulolabral complex, which should be relieved with posterior translation of the humerus (relocation maneuver). To summarize, the physical examination demonstrates instability if the apprehension test is positive, multidirectional hyperlaxity when the external rotation at side is equal or above 85°, and a pathological laxity of the inferior glenohumeral ligament if the hyperabduction test is positive.

17.4 Essential Radiology

Radiographic evaluation is based on whether the dislocation is acute or chronic.

17.4.1 Acute Dislocation

Three-view plain radiographs, including true anteroposterior of the glenohumeral joint, scapular Y (scapular lateral), and Velpeau axillary [43] views, are the mainstay of imaging in the setting of traumatic anterior instability. The latter view is crucial to obtain, as the first two alone do not allow to exclude a dislocation. The goal is to confirm the direction of dislocation and to evaluate associated lesions (Fig. 17.5). Once reduced, further imaging studies in the setting of an associated fracture (computed tomography (CT)), suspicion of rotator cuff injury (ultrasonography or MRI), or vascular impairment (injected CT) may be warranted.

17.4.2 Recurrent Dislocation

The first step is to analyze, if available, plan radiographs with the shoulder out of joint to confirm the direction of instability. Plain radiographs including anteroposterior in neutral, internal, and external rotations, scapular Y, and Bernageau [44] views are then obtained. Bone loss, static instability, post-dislocation arthropathy, and coracoid nonunion (if a Latarjet procedure is planned) have to be estimated. Magnetic resonance imaging (MRI) arthrogram is useful to assess for an anterior labral tear and Hill-Sachs lesion which confirms the instability. Associated intra-articular pathology such as SLAP, HAGL, and rotator cuff lesions or a paralabral cyst is also assessed (Fig. 17.6) [45]. The evaluation is completed by a 3D CT arthrogram in the setting of recurrent instability in which there is primary concern for bone loss. The extent of both glenoid bone loss and Hill-Sachs lesions is best assessed by CT scan and is used to determine the need for Latarjet as opposed to arthroscopic Bankart repair (Fig. 17.7).

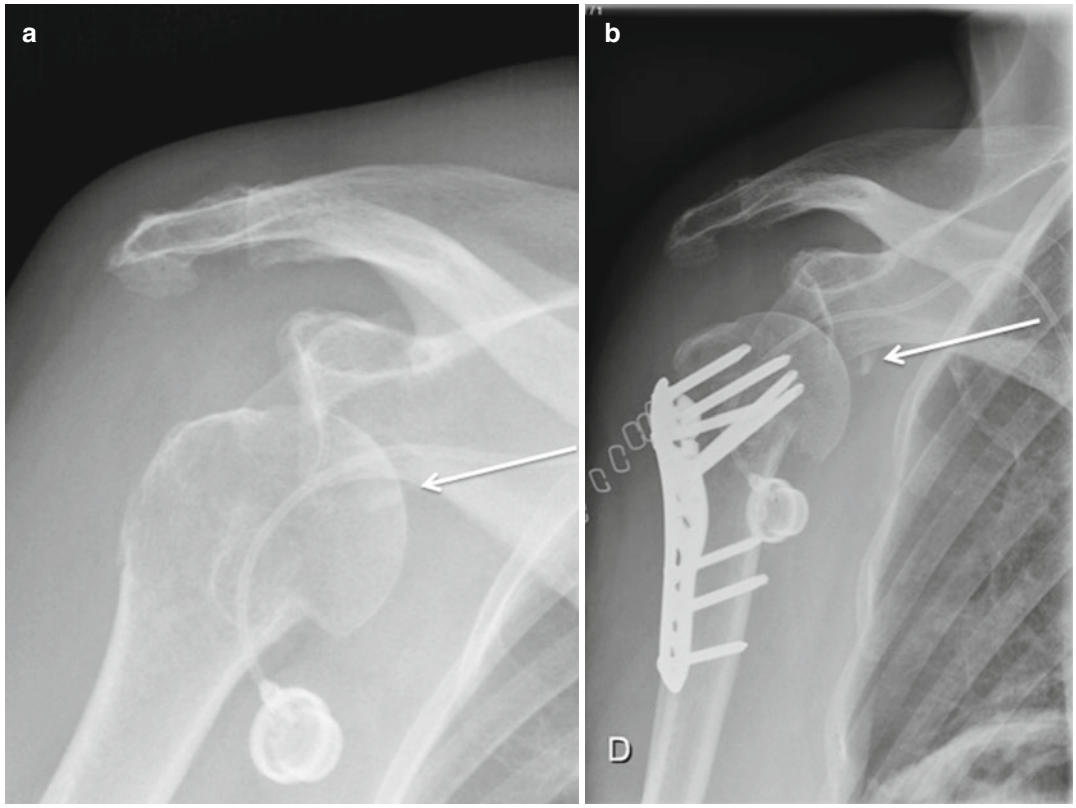

Fig. 17.5 (**a**) Plain radiograph of a right shoulder fracture dislocation. The anterior Bankart fracture (*white arrow*) was overlooked at the initial management. (**b**) The fracture has been fixed with a plate, but the joint will remain unstable if the Bankart fracture (*white arrow*) is neglected

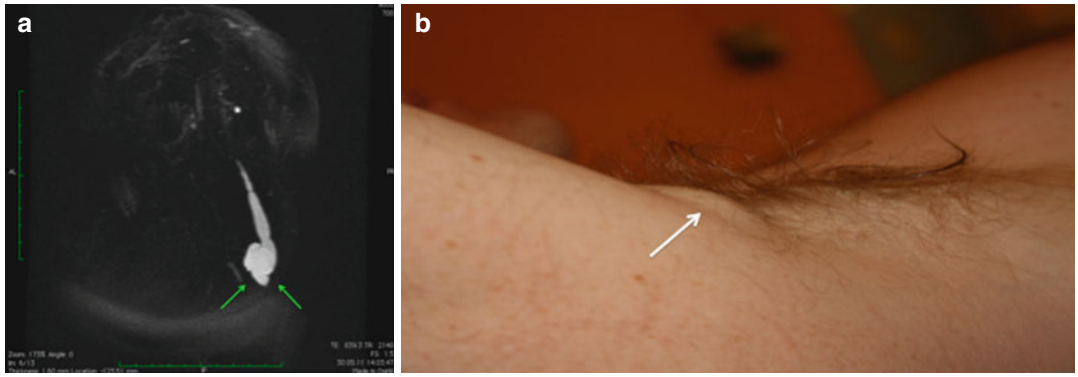

Fig. 17.6 (**a**) 3D maximum intensity projection reconstruction showing a remarkably voluminous cyst (green arrows) dissecting 9 cm from the glenoidal labrum through the long head of the triceps muscle (reproduced/reprinted with permission). (**b**) The patient complains from a lump (white arrow) in his axillary fold.

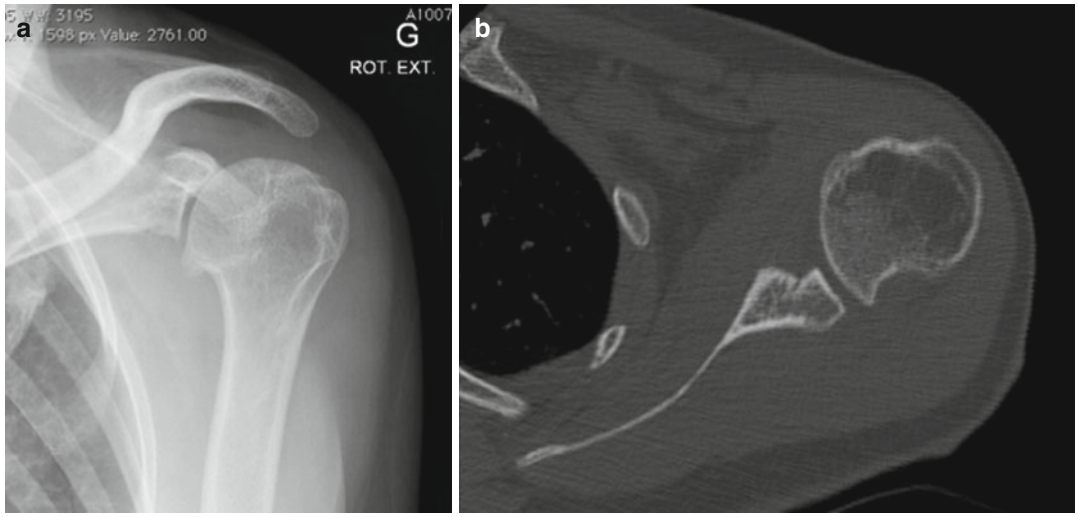

Fig. 17.7 (**a**) Anteroposterior plain radiographs of a left shoulder in a 32-year-old woman presenting with recurrent dislocations. Dislocation arthropathy Samilson [46] **b** is obvious. (**b**) CT scan of the same shoulder demonstrates significant glenoid and humeral bone loss

17.5 Treatment Options

17.5.1 Treatment of Acute First Traumatic Dislocations

The first step, whenever possible, is to obtain a complete set of radiographs before attempting a reduction. This will allow an assessment of the type of dislocation and associated bone injuries. Attempting to reduce a fracture dislocation can have troublesome clinical and legal consequences (Fig. 17.8). Exceptions are an impossibility to have reasonably fast access to radiology or a patient with neurological impairment. Because of the possible association of nerve injuries [47] and, to a lesser extent, vascular injuries (Fig. 17.9) [48], an essential part of the physical examination is an assessment of the neurovascular status of the upper extremity before reduction. There are numerous appropriate methods of reduction that have been described [49–58]. The second step is to use the technique of closed reduction which is mastered by the doctor who will perform the maneuver. The glenohumeral joint should be reduced as gently and expeditiously as possible. In the case of fracture dislocation, the reduction is best performed under general anesthesia to have adequate muscle relaxation. After reducing the dislocation, plain radiographs are obtained to verify the adequacy of the reduction. Results concerning conservative treatment are still debatable [59]. A stable shoulder is obtained at 10 years in only half of the patients with conservative treatment [60]. However, recurrence rate is highly dependent on age and activity of the patient; studies have reported a 72–95 % recurrence in patients under 20 years of age and 70–82 % recurrence between the ages of 20 and 30 years [61–66] and only 30 % in those over 30 years of age [67]. Many patients above the age of 30 would consequently undergo unnecessary surgery if proposed after the first dislocation. Conservative treatment after the first traumatic anterior dislocation may be thus recommended for patients who are not actively engaged in sports, above the age of 30 years old [61], with a low functional demand, with an associated humeral fracture [60], or for the athlete with an in-season shoulder dislocation [4]. For the latter situation, athletes are allowed to attempt to return to competition provided there is enough time left in the season to permit adequate rehabilitation with progression to sport-specific drills. Rehabilitation including return of range of motion and strengthening of dynamic stabilizers may facilitate return to sport within several

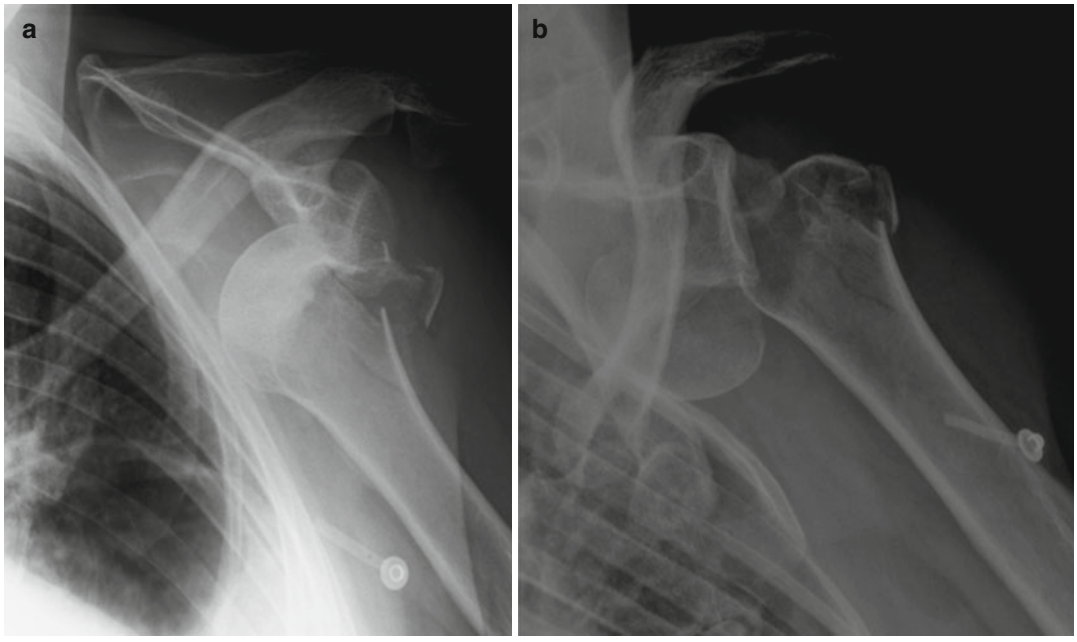

Fig. 17.8 (**a**) An anteroposterior plain radiograph of a left shoulder shows an anterior dislocation with a nondisplaced humeral neck fracture on the prereduction radiographs. (**b**) Radiographs after attempting a closed reduction without adequate muscle relaxation reveal displacement of the fracture with the humeral head remaining anteriorly

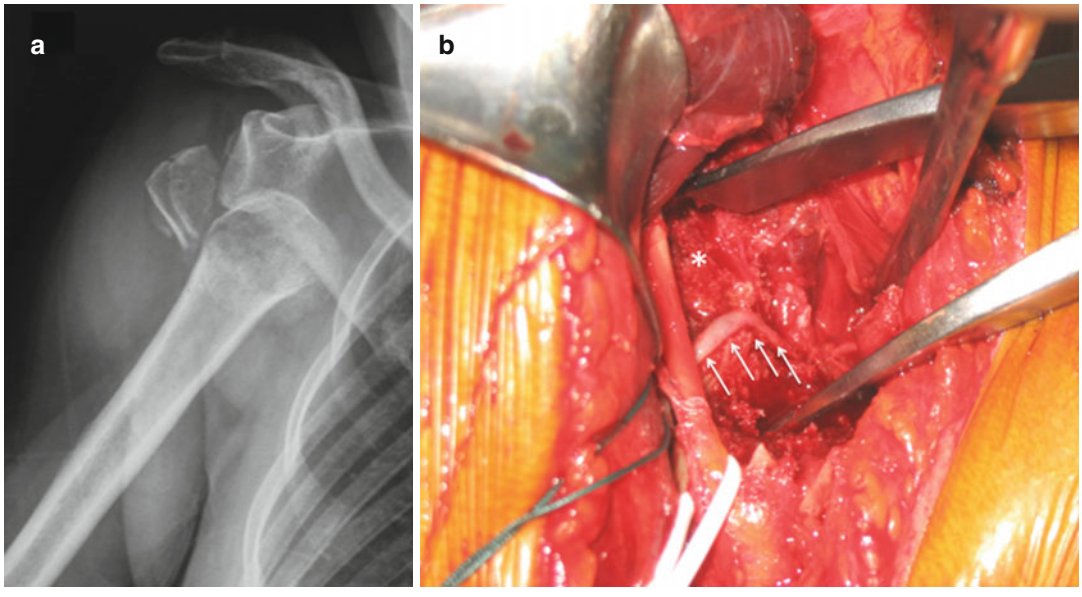

Fig. 17.9 (**a**) A 54-year-old patient sustained a fracture dislocation of the right shoulder. At clinical examination, no peripheral pulse was palpated. (**b**) During open reduction, the axillary artery (*white arrows*) was found kinked around the fractured humeral head

weeks. Motion-limiting braces that prevent extreme shoulder abduction, extension, and external rotation are often prescribed as it may reduce the risk of recurrence. However, such braces are not well tolerated in patients who must complete certain overhead tasks such as throwing. Moreover, a second in-season shoulder dislocation should lead to removal from sport and proceed with stabilization so as to avoid further glenohumeral damage.

A number of studies have compared nonoperative treatment and arthroscopic stabilization. Overall, these studies report a sevenfold reduction in the risk of recurrent instability after arthroscopic stabilization, when compared with nonoperative treatment for the first-time dislocator [59]. A Cochrane review concluded that early surgical intervention is warranted in young adults aged less than 30 years engaged in highly demanding physical activities [68]. Consequently, for patients who are actively engaging in a collision or contact or overhead sport, who risk their life in case of a new dislocation (e.g., firemen, proponents of extreme sports like base jumping and climbing), with associated glenoid fracture, static anterior subluxation, an interposed tissue, or a nonconcentric reduction, or patients with rotator cuff avulsion, conservative measures are usually inadequate and prompt surgery is indicated.

17.5.2 Surgical Treatment of Acute or Recurrence Traumatic Anterior Instability

Recurrent dislocation is not trivial. Each episode creates new lesions and increases the risk of developing dislocation arthropathy. The concept of early operative surgical management of the first-time dislocator has consequently been introduced to address the high recurrence rate in the young athletic population. A surgery should be proposed, as having the ultimate aim to achieve a pain-free stable shoulder while preserving range of motion. The surgical approach is based on the extent of bone loss and patient-specific risk factors for recurrence.

Boileau et al. proposed a simple 10-point scale scoring system (instability severity index score (ISIS)) based on factors derived from a preoperative questionnaire, physical examination, and anteroposterior radiographs to determine the risk of treatment failure following isolated arthroscopic Bankart repair (Table 17.1) [69]. In this model an ISIS of 3 or less was associated with a 5 % rate of recurrence, an ISIS of 4–6 was associated with a 10 % rate of recurrence, and an ISIS over 6 was associated with a 70 % rate of recurrence. Although it has imperfections, this score, validated since [70], has merit to easily remind the clinician of factors that are important to consider when evaluating a patient.

The aim of a Bankart repair is to restore anatomy by reattaching the labrum to the glenoid and tighten the inferior glenohumeral ligament by shifting from inferior to superior. It was previously believed that a minimum of three double-loaded suture anchors had to be used [71]. However, a recent study demonstrated that one to two anchors are enough [72]. One must take caution with the latter as the position of the anchors

Table 17.1 The instability severity index score is based on a preoperative questionnaire, clinical examination, and radiographs

Prognostic factors	Points
Age at surgery (years)	
<20	2
≥20	0
Degree of sport participation (preoperative)	
Competitive	2
Recreational or none	0
Type of sport (preoperative)	
Contact or forced overhead	2
Other	0
Shoulder hyperlaxity	
Shoulder hyperlaxity (anterior or inferior)	1
Normal laxity	0
Hill-Sachs on AP radiograph	
Visible in external rotation	2
Not visible in external rotation	0
Glenoid loss of contour on AP radiograph	
Loss of contour	2
No lesion	0
Total (points)	

AP anteroposterior

is probably the most important factor; in other words, the placement of three anchors likely reflects the fact that the surgeon is adequately placing inferior anchors and obtaining an inferior to superior shift of the pathological lesion. Although this surgery can be performed in an open manner, the advantage of an arthroscopic approach is that it preserves the subscapularis and allows assessment of associated pathology [73–75]. The literature demonstrates that patients with low risk of recurrence will benefit from either an anatomic open or arthroscopic repair with an acceptable rate of recurrence [76].

Remplissage has been described by Connoly [77] and may be used as an adjunct to arthroscopic Bankart repair in the setting of a large Hill-Sachs lesion with glenoid bone loss of <25 %. This technique consists of a posterior capsulodesis and infraspinatus tenodesis that fills the Hill-Sachs lesion. The purpose is to render the Hill-Sachs lesion extracapsular, avoiding its engagement. Wolf and Arianjam and Boileau et al. recently presented encouraging mid- to long-term results of arthroscopic remplissage and concomitant anterior Bankart repair [13, 78]. However, the indication for remplissage has not been well defined. Recently, DiGiacomo et al. introduced the concept of an "on-track vs. off-track" Hill-Sachs lesion [27]. This view incorporates the concept of the glenoid track by Yamamoto et al. which describes the contact of the posterior humeral head with the glenoid during abduction [79]. Based on study in normal individuals, this zone of contact or "glenoid track" averages 83 % of the glenoid width. In the proposal of DiGiacomo et al., the normal glenoid diameter is determined via CT scan of the contralateral glenoid or arthroscopic assessment (by doubling the distance from the glenoid bare area to the posterior glenoid rim). Then, the glenoid track is determined by multiplying the normal glenoid diameter by .83 and subtracting the anterior glenoid bone defect. Finally, the distance from the medial margin of the infraspinatus to the most medial aspect of the Hill-Sachs lesion (or Hill-Sachs interval) is measured. If the Hill-Sachs interval is greater than the remaining glenoid track, the lesion is considered "off-track," and they recommend a remplissage in addition to an arthroscopic Bankart repair. However, this elegant method has to be clinically confirmed and factors like ligament status (HAGL lesions, laxity, and translation that render all Hill-Sachs lesions engaging) or ability to obtain intraoperatively reliable measures are not taken into account in the evaluation.

In the setting of glenoid bone loss ≥25 % of the glenoid diameter, an arthroscopic Bankart repair has an unacceptably high rate of recurrence. Burkhart and DeBeer reported a 4 % recurrence rate for arthroscopic Bankart repair when glenoid bone loss was <25 %. However, with glenoid bone loss ≥25 %, the recurrence rate was 67 % with an arthroscopic approach [25]. They subsequently recommended a Latarjet procedure in the population with substantial glenoid bone loss. In 1954, Latarjet reported a coracoid transfer procedure in which the inferior aspect of the coracoid was secured to the anterior glenoid. The excellent stability of this procedure is obtained by a triple effect first proposed by Patte and Debeyre [80]: (1) the sling effect of the conjoint tendon when the arm is abducted and externally rotated, (2) the "bony effect" that increases or restore the glenoid anteroposterior diameter, and (3) the retensioning of inferior capsule to the stump of coracoacromial ligament, rendering the coracoid extra-articular. The Latarjet procedure is associated with a very low recurrence rate even in the setting of substantial glenoid bone loss and has become the gold standard of treatment in such settings. In addition, this nonanatomic method of anterior glenohumeral stabilization has progressively expanded and is actually the primary technique of choice for many European surgeons, as it prevents recurrent anterior instability in approximately 95–99 % of cases [81, 82]. This procedure is also favored by some as a first choice in many contact athletes [83]. While traditionally an open procedure, the technique can now be performed arthroscopically [10, 11, 84, 85]. To date, however, there is actually no proven benefit of an arthroscopic approach to the procedure, and the risk of complication remains high with a large learning curve [86].

Finally, some authors have recommended autogenous iliac crest or tibial allograft as a

means of restoring glenoid bone loss. However, it has been demonstrated that the most important effect of the Latarjet is provided by the conjoint tendon at both the end-range and the mid-range arm positions [87]. We therefore believe that the indication for iliac crest or tibial grafting should be limited to revision procedures such as following a failed Latarjet reconstruction.

17.6 Author's Preferred Treatment

Treatment is based on patient factors and associated pathology as previously discussed. In general, for patients under the age of 30, we offer a primary stabilization following a first traumatic anterior instability episode. Such patients are counseled on the natural history or anterior instability and the potential for subsequent injury. For the majority of patients over the age of 30, nonoperative treatment is advised with standard sling immobilization for 3 weeks followed by progressive strengthening and return to activities. For such patients with persistent weakness or recurrent instability, an MRI or MRI arthrogram is obtained to evaluate for an associated rotator cuff tear, and stabilization is performed.

In most cases of glenoid bone loss <25 % (based on preoperative imaging), we perform an arthroscopic Bankart repair using anterior and posterior working portals while viewing from an anterosuperolateral portal and base our repair on the extent of the pathology usually using two to four anchors. We have recently adopted the proposal of DiGiacomo et al. for intraoperatively determining the need for remplissage [27], but as previously noted, the outcomes of this algorithm are not clearly defined.

In the setting of glenoid bone loss ≥25 % of the glenoid diameter, we perform a Latarjet reconstruction [83]. One of us (AL) has experience with an arthroscopic Latarjet and utilizes this technique frequently. We believe that the arthroscopic Latarjet, after modifications, will be safer and thus a reliable technique but cannot at the current time recommend this for widespread use given the complexity of the procedure and high learning curve.

On the basis of the aforementioned elements, we use a treatment paradigm for all patients with anterior instability (Fig. 17.10). However, if one feels that risk for recurrence is high despite the lack of substantial glenoid bone loss (e.g., young male contact athlete), we do not hesitate to recommend Latarjet reconstruction.

17.7 Rehabilitation

17.7.1 Nonoperative Treatment of Acute First Traumatic Dislocations

Although positioning the arm in external rotation has been recommended, it has now clearly been demonstrated that immobilization of the shoulder in internal rotation after primary, traumatic anterior shoulder dislocation is sufficient [88, 89]. There is conflicting evidence regarding the length of immobilization required after dislocation, but 3 weeks is typically recommended, followed by physical therapy for strengthening of the rotator cuff and scapular stabilizers. Range of motion of the elbow, wrist, and hand is permitted immediately. Then, closed-chain exercises facilitate rotator cuff function to enhance joint stability and stimulate muscular coactivation and proprioception [90]. For throwing athletes, a program is initiated and advanced, beginning at 3 months. A full return to sports is typically permitted at 5–6 months.

17.7.2 Rehabilitation Protocol After Bankart and Remplissage Stabilization

The shoulder is immobilized for 4 weeks using a sling. Passive and assisted-active exercises are then initiated for forward flexion and external rotation. After 6 weeks, patients begin strengthening exercises of the rotator cuff and scapular stabilizers. For patients with a remplissage, strengthening is delayed until 12 weeks postoperative. Patients are permitted to practice noncontact

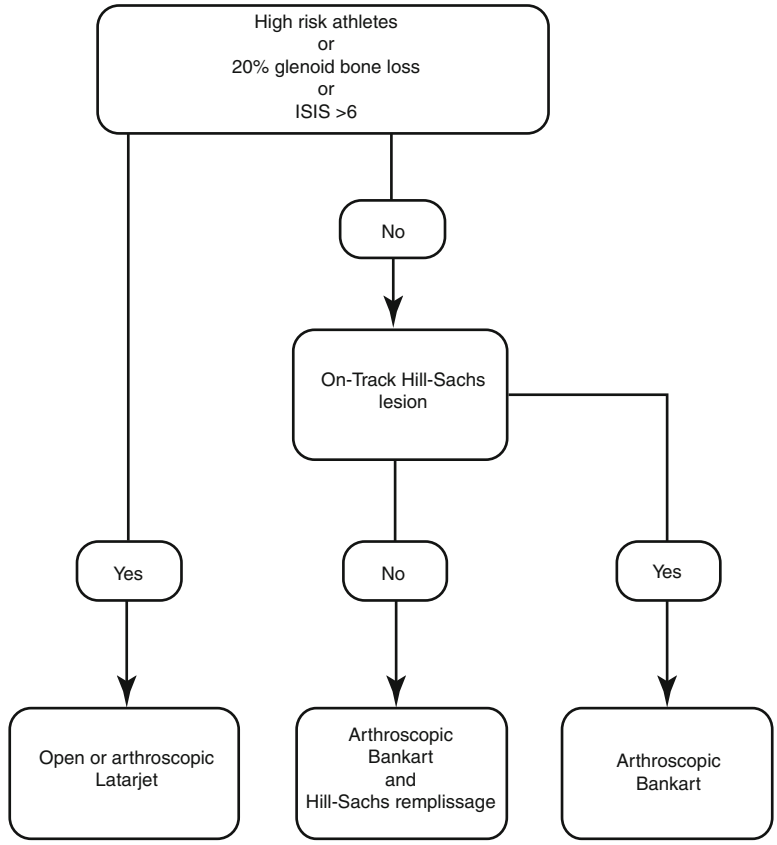

Fig. 17.10 Treatment paradigm proposed by the authors for patients with anterior instability

sports as soon as they recover their range of motion. Full return to throwing or contact sports is usually allowed after 6 months according to each individual's functional recovery.

17.7.3 Rehabilitation Protocol After Latarjet Reconstruction

The shoulder is immobilized for 10 days using a sling. The patient is asked to stretch in flexion and external rotation at least five times per day. No physical therapy is prescribed. The patient is not allowed to carry with his operated arm or to flex the elbow against resistance during the first 6 weeks. Activities of daily living are encouraged as comfort permits. At 6 weeks, noncontact sports are allowed. Return to contact sports is usually possible after 3 months assuming confirmation of bony union of the coracoid graft.

17.8 Advantages and Pitfalls, Complications

Arthroscopic Bankart stabilization with use of suture anchors offers the advantage of being minimally invasive, allows assessment of associated pathology, and allows the surgeon to restores anatomy while reattaching the labral lesion and retensionning the glenohumeral ligament. While the short-term outcome has been excellent, mid-term reported results show higher rates of recurrent of instability. According to the meta-analysis by Hobby et al. [91], recurrence (dislocation and subluxation) after arthroscopic Bankart repair with suture anchors varies between 0 and 29.6 %, with a mean of 8.9 %. This rate of course varies with patient factors, particularly the amount of bony deficiency [25, 71]. Preoperatively, pitfalls are consequently to underestimate risk factors for recurrence for this surgery [69]. Several technical

factors are also important to success. It is important to place anchors at the margin of the articular surface (as opposed to the glenoid neck) to allow recreation of the labral bumper. The surgeon must be sure to obtain a proper inferior to superior shift of the capsule (Neer's modification). We believe this is best performed by viewing from an anterosuperolateral portal which allows an on-face view of the glenoid and consequent ability to properly restore the anatomy and obtain this shift. Another complication is the development of dislocation arthropathy with a rate similar to other type of procedures [92].

Bankart repair combined with Hill-Sachs remplissage for large defects of the posterosuperior aspect of the humeral head may be an elegant approach in case of isolated humeral defect. Reported results are promising with a high rate of healing of the posterior aspect of the capsule and the infraspinatus tendon into the humeral defect and a moderate loss of external rotation with the arm at the side. Moreover, most patients were able to return to sport including those involving overhead activities, around 70 % at the same level [13, 78]. The Hill-Sachs remplissage is believed to be a posterior capsulotenodesis that acts as a checkrein diminishing anterior humeral head translation and reducing the risk of postoperative redislocation. However, the authors of this chapter have observed that according to the location of the impaction fracture, the procedure actually corresponds to a capsulomyodesis including the teres minor muscle, rather than a capsulotenodesis as classically described (Figs. 17.11 and 17.12). Even if this observation is not reassuring due to the importance of external rotators as active stabilizers, it may not be clinically relevant. One technical trick during remplissage is to pass the sutures through the posterior capsule prior to the Bankart repair but not tie the sutures until after the Bankart repair is completed. This order of steps allows the surgeon to access the posterior humeral head before this space swells, but not tightening the sutures immediately allows one to maintain the working space needed for Bankart repair. If the remplissage sutures are not tied blindly, one can either perform a subacromial bursectomy before performing the remplissage or proceed with the Bankart repair and then replace the anchor guides over the remplissage sutures during later bursectomy so as to protect the sutures from inadvertent damage. Concerns about remplissage may include the potential muscle lesion to the external rotators, increased cost, and increased difficulty and operative time. Yet, beyond a slight loss in postoperative external rotation, there is no documented additional complication to remplissage.

Open or arthroscopic Latarjet reconstructions are both demanding procedures. With this procedure, return to sports activities is possible for at least 83 % of patients regardless of the size of glenoid defect. In a study of 107 patients, Lädermann et al. reported a mean postoperative Walch-Duplay score [93] of 93, good or excellent results in 97 % of cases, and 95 % of patients very satisfied or satisfied with their outcome [81]. However, complications exist both in the short and long term. Short-term complications include infection, recurrent glenohumeral instability, and neurologic injury [81, 94, 95]. Neurological impairment of the musculocutaneous nerve is avoided by gently manipulating the coracoid process during preparation and avoiding excessive medial dissection [96]. The suprascapular nerve is at risk posteriorly from placement of the screws and can be avoided by parallel screw placement within 10° of the glenoid in the axial plane (Fig. 17.13) [94]. The rate of recurrent instability is 1.7–14.2 %, with a mean of 6.8 % [97–100]. In a recent study, the rate of recurrent instability following a Latarjet reconstruction was two times lower than that following an arthroscopic Bankart repair [101]. In long-term follow-up, Lädermann et al. reported recurrent instability or subluxation in only 2 of 117 (1.7 %) patients. Four of the 117 patients reported persistent apprehension that could correspond to persistent instability or to brain sequelae [30]. Recurrent instability is related to radiological complications including graft pseudarthrosis (1.7 %), osteolysis (3.4 %), fracture (0.9 %), and migration (0.9 %) [81]. In order to optimize bone graft healing, it is advisable to (1) halt tobacco use preoperatively [95, 102], (2) discontinue anti-inflammatory medication for 6

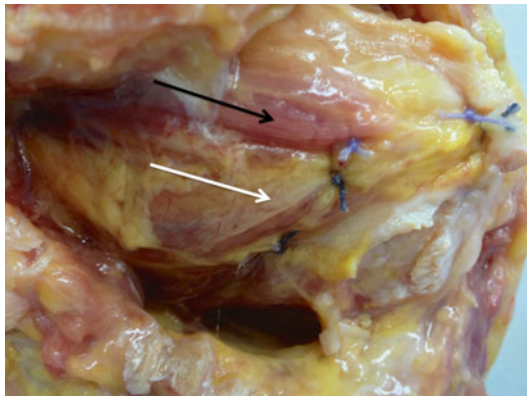

Fig. 17.11 Posterior view of a right shoulder specimen after rotator cuff repair and Hill-Sachs remplissage (three lower knots). Note the proximity of the superior knot to the infraspinatus muscle (*black arrow*). The two inferior knots perforate the teres minor muscle (*white arrow*) realizing a capsulomyodesis

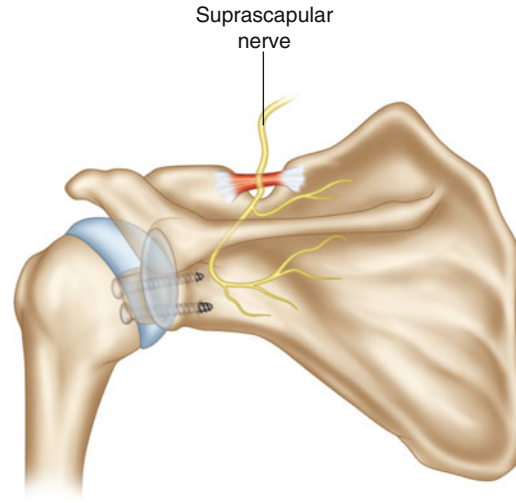

Fig. 17.12 The infraspinatus branches of the suprascapular nerve are at risk during screw placement for Latarjet reconstruction when a divergence of more than 10° between the screws and the glenoid surface in the axial plane is noted (Reprinted with permission)

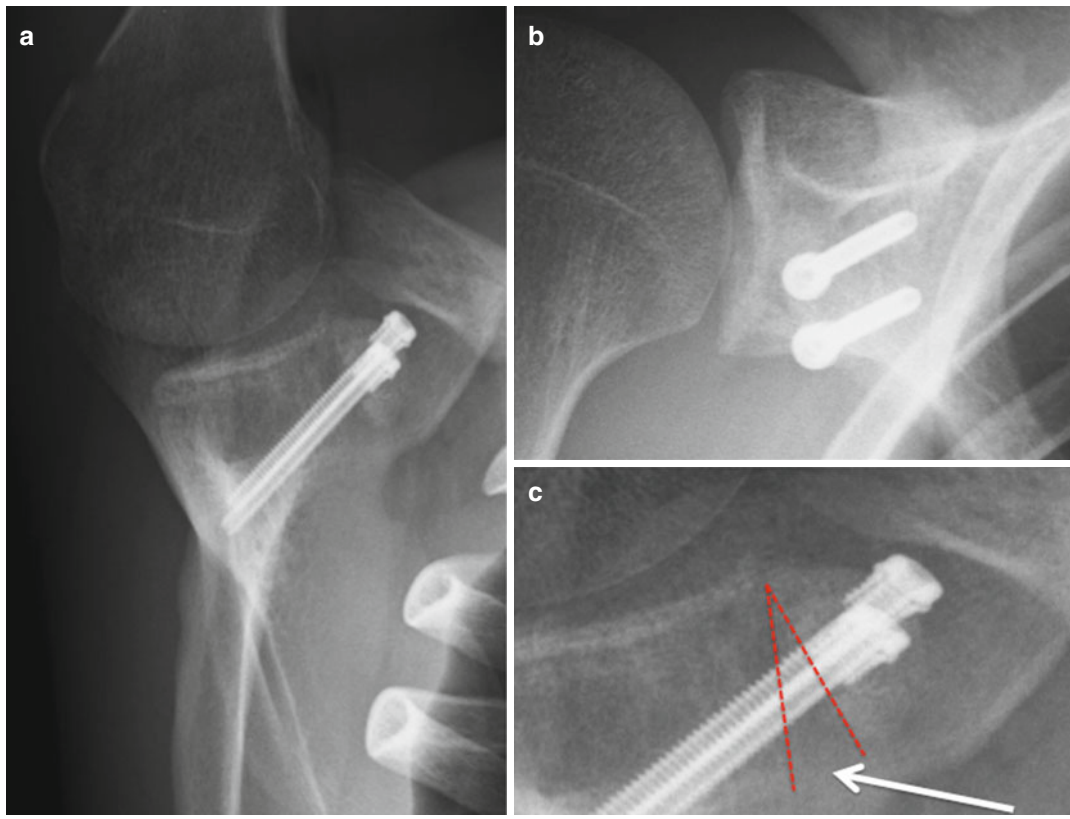

Fig. 17.13 Axial (**a**) and anteroposterior (**b**) plain radiographs of a left shoulder. The two screws diverge from the plane of the glenoid, pointing in direction of the spinoglenoid notch. A magnification (**c**) of the axial view demonstrates poor contact (*white arrow*) between the glenoid and the graft (delimited by *red dotted line*). Parallel screws would have led to better contact and a lower risk of suprascapular nerve injury

weeks postoperatively [103], (3) decorticate the coracoid surface to expose a broad flat cancellous bed, (4) use two screws (as opposed to one), and (5) prevent elbow flexion against resistance during bone healing (Fig. 17.14). Placing the coracoid graft flush with the glenoid is recommended, as a medial position is associated with recurrence. The principal long-term complications are dislocation arthropathy and associated pain. Post-dislocation arthropathy is found in approximately 30 % of patients [81, 92]. Of those, 30 % are graded Samilson [46] 1, 3 % Samilson 2, and 3 % are graded Samilson 3 [81]. The precise cause of arthropathy following Latarjet is unknown [104]. Reported risk factors include surgery in patients older than 40 years of age and lateral overhang of the transferred coracoid process in relation to the glenoid rim [81]. The former risk factor might be explained by a greater number of shoulder dislocations or subluxations prior to stabilization. Indeed, a prolonged delay between the initial dislocation and surgery contributes to a greater likelihood of developing dislocation arthropathy. An additional factor may be less favorable biology secondary to aging which correlates with poorer cartilage properties and less capacity for self-repair, leading to extended cartilage damage at the time of stabilization. Contrarily, the presence of hyperlaxity is protective against arthropathy [81]. We believe that hyperlaxity may decrease postoperative contact pressure of the humeral head on the glenoid and thus prevent development of secondary arthritis. No difference is seen with regard to sex, sports activity, or arm dominance [81].

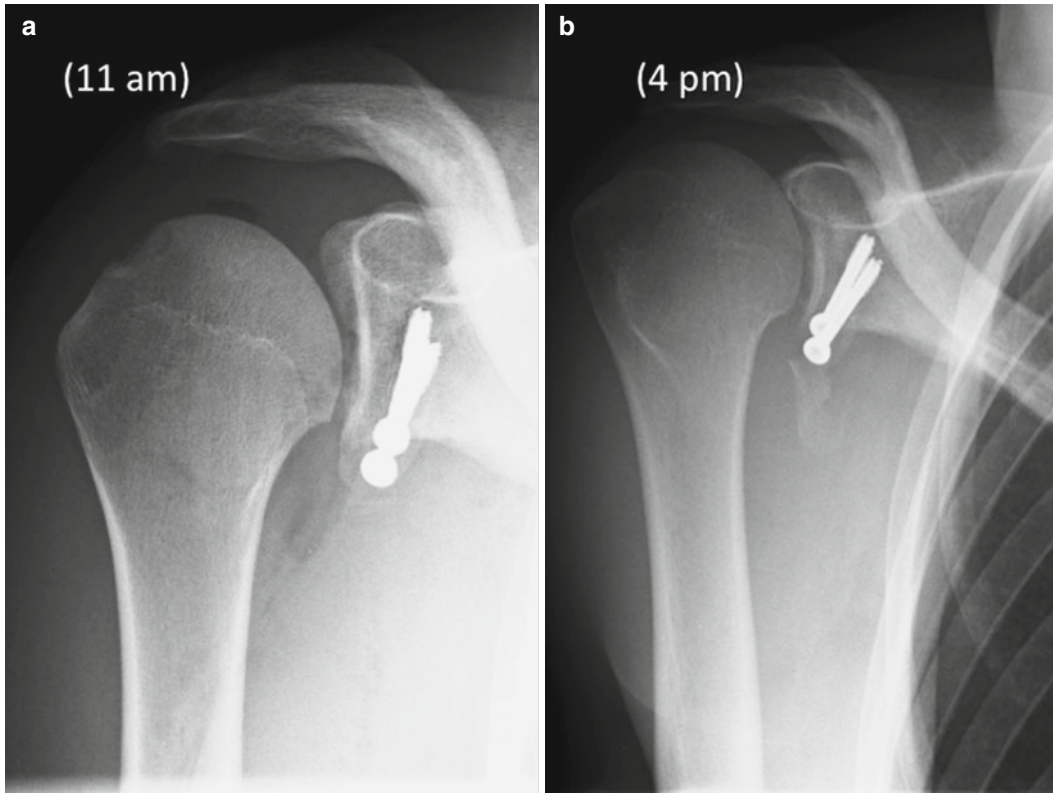

Fig. 17.14 (**a**) Radiographs of a left shoulder 10 days after a Latarjet reconstruction. The patient has not been allowed to remove the sling. (**b**) The patient ignored the recommendation and was immediately jogging. He returned 5 h after the last examination. Controlled radiographs revealed pullout of the graft due to contraction of the short head of the biceps on the coracoid graft

17.9 Experience in Treatment of Athletes

The goal of surgical treatment of recurrent instability in contact athletes is to achieve a stable shoulder, allowing early return to sports participation without recurrence and with minimal risk of complications. The management of anterior instability in athletes does not differ substantially from the principles previously discussed since the majority already occurs in a young athletic population prone to bone loss because of the high-energy injuries they sustain. Shoulder instability can occur in overhead throwing athletes (chronic overuse injuries) but more commonly occurs in contact athletes (acute traumatic dislocations). The latter category by definition has risk factors for recurrence both in terms of natural history and thus would score high on the ISIS, resulting in the Latarjet procedure being often recommended.

Cho et al. reported on 29 athletes treated with arthroscopic stabilization and noted a 17 % rate of recurrence overall, with a 7 % rate in non-collision athletes and 29 % rate in collision athletes [105]. However, the repairs in this series were mixed with some patients having a suture anchor repair and some having a repair with tacks which has been shown to be suboptimal compared to suture anchors. Similarly, Owens et al. reported a 14 % rate of recurrence following arthroscopic stabilization of first-time dislocation in a series of 40 athletes treated with bioabsorbable tacks [106]. On the other hand, Burkhart and DeBeer demonstrated that even in contact athletes, an acceptable recurrence rate (6.5 % in their series) can be achieved with a suture anchor-based arthroscopic Bankart repair [25]. However, recurrence in contact athletes was 89 % when glenoid bone loss exceeded 25 % or there was large Hill-Sachs lesion. Thus, the importance of recognizing substantial bone loss is as if not more important in collision athletes. In fact, our threshold for Latarjet is 20 % of glenoid bone loss (or lower in some cases) in this population.

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Traumatic Anterior Shoulder Instability: Part II. Bony Bankart – Small Versus Large Lesions

18

Laurent Lafosse, Simon Fogerty, and Claudio Rosso

18.1 Introduction

Anterior glenoid bone defects are frequently associated with shoulder instability and are considered one of the major causes of recurrence of instability after shoulder stabilization [22]. The glenohumeral joint is inherently predisposed to instability by its bony architecture, especially with the mismatch between the sizes of the humeral head and glenoid. Proper early recognition of glenoid bone injury in the setting of recurrent instability will assist in making safe and successful nonoperative or operative decision making, particularly in the athletic patient [1]. The bony Bankart lesion is one of the many fractures that can occur around the glenohumeral joint when an athlete sustains an anterior shoulder dislocation. If glenoid bone loss is present, the humeral head often easily subluxates over the glenoid in the midranges of abduction (30–90°) and lower levels of external rotation [1]. The lesion significantly predisposes the patient to recurrent instability [21]. Presence of a defect was significantly associated with recurrence of dislocation compared with a single episode of dislocation in a study by Milano et al. [22]. It can be difficult to accurately define the size of the fracture fragment as a percentage of the glenoid. To date, the literature has been sparse and evidence is lacking (no level 1 or 2 studies) regarding the treatment guidelines for repairing bony Bankart lesions according to size.

Recent advances in arthroscopic instrumentation and techniques presently allow minimally invasive and arthroscopic reconstruction of glenoid bone defects and osteosynthesis of glenoid fractures. This chapter underlines the role of glenoid bone deficiency in recurrent shoulder instability, provides an update on the current management regarding this pathology, and highlights the modern techniques for surgical treatment. Therefore, it can help orthopedic surgeons in the treatment and decision making when dealing with these difficult-to-treat patients in daily clinical practice [21].

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18.2 Pathoanatomy and Biomechanics

A challenging problem in shoulder dislocations can be significant bony resorption of the fragment taking place, starting from the time of initial injury. This means that at the time of fracture fixation, the fragment could be considerably smaller than it was initially and therefore too small to succeed in regaining stability of the joint [24]. Nakagawa demonstrated that most bony fragments showed severe absorption within 1 year after the primary traumatic episode. They recommended that before arthroscopic Bankart repair, not only glenoid defects but also bone fragment resorption should be assessed [24]. Park et al. reported that the fragment size decreased from that measured preoperatively to the size measured 3 months after surgery [27]. However, the size of the fragment was maintained between 3 months and 1 year postoperatively. They concluded that reattachment of small bony fragments fixed in the anatomical position together with the labrum can be successful [27].

The presence of a bony defect has been significantly associated with recurrence of dislocation compared with a single episode of dislocation, increasing number of dislocations, male gender, and type of sport. The size of the defect was significantly associated with recurrent dislocation, increasing number of dislocations, timing from first dislocation, and manual work [10, 22]. The presence of a critical defect was significantly associated with a number of dislocations and age at first dislocation. Bony Bankart lesion was significantly associated with male gender and age at first dislocation [22].

Bony Bankart lesions have been observed more often in patients with a higher frequency of dislocation [10]. Early surgical treatment is a good option for young athletic patients with bony Bankart lesions and a short interval between the first and second dislocation [32].

Jiang et al. reported functional results after bony fragment reduction and fixation with suture anchors [13]. They determined the correlation between reduction and healing of the fracture and postoperative stability of the glenohumeral joint after arthroscopic repair. The CT scans during the follow-up period showed a nonunion of the bony fragment in 13.5 % of cases (5/37). The reconstructed size of the glenoid was <80 % in three of the four failure cases but >80 % in all of the successful cases. Arthroscopic reduction and fixation of a bony Bankart lesion can achieve good results in selected cases. They concluded that the size of the reconstructed glenoid is crucial to the success of the surgery [13].

The effect of a glenoid defect on stability was studied by Itoi et al. creating an anteroinferior bony defect. The authors determined a threshold of 21 % of glenoid length to result in instability even after a Bankart repair [12]. In another study, Gerber et al. demonstrated that a glenoid osseous defect of more that 50 % of glenoid width would lead to a 30 % diminution of dislocation forces [9]. Another study showed that the critical angle of glenoid bone loss is 80–100° if the 12 o'clock position is considered 0° and the 6 o'clock position is considered 180° [41]. From these studies, the clinical indication for bone grafts in anterior shoulder instability continuously developed. Today, based on the above biomechanical data, a bony reconstruction is recommended in patients with bone loss of greater than 20–25 % of the glenoid surface area [21].

The importance of soft tissue lesions around the glenoid should not be overlooked, and while this chapter deals principally with bony lesions, the status of the soft tissue elements involved heavily affects decision making. When reviewing imaging, a CT may be useful for assessing the bony lesion and labrum but not so helpful when it comes to looking at the ligamentous tissue, for which an MRI is more appropriate.

18.3 Classification

There are several classifications dealing with glenoid bone loss in anterior shoulder instability.

Initially, Bigliani et al. classified glenoid bony defects into three types with type I representing an avulsion fracture with the capsule still attached; type II being a malunited, medially displaced fragment; and type III representing an eroded glenoid (type IIIA <25 % and type IIIB >25 % deficiency of glenoid diameter) [2]. Itoi

et al. then described osseous glenoid defects as percentages of glenoid length with increments of 9, 21, 34, and 46 %. They found the increment of 21 % as a threshold of significantly increased anterior instability. However, they found this was not true if a Bankart repair had been performed and that this threshold could be extended into more severe bone loss [12]. Gerber and Nyffeler presented another classification describing static and dynamic instabilities. This classification seems to be of little help in the decision-making process if a Bankart repair suffices to treat different sizes of glenoid fragments.

It is important to note that instability does not only depend on bone loss but also on the location of the osseous defect. This fact was described as the critical angle of 80–100° corresponding to approximately the 2:30–3:30 position on a right shoulder [41] which supported the statement of a previous 3D study showing a mean glenoid defect in recurrent shoulder dislocations at the 3 o'clock position [34].

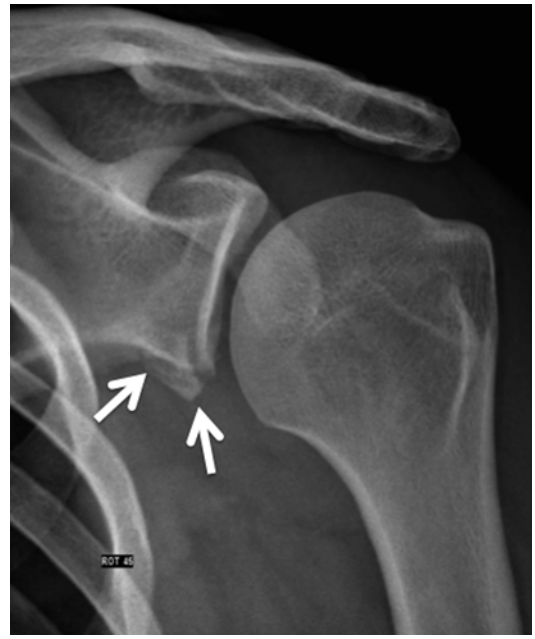

Fig. 18.1 This figure shows a large bony Bankart lesion (*white arrows*) in an AP standard X-ray

18.4 Clinical Presentation and Essential Examination

18.4.1 Essential Radiology

Imaging workup should begin with plain radiographs (anteroposterior (AP), Bernageau, or axillary view (Figs. 18.1, 18.2, and 18.3), Y-Neer), but advanced imaging such as computed tomography (CT) or magnetic resonance imaging (MRI) scans should be obtained if there is any suspicion of bone loss [1].

A significant advancement in the care of patients with shoulder instability would be the ability to categorize the patients with first-time dislocations to an initial treatment plan with the most beneficial outcome. MRI could be a useful imaging modality to find lesions after shoulder dislocation. In a study by [35], 58 patients with traumatic anterior shoulder dislocation were treated with closed reduction and were examined by MRI after 2 weeks. The hemarthrosis or effusion present in the joint after the primary dislocation was used as a contrast for arthrography to identify the lesions present on MRI. At

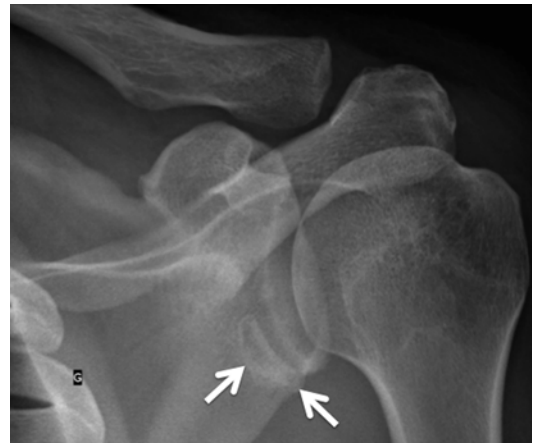

Fig. 18.2 This figure shows an AC joint-centered X-ray of the same patient as in Fig. 18.1. The large bony Bankart lesion can be seen (*white arrows*)

follow-up more than 8 years later, the MRI findings were compared to the shoulder function, shoulder stability, Rowe score, and Western Ontario Shoulder Instability Index (WOSI). Besides the age of the patient being above 30, the MRI findings analyzed showed that a bony Bankart lesion is a good prognostic factor for a good functional result and a stable shoulder after

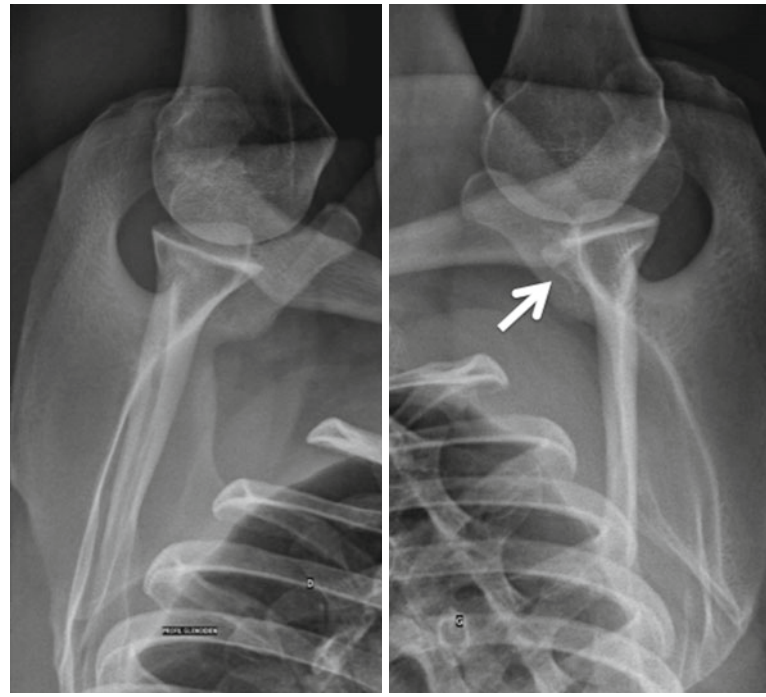

Fig. 18.3 This figure depicts the so-called Bernageau axillary view depicting adequately the anterior and posterior parts of the glenoid. The *white arrow* marks the medialized large bony Bankart lesion (same patient as Figs. 18.1 and 18.2). In case of resorption, the glenoid osseous defect can already be estimated

a primary dislocation. The glenoid rim fracture was only detected on plain radiographs in 60 % of those found on MRI [35].

For this reason, the authors are working on a study creating a threshold of anterior translation in a loaded open-MRI condition for patients after shoulder dislocation. Briefly, the humerus is pulled anteriorly with 20 N and the arm weight neutralized by a lever-arm system. On a 3D reconstruction of the open-MRI acquisitions, glenohumeral translations are calculated. The system was validated on healthy subjects and now patients with status post shoulder dislocation are being analyzed.

Accurately measuring the size of the osseous defect requires advanced imaging modalities. Although computed tomography (CT) is generally thought to be more accurate to estimate bony fragments (Fig. 18.4), a recent study on 18 cadaveric glenoids showed a similar accuracy of MRI when compared to CT and 3D CT, using the circle method [11]. However, the drawback of this study was that the cadaveric glenoids were not surrounded by soft tissues, which makes the distinction between bone and labral-capsular complex difficult. One study correlated glenoid bone loss seen on a 3D en face view to arthroscopic findings. Arthroscopic findings were assessed with a probe measuring the anterior-posterior width at the level of the bare spot. The authors found a high sensitivity of 92.7 % and specificity of 77.8 %. Currently the "circle method" in a 3D CT seems to remain the gold standard for the estimation of glenoid bone loss preoperatively [37]. The circle method establishes a percentage out of two areas. It uses the area (area 1) of a circle around the glenoid touching the superior and inferior glenoid rim. The area of the fragment (area 2) is estimated with a freehand measurement tool. The percentage bone loss is the ratio between area 2 and area 1. Yet, this method does not account for the location of the fragment.

A possible Hill-Sachs lesion should be ruled out, and if present, the glenoid track should be estimated according to Yamamoto et al. to control if the Hill-Sachs lesion might be too medial and thus engage [42]. The glenoid track is defined as 84 % of the glenoid width implying the importance of the glenoid bone loss decreasing the glenoid width.

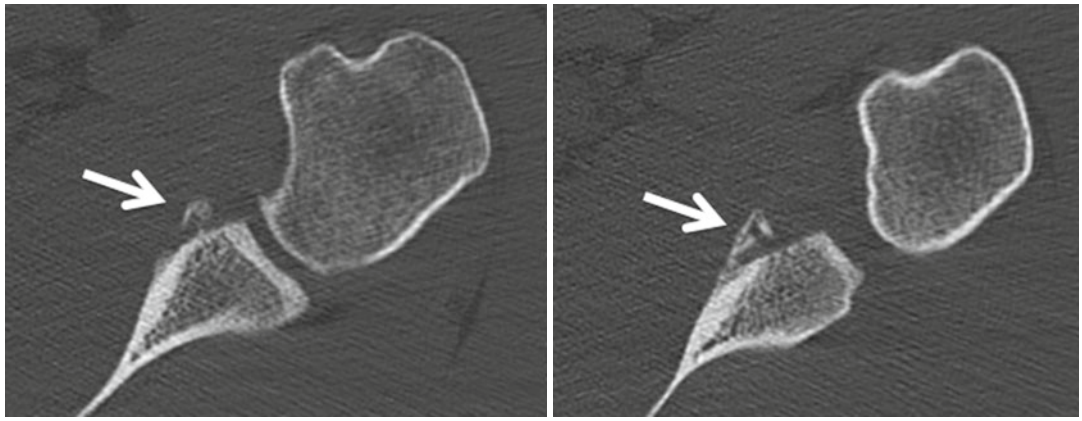

Fig. 18.4 This figure shows a medium-sized (>5 mm) bony Bankart lesion that is medialized. Note the difference in lesion size between the two CT slices. This lesion already demonstrates medialized bony healing and thus does not qualify for a simple Bankart repair. Additionally, the estimated glenoid bone loss represents more than 25 % of glenoid width. The arrows point to the medialized bony Bankart lesion

The authors' preferred method is the estimation of anterior glenoid bone loss on the en face view in a 3D CT scan, which is obtained for all patients preoperatively. Nevertheless, the authors would like to emphasize that bony fragments are usually underestimated even in a 3D scan, and this should be taken into consideration in decision making.

When reviewing imaging, a CT may be useful for assessing the bony lesion and labrum but not so helpful when it comes to looking at the ligamentous tissue, for which an MRI is more appropriate. Decision making depends upon information being available about the status of the ligamentous tissue as well as the bony defect. It may be that only on arthroscopic examination of the shoulder does the surgeon become aware of significant ligamentous injury necessitating a bony reconstruction.

18.5 Disease-Specific Clinical and Arthroscopic Pathology

Firstly, a comprehensive and specific history of the patient should be noted. Demographic parameters such as age, gender, job, type of sport (contact vs. non-contact), and level of sport (competitive vs. recreational) should be recorded. Next, the details of the dislocations should be taken including how many shoulder dislocations have taken place, direction of the shoulder dislocation, who reduced the shoulder joint (patient, physician, physician in operating room), and previous shoulder operations.

The above-stated factors are important in determining the needs of the patient and the severity of the injury (reduction in the operating room suggests a severe injury with Hill-Sachs lesion and glenoid bone loss).

Clinically, range of motion is tested including internal and external rotation at 90° of humeral abduction (IR2 and ER2, respectively). Most patients will complain of apprehension at ER2, which should be noted besides the measured angles. Apprehension should not only be tested at 90° but also at 0° and 140° of humeral abduction. The Gagey test gives an estimate of inferior glenohumeral ligament laxity [8]. Rotator cuff integrity is tested with the Jobe (supraspinatus), the palm-up (infraspinatus), and the belly-press (subscapularis) test.

Once the indication for an operative treatment has been made, arthroscopy is performed in a beach chair position. Regarding the indications, please see below.

Two portals are used to assess glenoid bone loss: the posterior portal for the camera (Figs. 18.5 and 18.6) and the rotator interval portal for instrumentation. Firstly, a diagnostic arthroscopy is

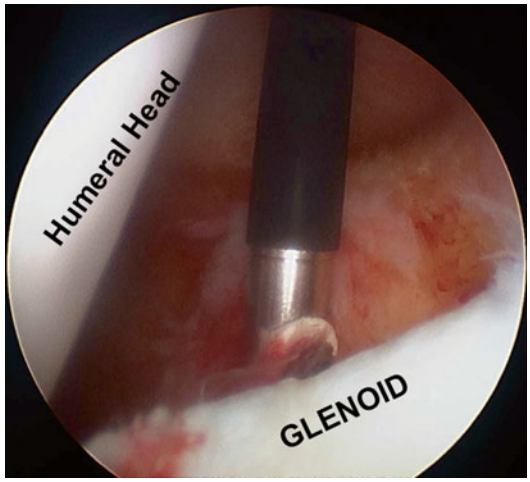

Fig. 18.5 Posterior view on the anterior glenoid. The lesion seems misleading and a soft tissue repair might be performed (same patient as in the Figures showing the CTs)

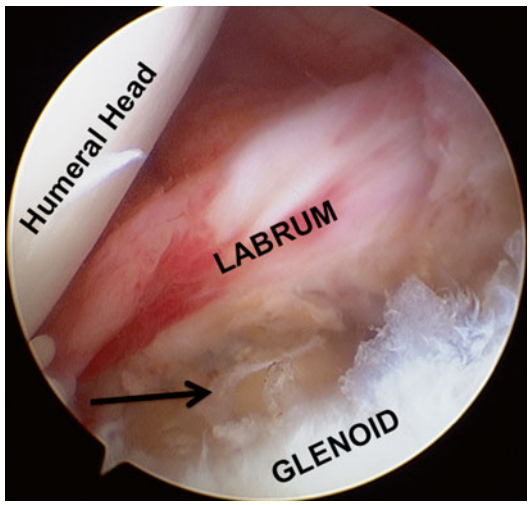

Fig. 18.6 Similar situation as in Fig. 18.5 with a posterior view. The big bony Bankart (same patient as in X-rays) is marked with a *black arrow*. The size can easily be underestimated intraoperatively

performed for concomitant pathologies, such as labral detachment, SLAP, biceps, rotator cuff, Hill-Sachs, HAGL (humeral avulsion of the glenohumeral ligament), and posterior labral lesions. The glenoid is now viewed with the best "en face" view possible. The anterior sleeve is checked for a possible ALPSA (anterior labral periosteal sleeve avulsion) lesion [25, 26]. The presence of an "inverted pear" is noted suggesting a glenoid width loss of at least 25–27 % [20]. A probe is inserted in the rotator interval portal and the percentage of glenoid osseous is measured at the height of the bare spot as proposed by Burkart et al. [4]. For better estimation of the lesion, the camera is inserted into the rotator interval portal (Fig. 18.7) using a switching stick.

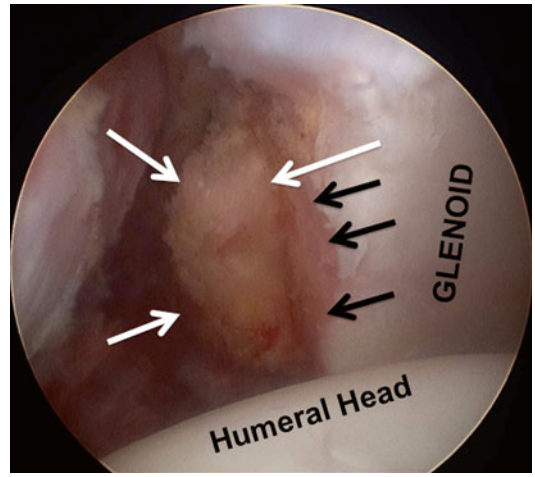

Fig. 18.7 The rotator interval view and the debridement reveals the medialized healed bony Bankart lesion (*white arrows*). This is the same patient as in the figures showing the CTs. The *black arrows* show the glenoid osseous defect

18.6 Treatment Options

Treatment algorithms have traditionally included a period of nonoperative management in all patients; however, young athletic patients may often benefit from early operative treatment [7].

The indication for an operative approach should be based on multiple factors arising from the history of the patient, the clinical examination, the radiographic evaluation, and the arthroscopic findings.

18.6.1 Nonoperative Treatment

After a thorough history, clinical examination, and appropriate imaging (3D CT) indicating a small bony fragment in an anatomical position, nonoperative treatment can be tried with the arm

in an internally rotated position in a sling. A recent meta-analysis has shown no benefit of the arm in external rotation [40]. In the majority of the cases, in the presence of a bony fragment, operative treatment is the treatment of choice after a traumatic anterior shoulder dislocation.

18.6.2 Operative Treatment

Both open and arthroscopic surgical approaches have been well described, with recent studies of arthroscopic soft tissue techniques reporting results equal to those of the more traditional open techniques [5].

The double-row suture technique is a new concept for arthroscopic treatment of bony Bankart lesion in shoulder instability. It presents a new and reproducible technique for arthroscopic fixation of bony Bankart fragments with suture anchors. This technique creates double-mattress sutures, which compress the fragment against its bone bed and restores better bony anatomy of the anterior glenoid rim with stable and non-tilting fixation that may improve healing [45]. Lafosse et al. described the Cassiopeia technique for creating such a double row with suture anchors [16]. This technique creates a W-shaped configuration with a strong tissue grip as the main advantage.

Although small bony lesions may be relatively rare compared with soft tissue pathology, they constitute a critically important entity in the management of shoulder instability. Smaller bony lesions may be amenable to arthroscopic treatment, but larger lesions often require open surgery to prevent recurrent instability [5]. There is evidence from multiple studies, although only level of evidence III–IV, that arthroscopic soft tissue repair for small glenoid bony fragments in an acute setting is associated with a successful treatment [5, 28, 29, 32]. Park et al. showed in a CT follow-up study that small bony Bankart fragments survived without resorption until 1 year postoperatively and that this soft tissue reattachment could survive [27]. The possibility of a Bankart repair combined with a so-called remplissage (filling up a Hill-Sachs defect with the help of the rotator cuff) is not discussed in this section as it is discussed in the previous chapter.

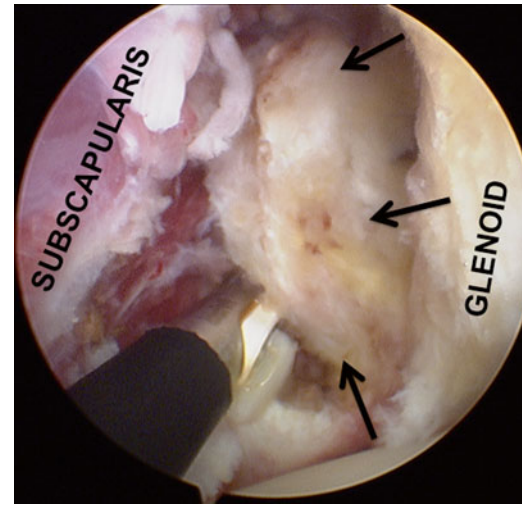

Fig. 18.8 (Same patient as X-ray and Fig. 18.6) Only after debridement can the real size of the fragment be unveiled (*black arrows*)

For bigger fragments and small Bankart fragments in chronic cases (>3 weeks post-trauma) as seen in Figs. 18.7 and 18.8, outcomes for soft tissue repairs were less favorable and associated with failure [5, 6, 24, 28]. This might be due to poor soft tissue quality or associated lesions that were not addressed during the repair (ALPSA or HAGL lesion) as those are linked with poorer outcomes [19, 26, 31].

Kim et al. published a study about treatment guidelines for arthroscopic repair according to the size of bony Bankart lesions of less than 25 % of the glenoid width [15]. For small lesions (<12.5 %), capsulolabral repair using suture anchors without excision of the bony fragment was performed. For medium lesions (12.5–25 %), anatomic reduction and fixation using suture anchors was performed, and the adequacy of reduction was assessed by CT postoperatively. The visual analog scale (VAS) for pain score and modified Rowe score for bony Bankart repair were compared and the postoperative recurrence rate investigated. They concluded that in small bony Bankart lesions, restoration of capsulolabral soft tissue tension alone was enough. However, in medium lesions, the osseous architecture of the glenoid should be reconstructed for more functional improvement and less pain [15].

A recent biomechanical study showed that 38–49 % of the contribution to stability was due to bony reconstruction of the glenoid area [43]. This is why several authors prefer an osseous glenoid reconstruction using the Latarjet, Bristow, or free bone graft technique [18, 21, 23, 36, 38, 39, 42, 44]. In the last 5 years, the all-arthroscopic techniques for bony augmentations such as the Latarjet or Bristow technique have been constantly evolving mostly out of France (Lafosse et al., Boileau et al., Figs. 18.9, 18.10, and 18.11) [3, 18]. These techniques combine the advantages of a bony augmentation and minimally invasive arthroscopy [17].

Rare indications are glenoid fractures with a single fragment after shoulder dislocations. In these circumstances, and if the fracture fragment is big enough, an acute reduction of the fragment using cannulated screws can be achieved [14, 28].

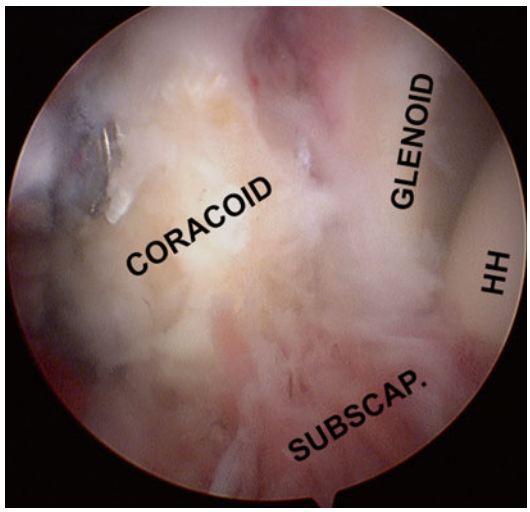

Fig. 18.9 This figure depicts an anterior view of the coracoid process attached to the glenoid to create a bony augmentation of the anterior glenoid rim. Also, the subscapularis tendon can be seen that, together with the conjoint tendon (*left-hand lower corner*), creates the sling effect

18.7 Authors' Preferred Treatment Option

We propose the following treatment algorithm (Fig. 18.12).

We perform an arthroscopic Bankart repair in the following cases:

- Isolated Bankart lesions with an intact labral ring
- Good soft tissue quality
- Absence of associated lesions, e.g., HAGL, ALPSA
- Absence of significant or non-reconstructable bony defect of the glenoid and/or humeral head (engaging Hill-Sachs)

If those soft tissue lesions mentioned above are present and the glenoid fragment is big enough to be seen, more than just a mere soft tissue repair needs to be performed. So, for example, a small bony defect but associated with a ligament tear should have a bone block procedure performed.

We thus perform a bony augmentation of the glenoid. Our preferred technique is the fully arthroscopic Latarjet procedure as first published

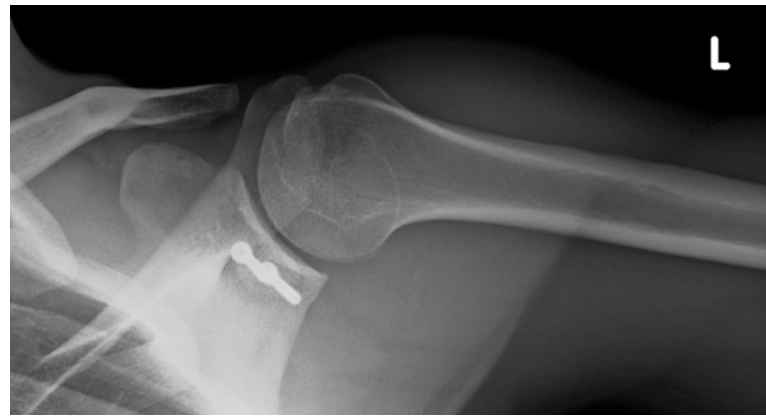

Fig. 18.10 Although this is not a standard X-ray taken at our clinic, this picture nicely shows the screw positioning parallel to the glenoid 6 months postoperatively (same patient as in Figs. 18.4, 18.5, 18.7, and 18.11)

by the senior author in 2007 [18]. It was associated with favorable outcomes. In our 6-year mean follow-up that will be published shortly, we found a recurrence rate of 1.6 % with a mean aggregate WOSI index of 9.4 representing a high score. This technique has developed over the years and the update is about to be published this year by the authors of this chapter [33].

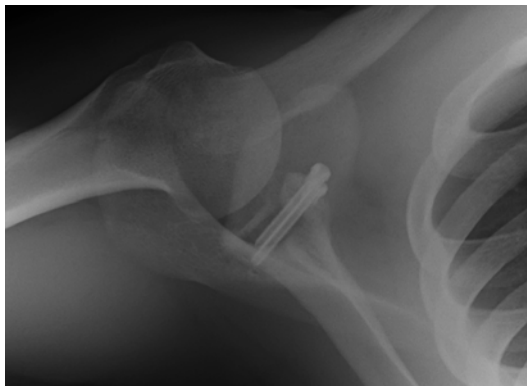

Fig. 18.11 This axillary view shows the bony augmentation of the anterior part of the glenoid. Also, the parallel nature of the screws to the glenoid can be seen 6 months postoperatively (same patient as in Figs. 18.4, 18.5, 18.7, and 18.10)

18.8 Rehabilitation

For Bankart repairs, the shoulder is immobilized in internal rotation for 6 weeks. External rotation does not create any advantage as recently shown [40]. Afterwards, a range of motion (ROM) exercises are begun and, depending on patient's progress, strengthening exercises are initiated at week 8. Return to sport is usually at about 3–6 months postoperatively depending on the kind of sports. In a recent study, we found competitive athletes return faster to their preoperative sports and level compared to recreational athletes [10]. We also found male athletes return faster than female athletes. These two facts might be due to the more readily access to physiotherapy for elite athletes and males.

For osseous reconstructions, rehabilitation can be significantly accelerated assuming the construct is stable having used screw fixation. A sling is worn for comfort purposes only for approx. 5 days. Afterwards, ROM exercises are initiated. After 6 weeks, patients usually perform their first push-up in the outpatient clinic. Complete osseous consolidation can be seen at 3 months postoperatively. Return to sports is usually constituted at 2–3 months postoperatively.

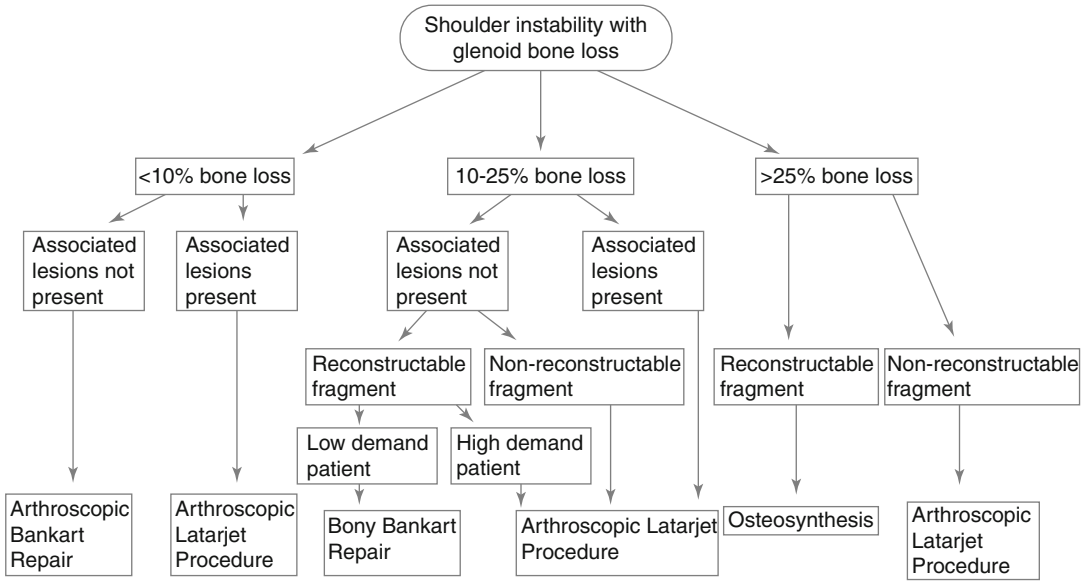

Fig. 18.12 Treatment algorithm for bony Bankart lesions according to size of fragment

18.9 Advantages, Pitfalls, Complications, and Experience of Treating Athletes

18.9.1 Advantages

Advantages of the all-arthroscopic Latarjet procedure are the triple-blocking effect [43] combined with the advantage of arthroscopic surgery with reduced scarring and improved cosmesis. The triple-blocking effect includes the newly formed labrum as an anterior bumper, the bony reconstruction of the glenoid rim, and the dynamic stability provided by the subscapularis/conjoint tendon sling effect. If we have any doubt about the stability of a possible Bankart repair, an arthroscopic Latarjet is performed. The patients are advised accordingly during the preoperative outpatient clinic visit.

18.9.2 Pitfalls and Complications

There are, however, several pitfalls of this surgery as it is not a mere arthroscopic surgery but more an endoscopic surgery in the front of the shoulder. The all-arthroscopic Latarjet technique should only be performed by skilled arthroscopists aware of the plexus anatomy. According to Lafosse's recent report, there are several pitfalls that can be encountered [33]. Firstly, correct placement of the anteromedial portal for graft placement can be difficult, but this can be learned by experience. Due to its position anterior to the pectoralis minor, the plexus is not in danger. Secondly, a proud graft can be avoided by viewing the graft placement from several portals as the 30° optic of the arthroscope introduces a slight distortion. Thirdly, the subscapularis split is the most crucial step in this 10-stage procedure as the axillary nerve is very close. To avoid injury to the axillary nerve, it should be searched for and visualized throughout the split. A correct split ensures an accurate positioning of the coracoid graft on the glenoid. Fourthly, the musculocutaneous nerve, lying medial to the conjoint tendon and inferior to the pectoralis minor, should be respected.

Complications might include axillary nerve damage and non-/malunion of the graft as being the most important besides infection which is rarely encountered.

18.9.3 Experience with Treating Athletes

The senior author has extensive experience with treating elite athletes. Water skiers, contact sportsmen, and other active professionals, such as dancers, reported very good outcomes with international medals after this procedure. In elite and competitive athletes, an all-arthroscopic Latarjet is the preferred treatment method as Bankart repairs have been shown to have high failure rates increasing after 2 years of follow-up [7, 30].

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19 Traumatic Anterior Shoulder Instability: Part III. Hill–Sachs Lesions

Nobuyuki Yamamoto and Eiji Itoi

19.1 Introduction

The Hill–Sachs lesion is one of the most common findings seen in patients with recurrent anterior dislocation of the shoulder. The prevalence of the Hill–Sachs lesion is very high. Most of these lesions are small to medium in size and do not necessarily require treatment. However, we may encounter a large Hill–Sachs lesion, which is known to be a risk factor of postoperative recurrence. A large Hill–Sachs lesion which engages with the glenoid rim is called an "engaging Hill–Sachs lesion," which needs to be treated [2]. Which size of the Hill–Sachs lesion should be treated? We have proposed to evaluate the risk of engagement using a concept of "glenoid track." In this chapter, we describe how to evaluate the risk of engagement with use of the glenoid track and our treatment strategy to the bipolar lesion of the glenoid and the humeral head.

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19.2 Pathoanatomy and Biomechanics

Regarding the location of the Hill–Sachs lesion, Saito et al. [9] determined it using computed tomography images of 35 shoulders with recurrent anterior dislocation. They concluded that the Hill–Sachs lesion exists in the area between 0 and 24 mm from the top of the humeral head. Generally, it is believed that a Hill–Sachs lesion is created when the humeral head is compressed against the glenoid rim with a force generated by the rotator cuff muscles. The question is how much force is necessary to create a Hill–Sachs lesion. We performed a simple experiment to determine the amount of compression force when a Hill–Sachs lesion was created by the glenoid rim (unpublished data) (Fig. 19.1a, b). From this experiment, it was demonstrated that the maximum compression force was 946 N (96.5 kg), which is probably greater than the body weight of the patient.

We clarified, in a biomechanical study using fresh cadavers, which Hill–Sachs lesion is risky by determining the location of the glenoid on the humeral head with the arm elevating along the posterior end range of motion [12]. As the arm was elevated with maximum external rotation and horizontal extension, the contact area of the glenoid shifted from the inferomedial to the superolateral portion of the posterior aspect of the humeral head, creating a zone of contact. We defined this contact zone as the "glenoid track" (Fig. 19.2). According to the measurements in

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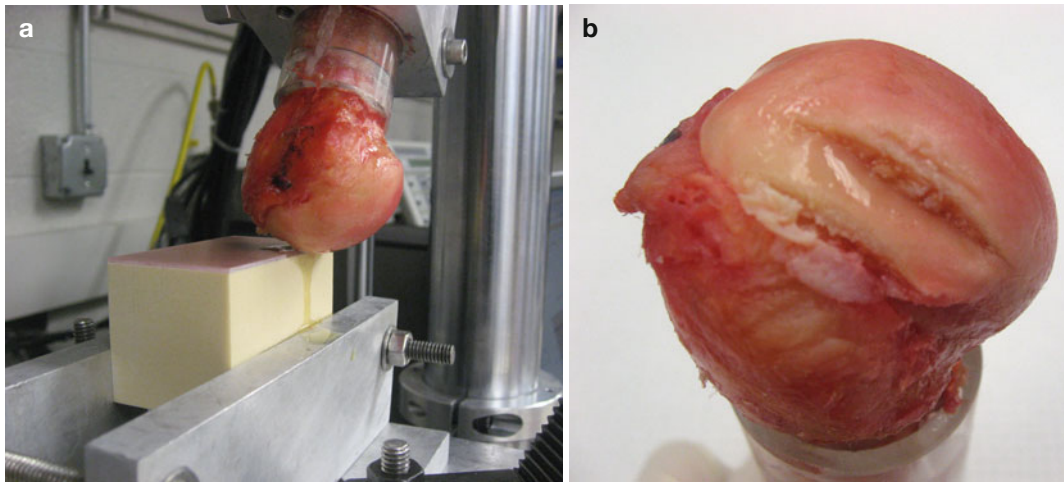

Fig. 19.1 (**a**) Photograph of the experiment creating a Hill–Sachs lesion. The humeral head was compressed to the square sawbone simulating the glenoid. (**b**) A created Hill–Sachs lesion. The Hill–Sachs lesion was artificially created on the humeral head by compressing the sawbone

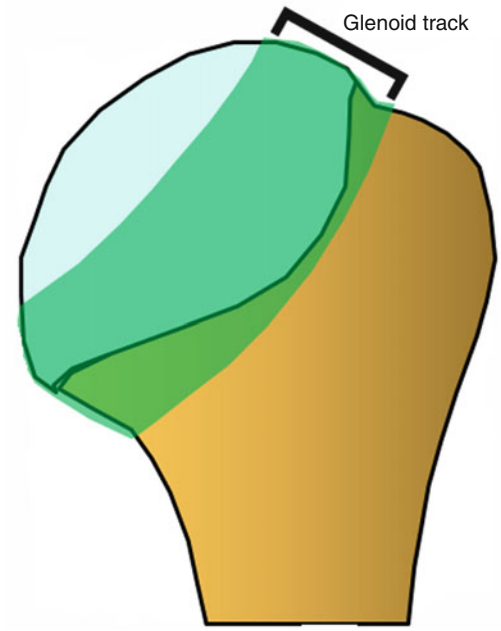

Fig. 19.2 Glenoid track. As the arm was elevated with maximum external rotation and horizontal extension, the contact area of the glenoid shifted, creating a zone of contact. We defined this contact zone as the "glenoid track"

cadaveric shoulders, the glenoid track width was equal to 84 % of the glenoid width in cadaveric shoulders [12] and 83 % in live shoulders [7]. With use of this glenoid track concept, we have proposed to evaluate the risk of engagement with the glenoid [4]. If a Hill–Sachs lesion is located more medially over the medial margin of the glenoid track, such a lesion needs to be treated. On the other hand, if a Hill–Sachs lesion stays on the glenoid track, there is no risk of engagement between the Hill–Sachs lesion and the anterior rim of the glenoid. Such a lesion needs no treatment.

Engagement between a Hill–Sachs lesion and the glenoid rim occurs in 100 % of the cases theoretically because the Hill–Sachs lesion is the result of engagement. This means both the Hill–Sachs lesion and the glenoid are responsible for engagement, not just one or the other. In fact, the engagement is known to be observed more frequently if there is a large glenoid defect [13]. Therefore, when we consider the critical size of the Hill–Sachs lesion, we also have to consider the bony defect of the glenoid at the same time. Our new concept, the "glenoid track," enables us to take both lesions into consideration. If there is a glenoid defect, the defect width should be subtracted from 83 % of the glenoid width (Fig. 19.3).

Previously, we referred to the lesion as an "engaging" or "nonengaging" Hill–Sachs lesion. However, this terminology is confusing because a lesion that engages before the Bankart repair usually becomes a nonengaging lesion after the Bankart repair. Only 7 % of all the Hill–Sachs lesions remained as an engaging lesion after the Bankart repair [8]. In order to avoid this confusion, Di Giacomo et al. proposed a new terminology "on-track" and "off-track" lesion [3]. A Hill–Sachs

lesion that stays on the glenoid track, which means there is no risk of engagement after the Bankart repair, is an "on-track" lesion, whereas a Hill–Sachs lesion that extends out of the glenoid track, which means there is a risk of engagement even after the Bankart repair, is an "off-track" lesion.

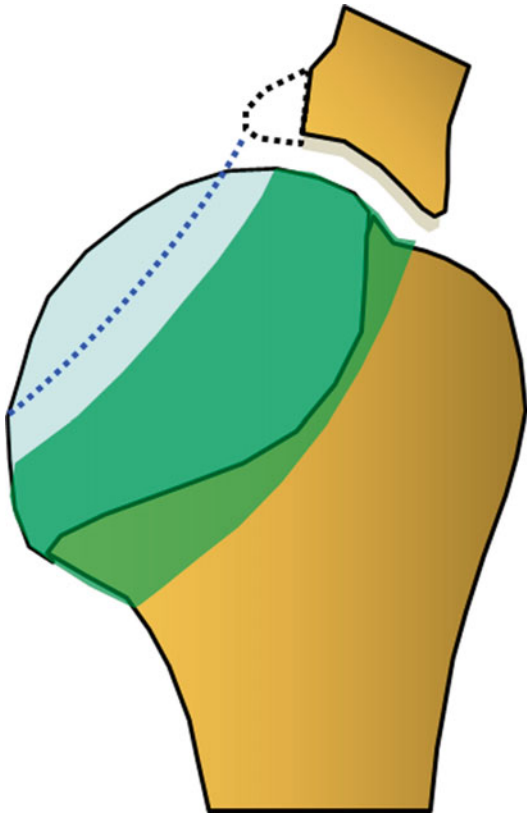

Fig. 19.3 Glenoid track when there is a glenoid defect. If there is a glenoid defect (*dotted line*), the defect width should be subtracted from 83 % of the glenoid width

19.3 Clinical Presentation

Case 1

A 51-year-old female sustained a traumatic dislocation of her left shoulder after a fall down the stairs. She reported 15 subsequent episodes of dislocation since then. She had a small glenoid defect (3 mm in width) and a large Hill–Sachs lesion (86 % of the glenoid width), which was located off the glenoid track (off-track lesion) (Fig. 19.4a, b). We performed arthroscopic remplissage procedure combined with the Bankart repair. One and a half years after the surgery (Fig. 19.5), she has had no re-dislocation and enjoys playing golf although she has a mild limitation in the range of external rotation (20° each in adduction and in abduction).

Case 2

An 18-year-old, right-hand-dominant male initially dislocated his left shoulder during a rugby game. He reported three subsequent episodes of dislocation during rugby games. He had a large glenoid defect (23 % of the glenoid width) and a large Hill–Sachs lesion (80 % of the glenoid width), which was within the glenoid track but very close to the medial line of the glenoid track (Fig. 19.6a, b). We performed the Latarjet procedure for this patient.

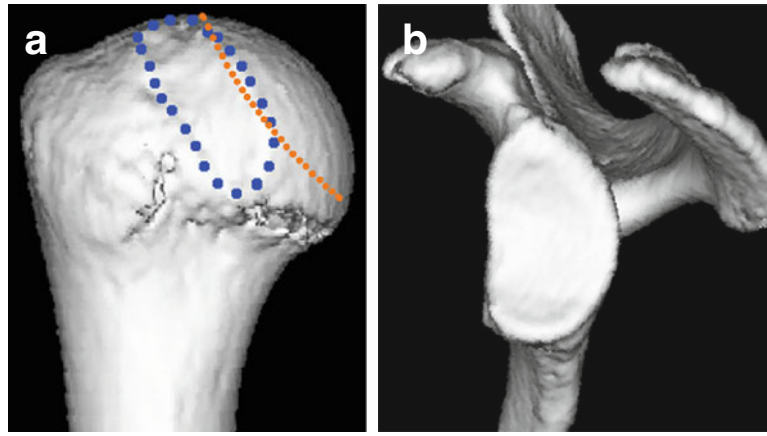

Fig. 19.4 (**a**) 3D CT image of the humeral head. This Hill–Sachs lesion (*blue dotted line*) was located more medially over the glenoid track (*orange dotted line*). (**b**) 3D-CT image of the glenoid. The size of the glenoid bony defect was 3 mm in width, a small defect

Two years after the surgery (Fig. 19.7), he had neither restriction of the range of shoulder motion, recurrence, nor anterior apprehension. He could return 100 % to his previous level.

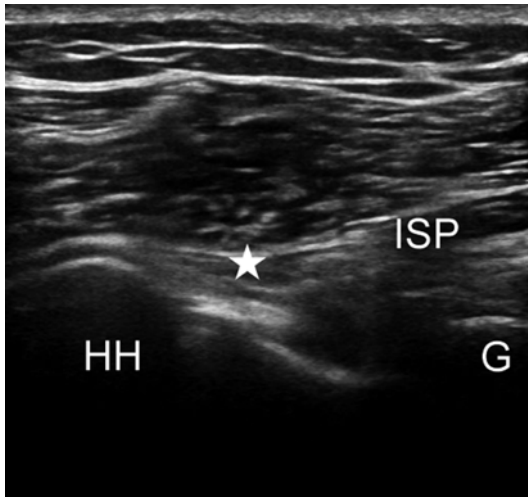

Fig. 19.5 Posterior view of the postoperative ultrasonographic image. The infraspinatus (*ISP*) tendon (★) is fixed into the humeral head (*HH*) defect. *G* glenoid rim

19.4 Essential Radiology

19.4.1 X-ray

We advocate three views: (1) an AP view to evaluate the bony fragment of the glenoid, (2) an axillary view (glenoid bony defect or fracture or a Hill–Sachs lesion), and (3) a Stryker notch view (a Hill–Sachs lesion). However, unfortunately, x-ray is not reliable for the accurate assessment of the size of the Hill–Sachs lesion.

19.4.2 CT (3D-CT)

This gives a lot of information on bony lesions, but a small Hill–Sachs lesion and an erosion-type glenoid defect may not always be recognized. In such cases, three dimensionally reconstructed computed tomography (3D-CT) with the humeral head eliminated is useful and gives excellent information. Sagittal and axial CT images or 3D-CT images of bilateral shoulders are useful

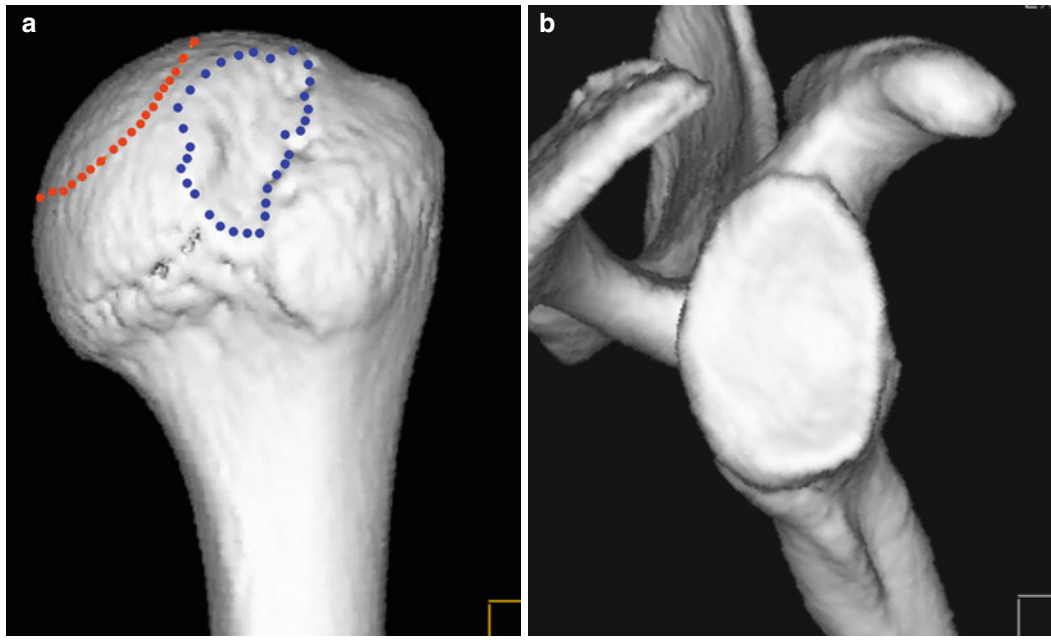

Fig. 19.6 (**a**) A large Hill–Sachs lesion. There was a large Hill–Sachs lesion (*blue dotted line*), which was within the glenoid track (*orange dotted line*) but very close to the medial margin of the glenoid track. (**b**) Large glenoid track. The size of the glenoid bony defect was 4.5 mm in width, which was 23 % of the glenoid width compared to the uninvolved side

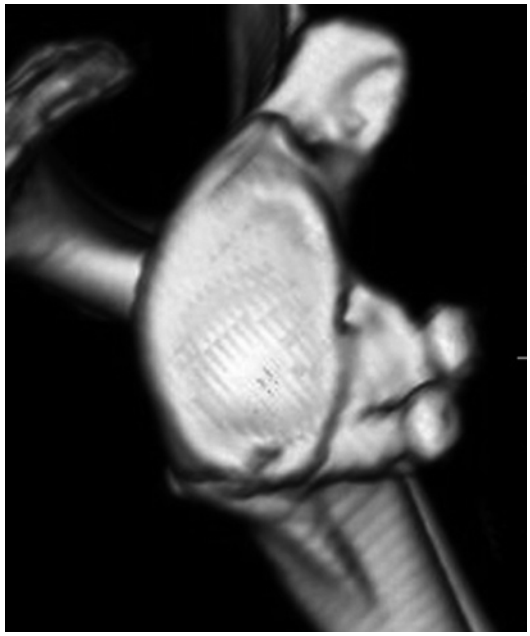

Fig. 19.7 Postoperative 3D CT image. Union of the grafted bone was observed

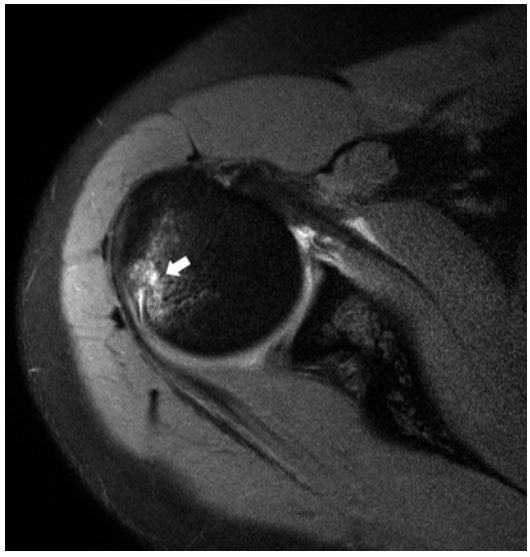

Fig. 19.8 A Hill–Sachs lesion after an initial dislocation. A Hill–Sachs lesion is observed as the concavity with high intensity on the posterior humeral head (*arrow*)

and reproducible in measuring the size of the Hill–Sachs lesion. We routinely perform 3D-CT and magnetic resonance arthrography in patients with recurrent anterior glenohumeral instability.

19.4.3 MRI

In axial MR images of first dislocators, the concavity with high intensity on the posterior humeral head is observed, which means the compression fracture of the humeral head (Fig. 19.8).

19.4.4 Ultrasonography

A Hill–Sachs lesion can be easily detected from the posterior approach with the arm flexed slightly (Fig. 19.9).

19.5 Disease-Specific Clinical and Arthroscopic Pathology

An "engaging Hill–Sachs lesion" was first reported by Burkhart and De Beer. The prevalence of this engaging Hill–Sachs lesion was reported to be 1.5 %. Kurokawa et al. [5] reported that in their series of 100 shoulders with recurrent anterior dislocation, 94 shoulders had a Hill–Sachs lesion, and 7 of them (7.4 %) were defined as an engaging Hill–Sachs lesion based on the glenoid track. Interestingly, the value of the prevalence was reported by Park et al. [8] who evaluated the engagement after an arthroscopic Bankart repair. According to their report, 70 out of 983 cases (7 %) showed an engagement after the Bankart repair. It is clear that the prevalence of the engaging Hill–Sachs lesion is low. However, the prevalence of the engaging Hill–Sachs lesion treated by remplissage is surprisingly high: 27–43 %. This is probably due to the misunderstanding of the engaging Hill–Sachs lesion. Engagement should be assessed after the Bankart repair. However, these authors performed dynamic assessment of the Hill–Sachs lesion before the Bankart repair. Evolving of the concept from "engaging/nonengaging" lesion to "on-track/off-track" lesion would be very helpful to avoid this misinterpretation of bipolar lesions.

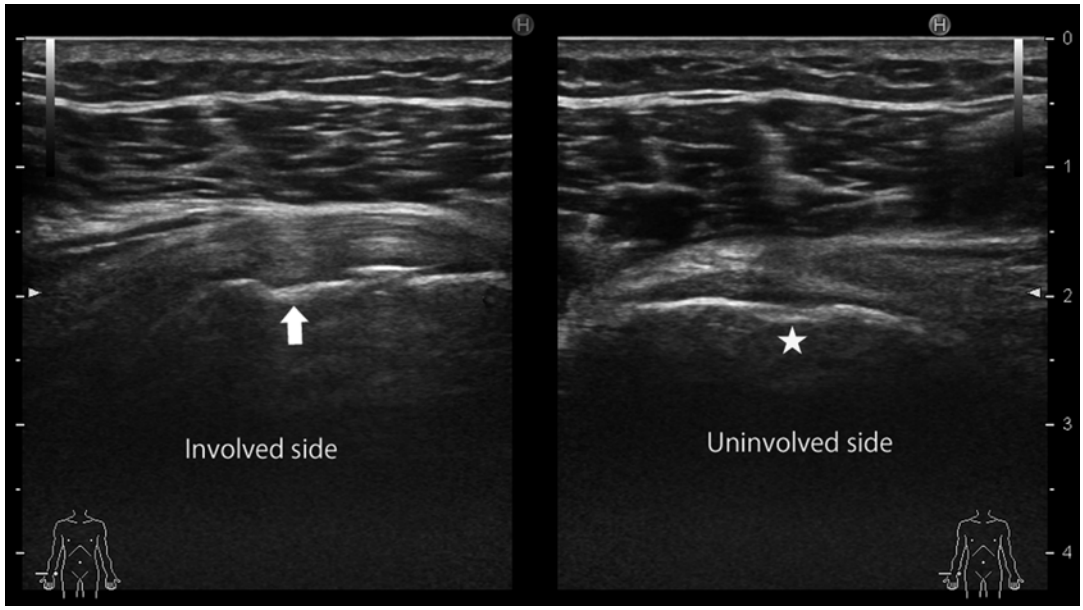

Fig. 19.9 Ultrasonographic image of the Hill–Sachs lesion of bilateral shoulders. A Hill–Sachs lesion (*arrow*) which needs to be differentiated from the bare area (*asterisk*) between the articular surface of the humeral head and the greater tuberosity

19.6 Treatment Options

Two types of surgical procedure are available, by which engagement can be avoided between the Hill–Sachs lesion and the anterior glenoid rim: (1) reducing the range of motion in external rotation and (2) filling the humeral head defect. The former includes anterior soft tissue shortening or rotational osteotomy of the humerus. The latter includes filling the Hill–Sachs lesion with a bone graft or the soft tissue, or percutaneous transhumeral head plasty. Remplissage procedure is one of the latter procedures.

19.7 Author's Preferred Treatment (Table 19.1)

In the case of an off-track Hill–Sachs lesion, the treatment depends on the size of the glenoid defect. If the glenoid defect is less than 25 %, no treatment is required for this glenoid defect [13]. The off-track Hill–Sachs lesion would be successfully treated by arthroscopic remplissage. The Latarjet procedure may be another option because the Latarjet procedure makes the glenoid track wider and converts an off-track Hill–Sachs lesion to an on-track lesion. If the glenoid defect is more than 25 %, treatment of the glenoid defect is definitely required. We recommend the coracoid transfer to the glenoid defect, which not only reconstructs the glenoid concavity but also converts an off-track lesion to an on-track lesion.

There are several modifications regarding the Latarjet procedure. Some repair the capsule to the remnant coracoacromial ligament attached to the coracoid process instead of repairing a Bankart lesion. Others repair a Bankart lesion to

Table 19.1 Our treatment strategy

Glenoid defect (%)[a]	Hill–Sachs lesion	Treatment	Our series (%)[b]
<25	On-track	ABR	93
>25	On-track	Latarjet	0
<25	Off-track	ABR + remplissage (or Latarjet)	5
>25	Off-track	Latarjet	2

ABR arthroscopic Bankart repair
[a]Percentage of the glenoid width
[b]Kurokawa et al. *JSES* 2013 (Ref. [5])

the native glenoid using suture anchors. Recently, some surgeons perform the Latarjet procedure with all-arthroscopic technique. Basically, we follow the technique described by Walch [10]. The subscapularis muscle/tendon is split at the superior two-third junction. Two screws are used to fix the coracoid process. The only difference is we repair a Bankart lesion to the native glenoid using suture anchors instead of suturing the coracoacromial ligament to the capsular flap.

19.8 Rehabilitation

The arm is immobilized in adduction and internal rotation for 3 weeks, followed by pendulum exercises (see Sect. 31.2.1.1). After 3 weeks, the pendulum exercises are begun. Gradually, the arm is free to move for activities of daily living, and active-assisted shoulder range of motion exercises (see Sects. 31.2.1.3 to 31.2.1.17) are initiated. Muscle-strengthening exercises (see Sects. 32.1.2.1 to 32.1.2.4) are started after 6–8 weeks if bone union is achieved. Jogging and running are authorized at 2 months. Full participation in sports is permitted after 4 months if the muscle strength returns to greater than 90 % of the contralateral shoulder.

19.9 Advantages and Pitfalls, Complications

The Latarjet procedure has an efficient stabilizing mechanism. Yamamoto et al. [14], in a biomechanical study using fresh cadavers, demonstrated that the main stabilizing mechanism was the sling effect produced by the subscapularis and conjoint tendons, at both the end-range and mid-range arm positions. The remaining stability arose from suturing of the coracoacromial ligament to the capsular flap at the end-range position and from glenoid cavity reconstruction at the mid-range position. Thus, it was proved that the Latarjet procedure has a reasonable stabilizing mechanism, and it can be performed for patients at high risk of recurrence, such as athletes who participate in collision sports. Regarding the pitfalls and complications of the Latarjet procedure, we need to be careful in positioning the grafted bone. The precise positioning of the coracoid graft is crucial for prevention of early osteoarthritis. Recent reports [10] have shown that postoperative arthritis can be avoided by appropriate positioning of a coracoid bone graft. It should be flush with the glenoid articular surface, never overhang the glenoid. It is important to check the orientation of the articular surface and direct the drill parallel to this plane. Also, the bone block is secured bicortically by two screws without a washer. These technical details help to obviate complications such as pseudarthrosis, migration, and fracture of the coracoid.

Arthroscopic remplissage procedure is a commonly performed, effective procedure for a large Hill–Sachs lesion. This surgical technique was first reported by Wolf et al. [11]. Due to its simplicity, arthroscopic remplissage procedure combined with the Bankart repair has become widely used. Many clinical papers have demonstrated its excellent outcome. With the remplissage, the glenoid cannot move over the lesion because it is located extra-articularly: the glenoid has to move around the lesion. We hypothesized that this movement of the glenoid would cause a limited range of motion. Omi et al. [6] performed a cadaveric study to prove this hypothesis. Clinically, Boileau et al. [1] reported that the reduction in external rotation after the remplissage was 8° in adduction and 9° in abduction in their series of 42 patients. Considering the restriction of range of motion, usage of remplissage to overhead athletes should be avoided.

19.10 Experience in Treatment of Athletes

Arthroscopic Bankart repair has become the gold standard for the treatment of recurrent anterior dislocation of the shoulder. In order to verify the clinical outcome of the Bankart repair, 100 patients with recurrent anterior shoulder dislocation without a large bony defect were retrospectively reviewed [15]. Our data showed that the

recurrence rate of the Bankart repair in the contact athletes was two times higher in the open group and three times higher in the arthroscopic group than in the noncontact athletes. These data show that the Bankart repair alone is not enough when treating athletes in a high-risk group. Thus, we choose the Latarjet procedure for these high-risk patients.

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Posterior Shoulder Instability in the Contact Athlete

20

Eric P. Tannenbaum, Nathan J. Kopydlowski, and Jon K. Sekiya

20.1 Introduction

Shoulder instability is a relatively common condition in the athletic community; however, posterior shoulder instability is a significantly less common entity when compared to anterior instability. The incidence of posterior shoulder instability has been reported to be between 2 and 12 % of all reported cases of instability [1]. However, in the past, this condition was not as well recognized, and as a result these reports may be lower than the actual real incidence. Posterior shoulder instability has become increasingly more recognized within the athletic population and presents as a complex problem for many orthopedic surgeons. The broad term "posterior instability" may be used to describe either a chronic locked posteriorly dislocated shoulder or a shoulder subject to recurrent posterior subluxation (more common) [2].

Typically athletes involved in sports requiring overhand throwing or contact sports are more likely to develop a component of posterior instability due to repetitive anterior-to-posterior forces imposed on the glenohumeral joint. A recent prospective study by Bradley et al. looked at 200 shoulders with isolated posterior shoulder instability and found that 58 % (117/200) of the athletes were involved in contact sports [3]. The most commonly involved contact sports were football (60 %), basketball (21 %), wrestling (12 %), lacrosse (4 %), hockey (2 %), and martial arts (1 %).

It is crucial for the orthopedic surgeon to understand what athletes are at risk for developing this condition and how to properly classify, evaluate, and treat patients presenting with symptoms suspicious for posterior instability. A comprehensive understanding of the complex pathoanatomy of the shoulder, a working knowledge of specific clinical examination techniques, and the utilization of appropriate imaging modalities are necessary to make the correct diagnosis and guide appropriate treatment. This chapter will outline the workup and treatment options for patients presenting with posterior shoulder instability and prepare the surgeon to effectively diagnose and treat athletes presenting with posterior shoulder pathology.

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20.2 Pathoanatomy, Biomechanics, and Classification

The shoulder has the widest range of motion of any joint in the human body, but it is also associated with the highest propensity for instability. The classic model used to describe the glenohumeral joint is a golf ball (humeral head) on a tee (glenoid surface). This model represents the small amount of humeral head surface area that articulates with the glenoid fossa throughout the entire arc of motion. Specifically, just 25–30 % of the humeral head articulates with the glenoid fossa in any one position [4].

The shoulder joint therefore relies on both static (or passive) and dynamic (or active) stabilizers to maintain joint congruency and prevent pathologic glenohumeral translation. The negative intra-articular pressure of the joint creates a vacuum effect that also contributes to shoulder stability. Static stabilization is accomplished through both bony and soft tissue anatomy. The bony morphology of the joint, including the articular conformity, the glenoid and humeral version, the glenoid inclination, the coracoacromial arch, and the glenoid size all contribute to stability. Bony defects such as posterior glenoid erosion, glenoid hypoplasia, excessive glenoid retroversion, or excessive humeral retroversion may decrease the effectiveness of these static stabilizers and thus increase the likelihood of posterior shoulder instability. Owens et al. performed a prospective study involving 714 athletes and found that increased glenoid retroversion was the most significant prospective risk factor for posterior shoulder instability [1].

The soft tissue static stabilizers include the glenoid labrum, the capsule, and the capsular ligaments (i.e., capsuloligamentous structures). Athletes often develop posterior instability as a result of repetitive, sport-specific motions that result in microtraumatic stress to the posterior capsulolabral complex resulting in posterior capsule attenuation and/or posterior labral tears [2, 5]. The labrum is a ring of densely packed fibrocartilage that attaches along the perimeter of the glenoid fossa and assists in stabilizing the joint by functionally increasing the depth, concavity, and surface area of the glenoid. The labrum has been shown to be responsible for approximately 10 % of the glenohumeral stability [6]. Furthermore, the labrum provides a stable fibrocartilaginous anchor for the capsular ligaments, which are thickenings within the shoulder capsule. These ligaments historically consist of the superior glenohumeral ligament, the middle glenohumeral ligament, and the inferior glenohumeral ligament. However, more detailed anatomic studies have further defined an inferior glenohumeral ligament complex made up of an anterior band, posterior band, and an axillary pouch in between [7]. With the arm positioned in adduction, flexion, and internal rotation, the posterior band of the inferior glenohumeral ligament complex and the posterior capsule provide the major constraints against excessive posterior translation [8].

The dynamic stabilizers of the shoulder include the rotator cuff, the deltoid, the long head of the biceps, and the scapular rotators. The subscapularis has been identified as the most important dynamic stabilizer in preventing posterior translation [9]. The scapular rotators work together to establish synchronous motion between the humerus and scapula to conserve joint alignment throughout shoulder range of motion. The rotator cuff, deltoid, and long head of the biceps load the humeral head into the glenoid socket resulting in a concavity-compression effect, thereby providing further dynamic stabilization of the joint. The rotator cuff attaches to portions of the capsule and provides additional support by tensioning the capsule when the muscles are recruited. Furthermore, capsular stretching or shoulder motion is detected by sense receptors within the joint and triggers a proprioceptive muscular response of the rotator cuff muscles. Of course, anatomic disruption of any of the aforementioned stabilizers will increase the risk for developing instability.

Various descriptions and classifications have been used to describe shoulder instability including degree, chronicity, mechanism of injury, direction, and volition [10]. However, no single system exists that effectively guides treatment, predicts outcome, and allows for precise and specific communication among physicians.

Degree simply refers to how far the humeral head translates outside of the normal joint limits

and is typically simplified into two categories of subluxation and complete dislocation. The mechanism of injury commonly associated with a complete posterior shoulder dislocation in a contact athlete is an acute episode of anterior-to-posterior-directed shoulder trauma leading to posterior capsule tears, posterior labral tears (reverse Bankart), rotator cuff tears, or bony disruption (reverse bony Bankart or reverse Hill-Sachs defect). After one episode of traumatic dislocation, patients are at increased risk for recurrent shoulder dislocation, pain, and weakness. On the other hand, the mechanism of injury characteristically associated with recurrent posterior shoulder subluxation is through repetitive cycling of the shoulder resulting in microtrauma to the shoulder often leading to gradual attenuation of the capsuloligamentous structures and eventual capsular redundancy. Athletes participating in contact sports are obviously at risk for developing instability through both mechanisms.

Posterior shoulder instability can further be classified into the following directions: unidirectional (posterior), bidirectional (posteroinferior), or multidirectional (posterior, inferior, and anterior). Unidirectional instability alone is much less common than either bidirectional or multidirectional instability. Lastly, although rare, it is important to recognize a particular subset of patients who voluntarily subluxate their shoulder (i.e., habitual or psychogenic, voluntary shoulder subluxations). These patients often have other underlying mental health issues that must be addressed prior to pursuing any further treatment, and surgery for these patients is generally contraindicated unless these issues have been appropriately addressed.

The lack of consistency in terminology has made it particularly challenging to appropriately diagnose and treat posterior instability. Furthermore, terms such as laxity and instability are often incorrectly used interchangeably. *Laxity* is defined as a loose joint with increased translation of the humeral head, whereas *instability* is defined as laxity combined with the subjective feeling that the head is not stable in the joint (i.e., patient apprehension in the setting of laxity). Therefore, a patient may have laxity without instability as commonly seen in patients with Marfan syndrome or Ehlers-Danlos.

20.3 Clinical Presentation and Essential Physical Exam

As always, it is essential to obtain a thorough history and physical exam in any athlete presenting with complaints of shoulder pain and/or symptoms concerning for instability. The athlete's age, sex, type of sport (contact vs. noncontact; throwing vs. non-throwing), position (i.e., lineman, quarterback, outfielder, center, etc.), dominant versus nondominant arm, and level of competition should all be noted. Athletes participating in contact sports including wrestling, football, basketball, hockey, rugby, and lacrosse should be considered an at-risk population, and a high level of suspicion for posterior instability is necessary in this cohort of patients [11].

Football players have been reported to have the highest incidence of posterior shoulder instability [3]. This is likely due to linemen blocking with their arm being held in the so-called provocative position of 90° of forward flexion, adduction, and internal rotation while an axial load is applied to the shoulder [12]. Kaplan et al. studied shoulder injuries in elite collegiate American football players at the National Football League Combine and discovered that approximately 50 % of players reported a history of some type of shoulder injury [13]. Of these reported injuries, 4 % involved posterior shoulder instability. The patient should also be questioned about any other past injuries or traumatic events involving the affected shoulder.

Patients presenting with posterior shoulder instability differ from patients with anterior instability in that most patients with posterior instability primarily complain of pain with specific motions and report instability only as a secondary concern [5]. The pain is often generalized and varies in location but may be reproduced with the shoulder in the provocative position of 90° of forward flexion, adduction, and internal rotation [12]. Clinically, the sensation of instability is often referred to as "apprehension" because the patient is apprehensive about bringing their arm into the provocative positions that reproduce the uncomfortable sensation of impending dislocation. They may also report clicking and/or catching in the shoulder or posterior joint-line tenderness if they have a concomitant reverse Bankart tear.

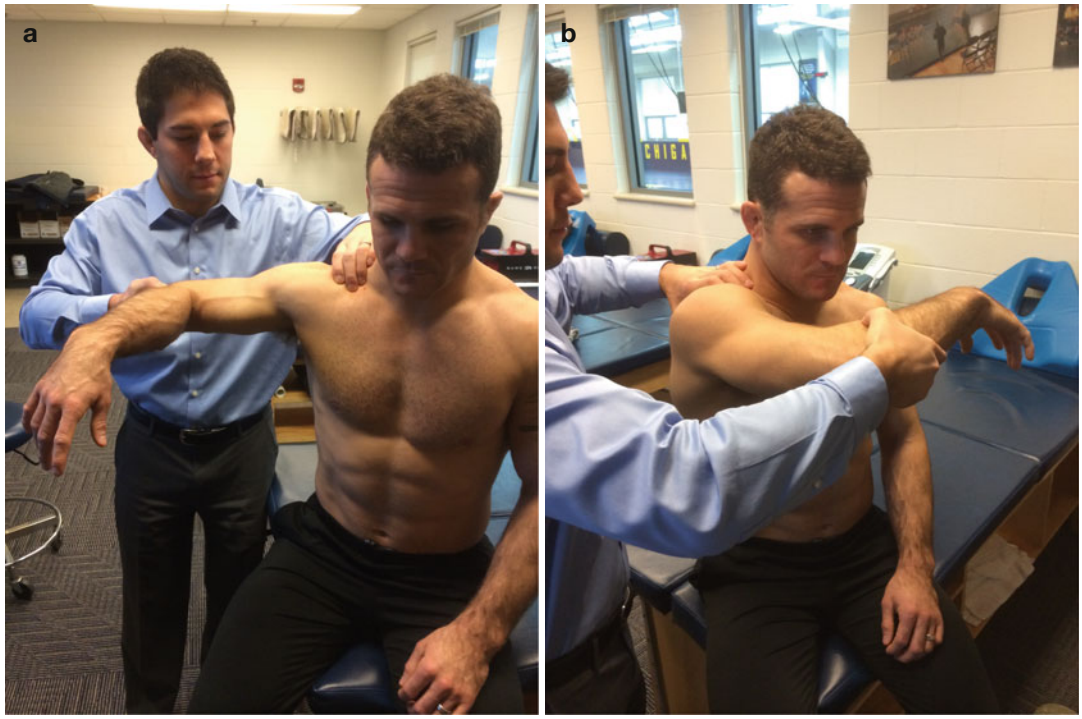

Fig. 20.1 Jerk test: (**a**) With the patient seated, the examiner stabilizes the shoulder girdle by grasping the scapular spine and clavicle with one hand. The other hand holds the patient's flexed elbow and with the arm in 90° of abduction and internal rotation. (**b**) The arm is then moved horizontally across the chest while applying an axial load through the elbow. A sudden clunk or "jerk" often associated with pain occurs in a positive test as the humeral head subluxates posteriorly over the glenoid rim. Maintaining this load, the arm is moved back to its starting position, and a second jerk may be appreciated as the humeral head relocates back into the glenoid fossa

Patients commonly describe instability as intensifying toward the end of long competitions because, over time, strenuous play results in rotator cuff muscle fatigue as well as the other dynamic stabilizers involved in maintaining the concavity-compression effect. This increased instability with play may significantly impact athletic performance as the athlete attempts to compensate for the loss of stability by adjusting his or her shoulder biomechanics. A similar effect is seen with professional baseball pitchers whose shoulders fatigue in the latter innings and therefore must be substituted with a "relief pitcher." Patients may sometimes recall specific events of posterior dislocation where the shoulder "pops out the back" and may have required reduction on the field/court or in the emergency department. As emphasized by Hawkins et al., recurrent posterior shoulder instability involving multiple episodes of posterior shoulder *subluxation* is much more common than multiple episodes of true posterior *dislocation* [2].

A routine physical examination of the shoulder should include looking for shoulder asymmetry, measurement of active and passive ranges of motion, palpation for joint-line tenderness, assessment for impingement, and strength testing. In addition, anterior, inferior, and posterior translation as well as anterior apprehension and generalized ligamentous laxity should be assessed. Inferior laxity may be evaluated by performing the sulcus test, where the examiner distracts the shoulder inferiorly with the patient's arm adducted and in neutral rotation. A positive sulcus sign is demonstrated when a gap forms beneath the acromion and above the humeral head.

In addition to the standard physical examination, multiple techniques have been described to specifically evaluate posterior instability. These techniques include the jerk test, posterior stress test, Kim test, and posterior load and shift test as demonstrated in Figs. 20.1, 20.2, 20.3, and 20.4,

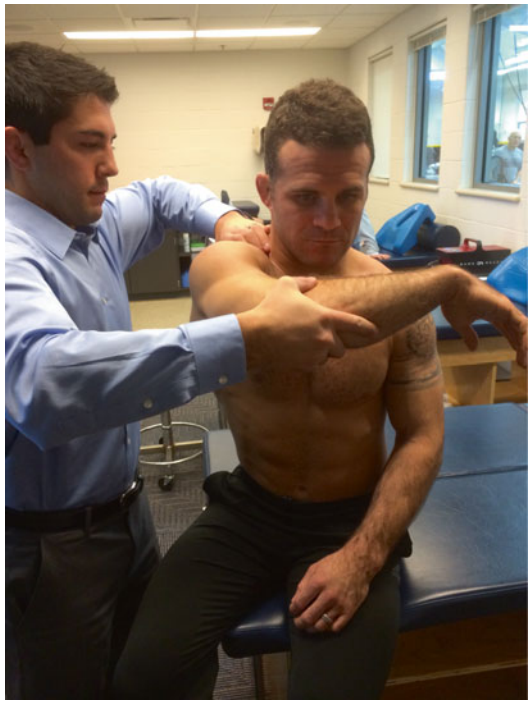

Fig. 20.2 Posterior stress test: With the patient in the seated position, the examiner immobilizes the shoulder by grasping the scapular spine and clavicle with one hand. The other hand is used to apply a posterior force to the arm held in 90° of forward flexion, adduction, and internal rotation. A positive test is determined when subluxation combined with pain/apprehension is present

respectively. A study performed by Kim et al. demonstrated the jerk test to be more sensitive for detecting predominantly posterior labral lesions, while the Kim test was found to be more sensitive for detecting predominantly inferior labral lesions [14]. Combining these two tests resulted in a 97 % sensitivity for detecting a posteroinferior labral lesion.

20.4 Essential Radiology

Athletes presenting with shoulder pain and instability should be sent for a 3-view radiographic series to include an anterior-posterior view, axillary (or West Point) view, and supraspinatus outlet (or apical oblique) view. Radiographs are frequently normal; however, this combination of views is necessary to accurately assess the athlete's shoulder for bony pathology including reverse Hill-Sachs lesions (Fig. 20.5), reverse bony Bankart avulsions (Fig. 20.6), glenoid rim abnormalities, glenoid dysplasia, or lesser tuberosity fractures that are often considered pathognomonic for previous posterior shoulder dislocations. Some studies have described stress radiography, where posterior loads are applied to the shoulder to assess posterior translation, but this is not typically performed.

Although plain radiographs are a good starting point and may be helpful to rule out any obvious bony defects, they are frequently negative, and magnetic resonance imaging (MRI) and magnetic resonance arthrography (MRA) provided much more useful information regarding the integrity of soft tissue structures that are more typically involved in the pathogenesis. Specifically, MRI provides visualization of posterior capsulolabral structures, biceps anchor, rotator cuff, and the rotator interval (Fig. 20.7). An MRA is helpful in detecting a Kim lesion, or a marginal labral tear, that may be missed with standard MRI.

If a bony abnormality is suspected based on plain films, computer tomography (CT) may be utilized to better evaluate the defect. CT is the best imaging modality available for evaluating the size of reverse Hill-Sachs lesions or glenoid pathology as it allows the surgeon to determine what percentage of the humeral or glenoid articular cartilage is involved.

Dynamic ultrasound is another modality that has been described in the literature as a fast and inexpensive tool to objectively quantify glenohumeral laxity; however, it is not commonly used in the clinical setting due to the difficulty involved in interpreting the ultrasound imaging by most orthopedic surgeons [15].

20.5 Treatment Options

An initial course of conservative management involving at least 6 months of activity modification (or rest) with physical therapy focused on strengthening the dynamic muscular stabilizers is generally recommended, although high-profile

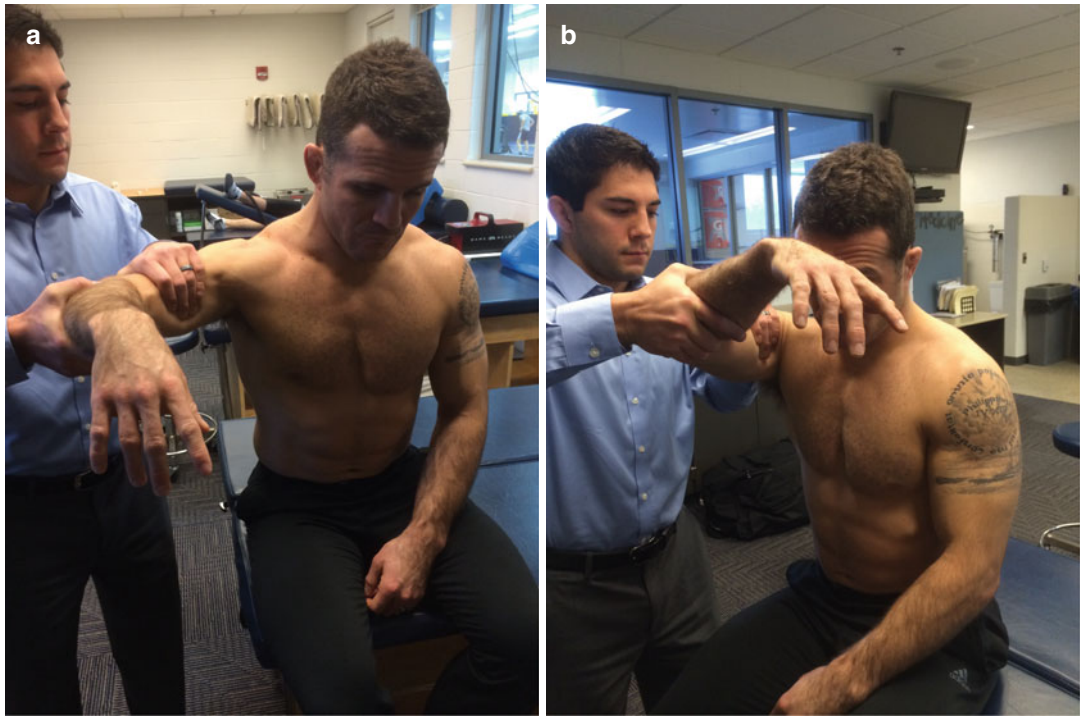

Fig. 20.3 Kim test: (**a**) The patient is seated and the arm is positioned in 90° of abduction. (**b**) The arm is then passively elevated an additional 45° while the examiner applies an axial load through the elbow and a downward and posteriorly directed load through the proximal arm. Posterior subluxation with pain signifies a positive test

athletes are often treated more aggressively to limit time away from competition. Currently, there is no clear evidence in the literature supporting the use of bracing or taping to help stabilize the shoulder. Conservative treatment has been shown to have successful outcomes in certain patients with posterior instability [2, 5, 16, 17]. In particular, athletes who have developed posterior instability from repetitive microtrauma as opposed to a single macrotraumatic event generally tend to do better with conservative management. Burkhead and Rockwood demonstrated success rates of approximately 80 % in patients with a history of atraumatic instability, compared to a success rate of just 16 % in patients who experienced posterior instability after a traumatic event [17].

Surgical management is generally considered after conservative management has failed but can usually be delayed until the off-season. Surgical techniques can be grouped into two broad categories: correction of soft tissue defects (more common) and correction of bony defects. Of course, some athletes may require correction of both soft tissue and osseous problems. Procedures involving correction of bony pathology are more commonly performed open, whereas procedures requiring soft tissue repair are generally performed arthroscopically. However, recent literature has also described arthroscopic techniques for treatment of bony defects including reverse Hill-Sachs lesions [18].

The most common soft tissue pathology associated with posterior instability is posterior capsular attenuation/redundancy with or without a reverse Bankart tear. These lesions routinely occur together. The redundancy of the posterior capsule is typically corrected arthroscopically via a vertical posterior capsular shift (i.e., capsular plication), and concomitant reverse Bankart lesions are corrected arthroscopically with suture anchors or tack fixation. Savoie et al. looked at arthroscopic management of posterior shoulder instability and found isolated reverse Bankart

lesions present in 51 % of the shoulders, a stretched posterior capsule in 67 % of shoulders, and a combination of the two problems in 16 % of all shoulders [19]. They also reported the presence of several other accompanying lesions including anterior-superior labral tears and SLAP tears (20 %), superior glenohumeral ligament injury (7 %), rotator interval damage (61 %), middle glenohumeral ligament injury (38 %), anterior glenohumeral ligament injury (27 %), and an enlarged axillary pouch (20 %).

Although bony lesions including reverse Hill-Sachs and posterior glenoid defects are less common and are generally associated with macrotraumatic events or frank posterior dislocations, it is worth mentioning the commonly described treatment options for these problems. Surgical treatment is generally indicated in

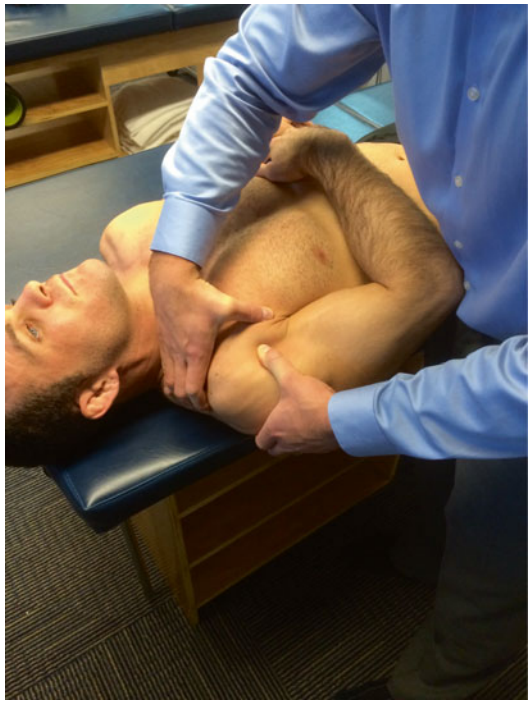

Fig. 20.4 Posterior load and shift test (or posterior drawer test): With the patient in the supine position, the shoulder girdle is stabilized with one hand, while the other hand holds the proximal arm and loads the humeral head medially into the center of the glenoid to confirm neutral joint position. A posterior stress is then applied to the arm to evaluate excessive posterior translation

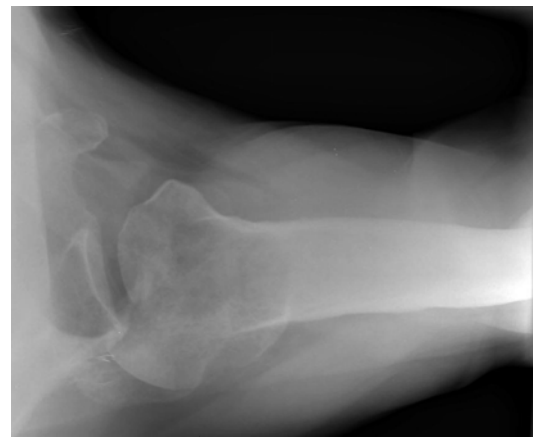

Fig. 20.5 Radiograph demonstrating large reverse Hill-Sachs lesion

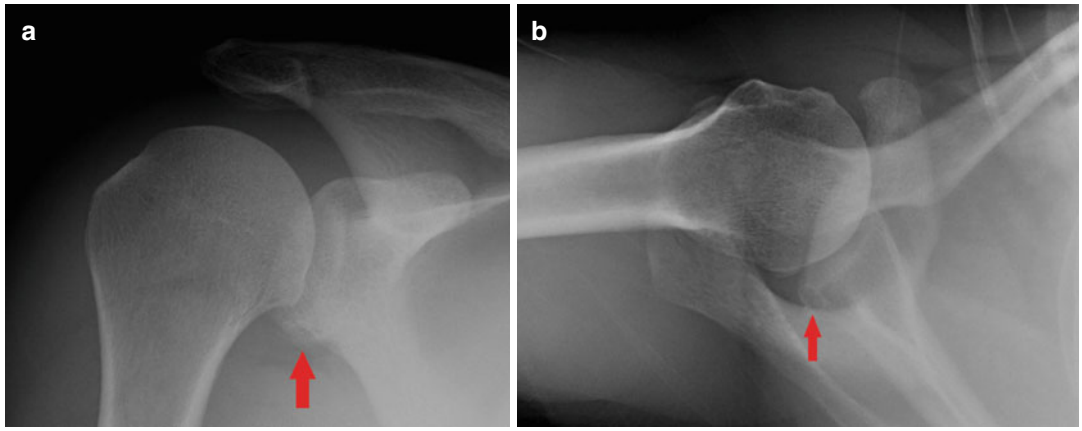

Fig. 20.6 AP (**a**) and Axillary (**b**) radiographs of the glenohumeral joint demonstrating a reverse bony Bankart lesion (*red arrows*)

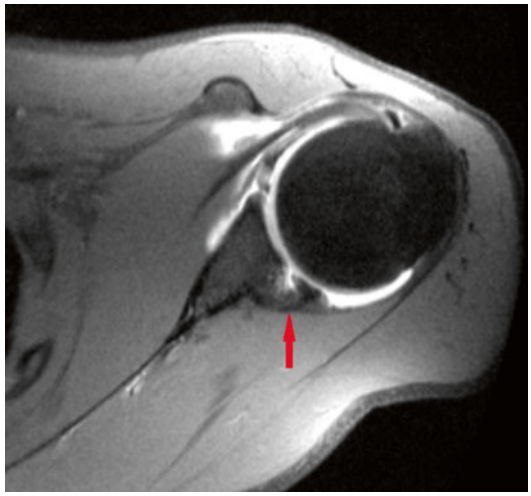

Fig. 20.7 MRI demonstrating a reverse Bankart lesion (*arrow*)

patients with reverse Hill-Sachs lesions involving more than 25–30 % of the humeral articular surface [20]. Both anatomic as well as nonanatomic techniques have been described to correct problems of instability caused by large reverse Hill-Sachs defects. The original McLaughlin procedure requires transfer of the subscapularis tendon into the humeral head defect, thereby preventing the edge of the defect from dislocating and locking behind the posterior glenoid [21]. The Neer modification of the McLaughlin procedure involves transferring the subscapularis tendon along with an osteotomized lesser tuberosity [22]. This modification can be very helpful when an associated lesser tuberosity fracture is present with a reverse Hill-Sachs lesion.

Anatomic reconstruction of a reverse Hill-Sachs lesion can be accomplished by filling the defect with bone graft. The decision to use autograft versus allograft is determined preoperatively depending on the size of the defect. For small reverse Hill-Sachs lesions where approximately 25 % or less of the articular surface is involved, autograft is generally recommended; however, osteochondral allograft is more commonly used for larger defects involving greater than 25 % of the surface [22]. Specifically, we recommend performing humeral head osteoarticular allograft transplantation for these larger defects [23, 24].

Posterior instability resulting from posterior glenoid deficiencies may be corrected using either a posterior iliac bone block or by performing a posterior glenoid opening wedge osteotomy. In addition, Skendzel and Sekiya described a novel technique of arthroscopic glenoid reconstruction using glenoid osteochondral allograft [25]. These posterior glenoplasty procedures are generally indicated in patients with osseous defects as their primary cause of instability; however, they may also be performed in patients who have failed previous posterior capsulorrhaphy.

20.6 Author's Preferred Treatment

We recommend that patients undergo physical examination under anesthesia prior to beginning surgery. The patient should be positioning in the lateral decubitus position with the affected shoulder facing superiorly and the table in a slight reverse Trendelenburg position. The placement of an axillary role under the adducted operative arm can help to better visualize the glenohumeral joint.

We prefer the use of both an anterior and posterior portals providing improved visualization of the posterior labrum and glenoid for precise placement of capsular plication sutures. Bony landmarks should be marked prior to posterior portal placement 1–2 cm distal and medial to the posterolateral edge of the acromion. The anterior portal should be created at a level just superior to the subscapularis and lateral to the coracoid, at a 5 o'clock position. A diagnostic arthroscopy should be performed prior to repair to determine if there is any preoperatively unrecognized pathology contributing to the instability.

During repair, the arthroscope should remain in the anterior portal for better visualization of the posterior labrum and glenoid, and the posterior portal is used primarily as the working portal. We recommend the use of a synovial shaver to debride any friable labrocapsular tissue and prepare the glenoid rim in order to create a more bioactive surface to facilitate healing. We believe that the multipleated plication method is most advantageous in this repair [26, 27]. The first

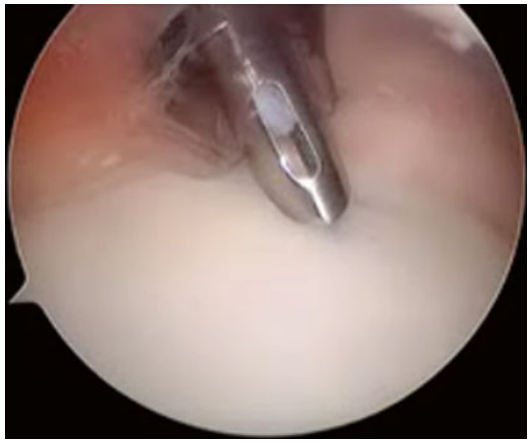

Fig. 20.8 Suture anchor placement: The first suture anchor should be placed at the 7 o'clock position

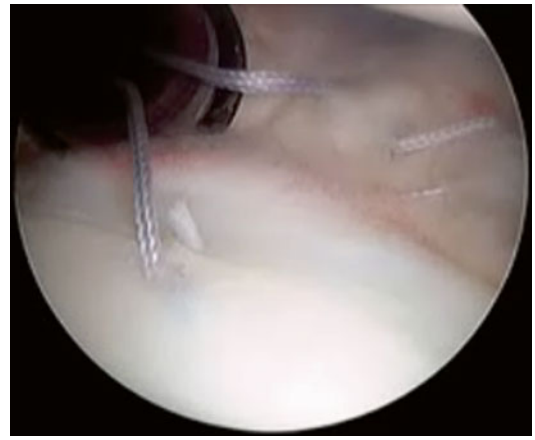

Fig. 20.9 Completed pleats: The pleat technique should be performed two to three times to create the multipleated pattern

suture anchor should be placed at the posterior 7 o'clock position (Fig. 20.8). The inferior suture anchor should be passed through the labrum directly adjacent to the anchor with the help of a soft tissue penetrator. The penetrator should then be used to pierce the posterior inferior and lateral point of the capsule. A shuttle suture technique is then used to pass the anchor suture through the inferior tuck of the capsule. This technique should be repeated two or three times in order to create multiple pleats or tucks of capsule in a superior direction until sufficient tightening has been achieved (Fig. 20.9). We recommend the use of a sliding-locking Weston knot backed with three half hitches to secure the plication [28]. Additional anchors may be placed at 5 o'clock, 9 o'clock, and 11 o'clock, based on the direction and severity of instability. At the completion of the plication, the capsule should feel slightly over tightened (Fig. 20.10). A rotator interval closure is rarely necessary, except by patients who present with a positive sulcus sign in the neutral rotation that does not decrease with 30° of external rotation. The diagnostic arthroscopy should be used to confirm this finding.

Patients should begin postoperative physical therapy on day 1. Patients should begin with passive and active range-of-motion exercises of the joints surrounding the shoulder with gentle passive pendulums of the shoulder. The sling should be removed after 6 weeks and range of motion

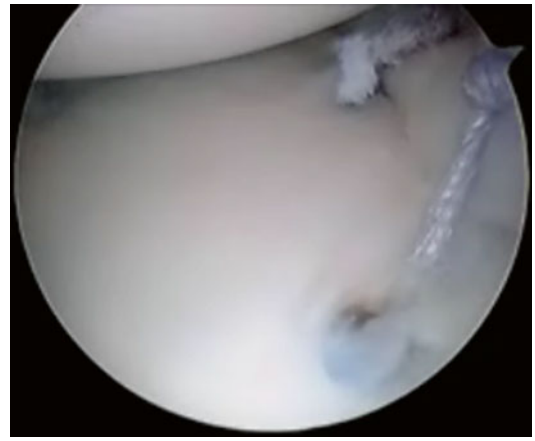

Fig. 20.10 Tightened capsule: The capsule should feel slightly overtightened once all pleats are tied down

should be restored by 2–3 months. Rehabilitations should focus on returning full strength with a goal to return to contact sports around 6 months following surgery.

20.7 Rehabilitation

Cryotherapy should be utilized immediately following surgery in order to control postoperative swelling. For the first month, an external rotation sling is to be worn at all times except when doing exercises. By 2 weeks, pendulum exercises (Fig. 20.11) should be initiated. In addition,

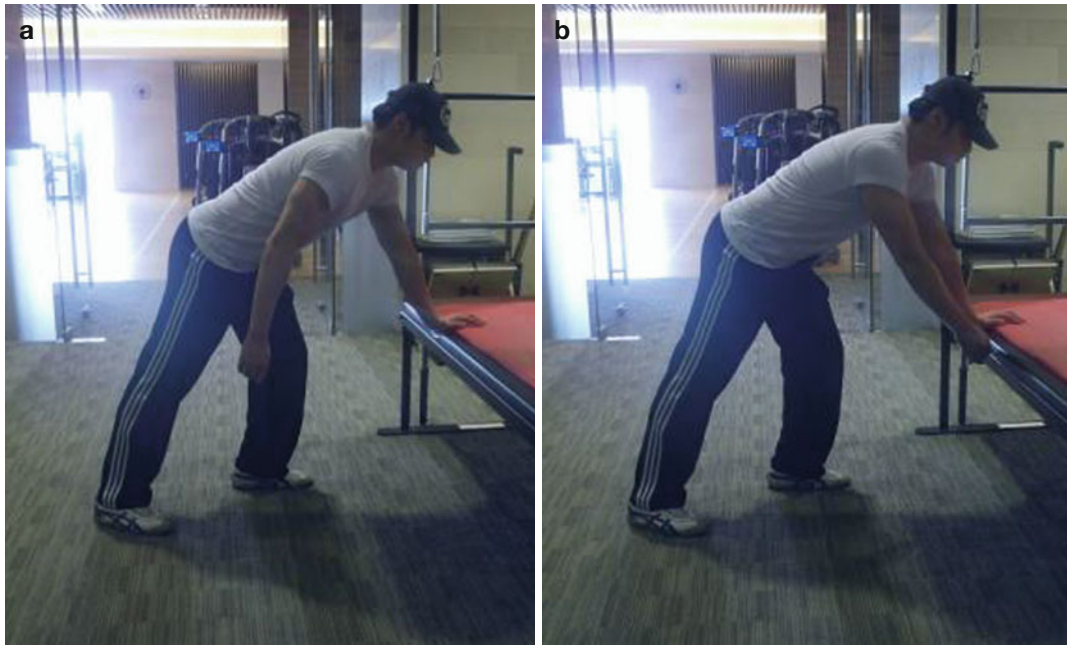

Fig. 20.11 Pendulum exercise: (**a**) The patient should bend forward and place their uninjured arm on a table for support. They should let their injured arm dangle toward the floor. (**b**) The patient should make small circles with their injured arm in a clockwise and counterclockwise direction

gentle passive elevation in the scapular plane to 90° and external rotation to 30° with the arm at the patient's side should be started at this time. Patients should avoid any active abduction, horizontal adduction, forward elevation, and internal rotation during the first month. Between 1 and 2 months postoperatively, passive and active range of motion can begin with limits from 15° of horizontal abduction to full horizontal abduction, full elevation in the scapular plane, 45° of internal rotation, and extension to 20°. During this time, periscapular strengthening and range-of-motion exercises such as shoulder shrugs and scapular retraction exercises should begin. The patient should remain in the sling until 6 weeks postoperatively. Two to four months postoperatively, a strong emphasis should be put on returning to full range of motion, concentrating on periscapular strengthening (protraction, retraction, and elevation) and rotator cuff strengthening with bands and dumbbells (Fig. 20.12). Patients' full range of motion should return by 4 months postoperatively, and active strengthening should begin with a goal to return to full strength, with high-repetition, low-weight exercises.

Once patients are more than 6 months out from surgery and are able to demonstrate full strength, they may begin to slowly return to work and activities under controlled conditions. Patients should advance strengthening of the muscles of shoulder girdle, rotator cuff, and periscapular areas, while avoiding exercises involving heavy weights with low repetitions. Patients may return to functional training exercises, such as swimming, tennis, or interval throwing gradually. Pending approval, patients may return to full work and activities if they are able to achieve full range of motion without pain and tenderness and have a satisfactory clinical exam. Patients that participate in contact sports should be judged on a case-by-case basis but should expect to return to full competition following 8 months of postoperative rehabilitation.

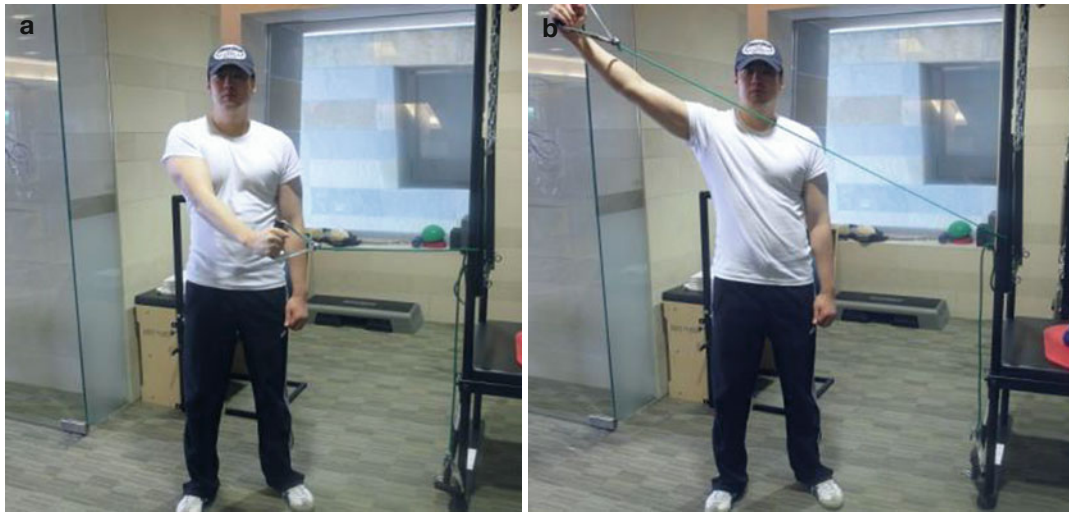

Fig. 20.12 Diagonal pattern exercise with TheraBand: (**a**) The patient should fix TheraBand to the wall or door handle and stand with the uninjured side to the fixed end. (**b**) The patient should hold the band in front of the hip on the uninjured side and pull the band diagonally with the injured shoulder

20.8 Advantages, Pitfalls, and Complications

There are many pearls and pitfalls that should be appreciated before proceeding with arthroscopic treatment. Achieving the correct diagnosis is crucial to the management of the patient's instability. It is important to obtain a thorough history and physical exam including instability and sulcus signs. Radiographic studies are also important in order to rule out bony defects that would be a contraindication for a purely arthroscopic repair. The patient may be positioned in the lateral decubitus or beach chair position; however, we prefer the lateral decubitus position because it allows better visualization and access to the posterior inferior and anterior aspects of the shoulder. Injection of 10–20 mL of sterile saline into the glenohumeral joint helps to inflate the joint to allow for safer placement of the cannula into the joint.

While repairing the soft tissue pathology, the primary pathology inferiorly and posteriorly should be addressed first because each successive repair decreases the working space within the glenohumeral joint. Suture anchors should be used even with an intact posteroinferior glenoid labrum to maintain a tight, stable capsular plication.

It is important to have a strong knowledge of the working anatomy of the shoulder, and care should be taken to avoid damage to the axillary nerve and posterior humeral circumflex artery during arthroscopic portal placement. The axillary nerve lies closest to the glenoid from the 5 to 6 o'clock position with an increasing distance in the posterior quadrant as it wraps around the posterior aspect of the arm exiting through the quadrangular space. It is important during the procedure to stay less than 15 mm medial to the glenoid to avoid the suprascapular nerve.

The risk of complications during this procedure is relatively low. As with all procedures, there is a risk of infection and damage to nerves and vessels such as the suprascapular nerve/artery, axillary nerve, and posterior humeral circumflex artery. It is also possible that patients may be unable to obtain full range of motion even after completing the necessary postoperative physical therapy. This can result from significant over tightening of the joint capsule. Recurrent instability commonly results from the failure to

correct underlying causes of instability such as bony lesions. Continued instability may lead to further surgery to correct previously unaddressed pathology. Lastly, when performing correction of osseous pathology such as posterior glenoid insufficiency with an opening wedge osteotomy, there is a possibility of intra-articular fracture, nonunion, degenerative arthritis, and osteonecrosis of the glenoid.

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Multidirectional Instability and Loose Shoulder in Athletes

21

Hiroyuki Sugaya

21.1 Introduction

In 1971, Endo et al. first introduced the term "loose shoulder" for the shoulders with hypermobility in Japanese literature [1]. In 1980, Neer and Foster provided the first description of multidirectional instability (MDI) and the surgical intervention, the open inferior capsular shift [2]. We may regard the loose shoulder and MDI as a similar disease concept. MDI is a symptomatic glenohumeral joint subluxation or dislocation occurring in multiple directions [2], and the pathoanatomy has been identified as capsular redundancy or constitutional laxity of the glenohumeral joint capsule. Patients with MDI or loose shoulder can present with a variety of symptoms ranging from shoulder discomfort without perception of instability to frequent occurrences of symptomatic subluxations or dislocations. Typically, these patients have reduced scapular upward rotation, an imbalance of muscle strength, and suboptimal neuromuscular control of shoulder function when compared with normal control subjects. The most commonly recommended treatment for MDI and loose shoulders is a nonoperative treatment with an emphasis on physical therapy. This is based on the rationale that strengthening the scapula and rotator cuff muscles compensates for the lack of passive stability and assists in active control of the shoulder. And most of the patients with MDI and loose shoulder respond well with physical therapy-based treatments. However, when it comes to athletes, MDI and loose shoulder can be a debilitating condition because the unstable shoulder renders athletes unable to meet the demands of sports activities, especially in overhead activities.

21.2 Pathoanatomy, Biomechanics, and Preferred Classification

The glenohumeral joint is a highly specialized structure responsible for a wide range of motion of the shoulder with a minimum of bony constraint. Static and dynamic stabilizers interact to provide glenohumeral joint stability. Malfunction of the stabilizers can lead to shoulder instability.

Static stabilizers include the glenoid concavity and version, the labrum, and the glenohumeral ligaments and capsule. Capsule redundancy is widely accepted as a main pathology for MDI and loose shoulder. These patients sometimes demonstrate generalized joint laxity. Dewing et al. [3] measured the cross-sectional area of the capsule with magnetic resonance arthrograms and reported that the area was increased in patients with MDI compared to control subjects. The glenoid shape and version is another

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important factor for the glenohumeral joint stability. Kikuchi et al. [4] reported with a cadaveric study that the anterior and posterior stability decreased with an anterior tilt of 5° and with a posterior tilt of 15°, respectively. Another in vivo study that measured three-dimensional glenoid shape and version showed an increased retroversion and a pronounced flatness of the glenoid in shoulders with MDI [5]. Insufficiency of these structures places higher demands on other shoulder stabilizers.

Dynamic stabilization of the glenohumeral joint is mainly provided by the rotator cuff and the scapulothoracic musculature. The rotator cuff is able to resist humeral head translation through the mechanism of concavity compression in which the humeral head is centered into the glenoid and the rotator cuff imparts a balanced contact pressure to the articulation [6]. Scapular stabilizers are important for shoulder stability, and abnormal scapular kinematics and periscapular muscle function have been reported in patients with MDI [7, 8]. Ogston and Ludewig [7] analyzed three-dimensional shoulder kinematics in patients with MDI and found decreased upward rotation and increased internal rotation of the scapula during scapular plane abduction in comparison with asymptomatic controls. Electromyographic study has shown that patients with MDI have abnormal patterns of muscle activity, such as shorter activity of the pectoralis major and deltoid and longer activity of the supraspinatus, infraspinatus, and biceps brachii [8]. Malposition and malfunction of the scapula may impair stability of the glenohumeral joint in association with the insufficient function of the rotator cuff.

Thus, the cause of MDI and loose shoulder is multifactorial, and most patients have combined factors for initiation of the instability. For example, young throwing athletes with joint laxity who are suffering from minor labral or capsular injuries due to the repetitive overhead activities can develop MDI or loose shoulder. Dysfunction of the scapulothoracic joint due to imbalance of the periscapular muscles can also cause MDI or loose shoulder in such young athletes.

21.3 Clinical Presentation and Essential Physical Examination

Patients with MDI or loose shoulder can present with a variety of symptoms ranging from shoulder discomfort without perception of instability to frequent occurrences of symptomatic subluxations or dislocations. Most patients present with insidious onset and nonspecific, activity-related pain in the second to the third decade of life [9]. The patients often complain decreased strength and deteriorating athletic performance.

Generalized joint laxity is frequently seen in patients with MDI and should be evaluated. Signs of laxity include elbow or metacarpophalangeal joint hyperextension, genu recurvatum, patellar instability, and the ability to place the thumb on the ipsilateral forearm. If severe generalized laxity is found, congenital collagen disorders such as Ehlers-Danlos syndrome should be considered. They can result in abnormal connective tissue properties and consequently contribute to joint laxity (Fig. 21.1). We should pay careful attention to these diseases because surgical stabilization is less successful in patients with these disorders.

An accurate physical examination is critical for diagnosis and adequate treatment of patients with MDI and loose shoulder. Patients should take their clothes off so that the scapula can be fully observed. First, the posture and position of the scapula should be examined. Typically, patients with loose shoulder or MDI demonstrate rounded back with bilaterally protracted scapulae. Next, shoulder motion is checked in both active and passive manner. We should pay attention to the scapular kinematics during the active motion. Passive range of motion is usually normal; however, many MDI or loose shoulder patients describe pain or apprehension during testing.

There are a variety of tests for evaluation of shoulder instability. However, the most important maneuver in the MDI examination is the sulcus test [2]. Inferior traction is placed on the limb with the arm at the side in neutral rotation. The test is positive when a dimple appears distal to the lateral acromion (Fig. 21.2). The test can be also performed with the arm in adduction, abduction, and

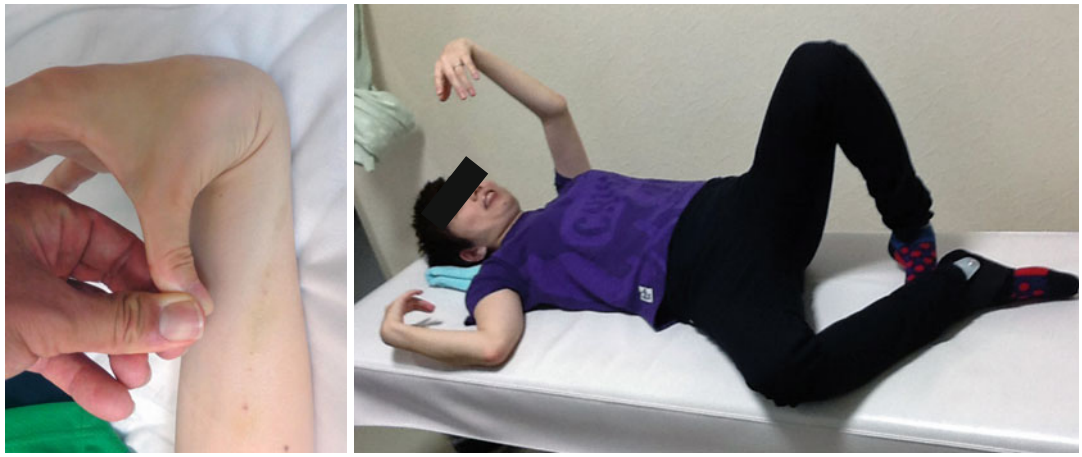

Fig. 21.1 Ehlers-Danlos syndrome (EDS). A 27-year-old lady with hypermobility-type EDS lying on the examination table with her most comfortable posture. She has extremely loose joints with both her shoulders, hips, knees, and ankles as well as upper extremities (*right*). Right metacarpophalangeal joint hyperextension in the same patient (*left*)

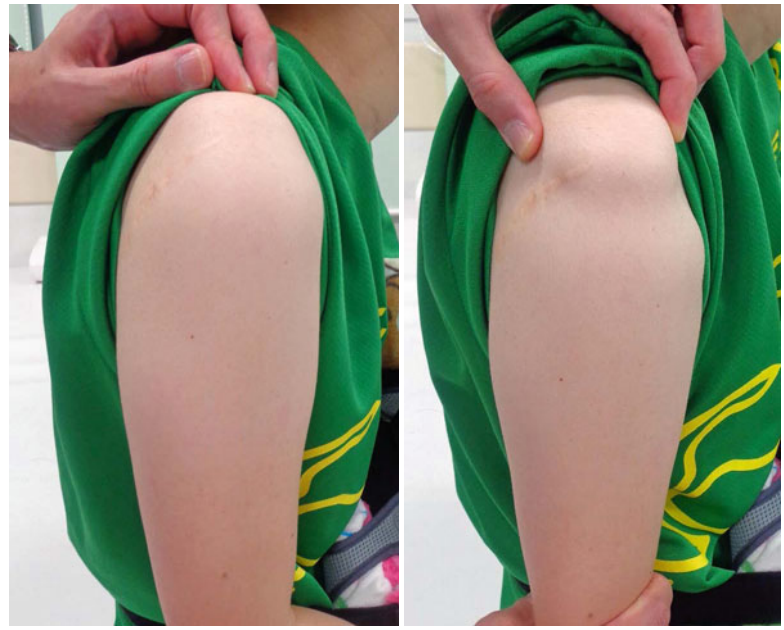

Fig. 21.2 Sulcus test in an MDI patient. *Left*: neutral (no traction), *Right*: inferior traction is placed and the dimple is clear

both internal and external rotation. High degree of glenohumeral laxity is suspected when the displacement of the humeral head is more than 2 cm from the acromion; however, it is not necessarily abnormal unless the patient is symptomatic.

The load and shift test is also frequently used to examine shoulder instability. The test is performed in the sitting position and with the arm at the side in neutral rotation. The humeral head is centered in the glenoid by applying an axial load. The proximal humerus is then translated to determine instability (Fig. 21.3). The test is graded in terms of the degree of translation: grade 0, no translation; grade 1, translation to the

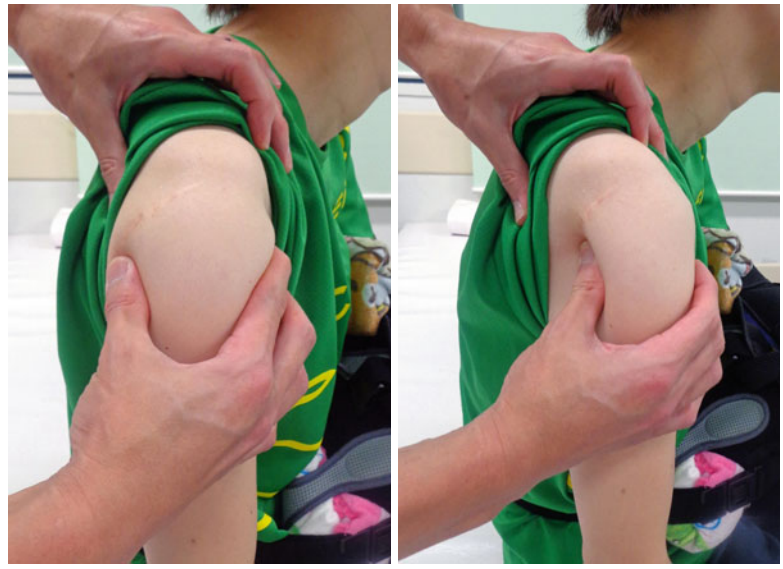

Fig. 21.3 Load and shift test in an MDI patient. *Right*: anterior translational force is applied. *Left*: posterior translational force is applied

glenoid rim; grade 2, dislocation with spontaneous reduction; and grade 3, dislocation without spontaneous reduction.

Other tests for anterior instability include the anterior apprehension test, the relocation test, and the fulcrum test. Tests for posterior instability include the posterior apprehension test and the jerk test. The jerk test is sensitive for posterior instability and is performed in the sitting position. While stabilizing the patient's scapula with one hand and holding the affected arm at 90° abduction and internal rotation, the examiner grasps the elbow and axially loads the humerus in a proximal direction. The arm is moved horizontally across the body. The test is positive when the humeral head slides off the back of the glenoid with a sudden clunk [10, 11].

21.4 Essential Radiology

Diagnosis of MDI is primarily clinical; however, imaging is also helpful in some circumstances. Occasionally, standard radiographs reveal abnormal glenoid version, dysplasia, or hypoplasia. In my institute, we always take bilateral shoulder radiographs with the arms elevated in addition to standard anteroposterior views for young athletes. The radiographs in the elevated position sometimes demonstrate slipping of the humeral head in patients with loose shoulder (Fig. 21.4).

CT scans are optional; however, they can be helpful for precise evaluation of the glenoid shape and version if abnormalities of the glenoid are suspected with standard radiographs. Reformatted and three-dimensional CT can be useful to evaluate the abnormal glenoid morphology.

MRI is frequently used for detecting pathologies in soft tissue in patients with shoulder instability. MR arthrography is preferred to evaluate unstable shoulders because the capsule can be distended, thereby improving definition of the labrum, rotator interval, and glenohumeral ligaments. Frequent finding is increased volume of the glenohumeral joint [3], and labral abnormalities including labral tears are sometimes seen in patients with MDI or loose shoulder (Fig. 21.5). However, these findings are nonspecific and may not reflect actual instability.

21.5 Disease-Specific Clinical and Arthroscopic Pathologies

Patients with MDI or loose shoulder complain pain with the arm in a certain position and/or involuntary dislocation/subluxation and sensa-

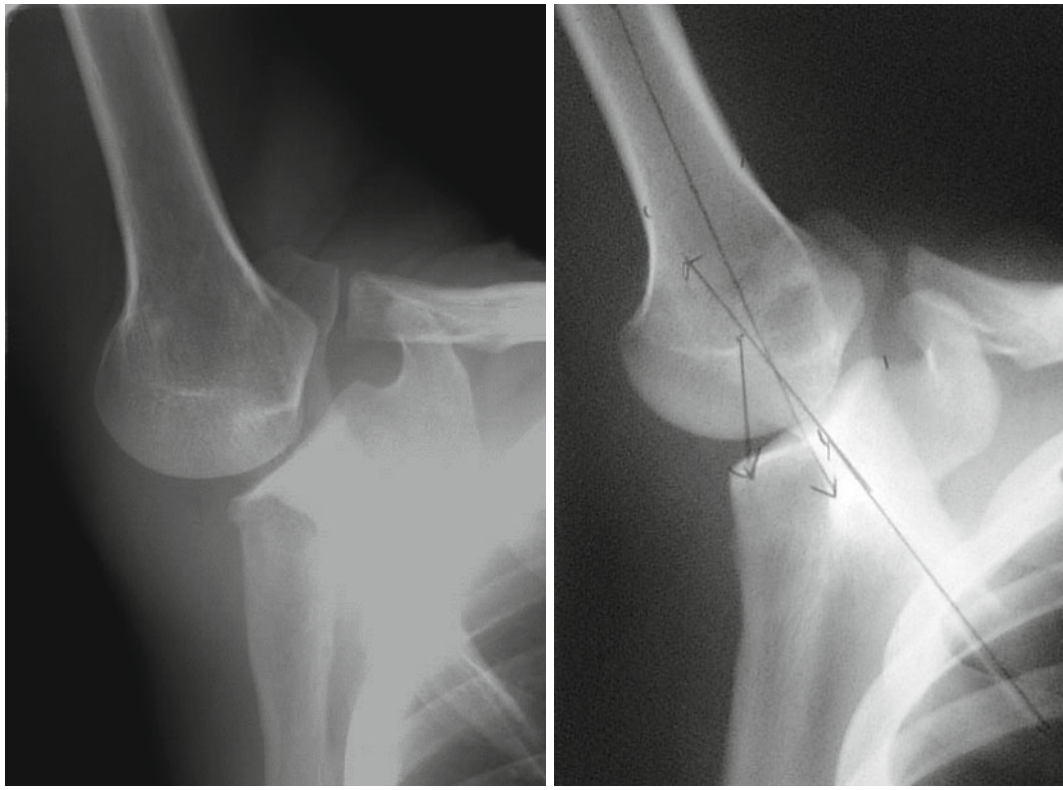

Fig. 21.4 Humeral head slipping in a throwing athlete with loose shoulder. *Right*: normal. *Left*: loose shoulder

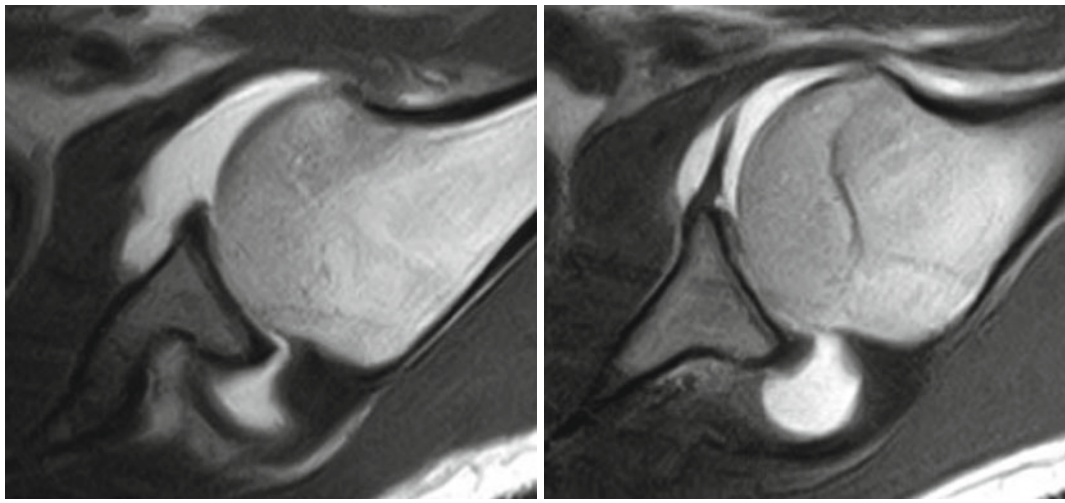

Fig. 21.5 MR arthrography (ABER view)

tion of looseness of their affected shoulder. As described above, majority of patients are accompanied by scapular dysfunction, such as protracted and anteriorly tilted scapula with downward rotation. In addition, they may also loose normal mobility/flexibility in their thoracic spine and rib cage associated with round back. Therefore, the first choice of treatment is

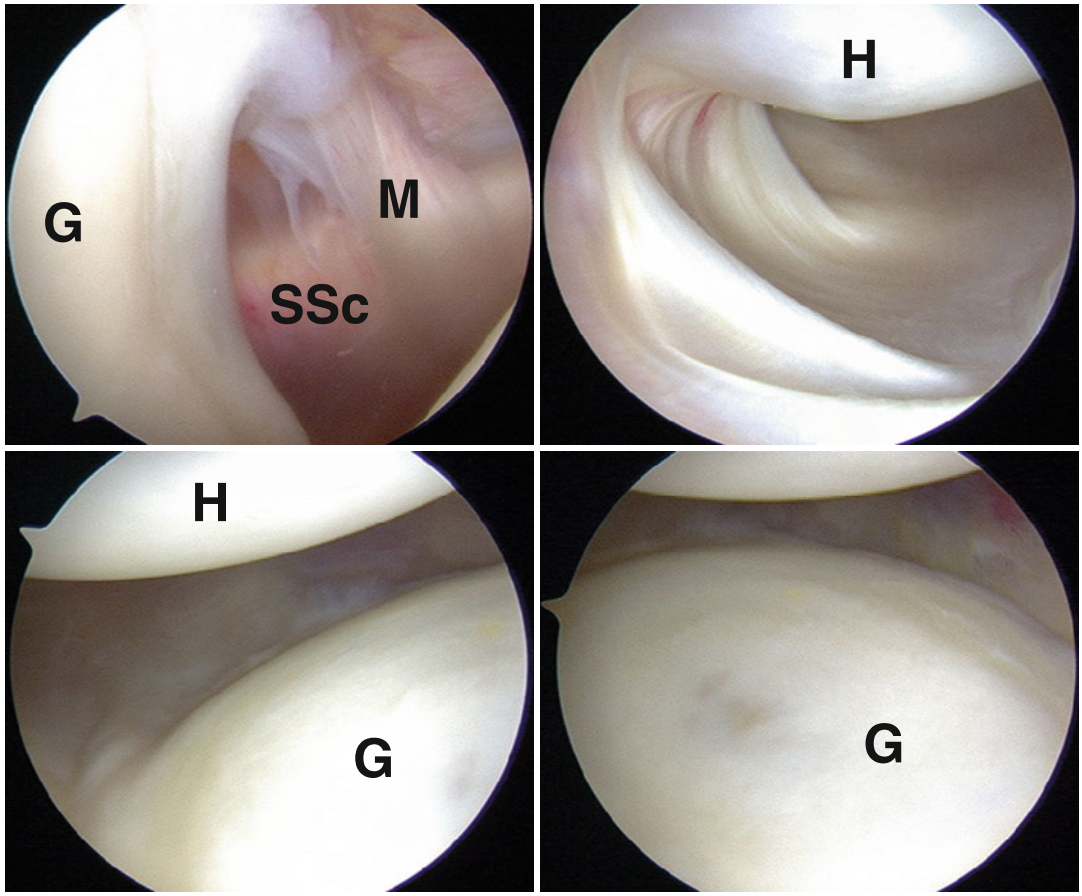

Fig. 21.6 Arthroscopic findings of an MDI patient. The right shoulder is viewed from the posterior portal. Although no anatomical disruption is identified, wide and redundant capsule and thin MGHL are observed. *G* glenoid, *H* humeral head, *SSc* subscapularis, *M* MGHL

to correct these functional problems, and this responds relatively well in the majority of patients [12].

However, even after optimal physical treatment for a certain period, maybe at least 3–6 months, if patients remain symptomatic, surgery is then indicated. As described below, the gold standard of surgical treatment is arthroscopic capsulorrhaphy. When surgeons put the scope into the glenohumeral joint, they often recognize redundant capsule characterized with wide intra-articular space and poor and thin capsular tissue (Fig. 21.6). Sometimes, the labrum and biceps tendon as well as the middle and inferior glenohumeral ligament are also hypoplastic. Although subtle labral injuries are sometimes associated in patients with MDI or loose shoulder, distinct traumatic lesion is not normally observed even after patients become dramatically symptomatic after a certain traumatic event (Fig. 21.7).

21.6 Treatment Options

The standard of care for initial treatment of MDI is rehabilitation. Nonoperative management is successful in approximately 80 % of patients with MDI [12]. Rehabilitation aims for rotator cuff strengthening to maximize the concavity-compression mechanism and scapular stabilization to stabilize the glenoid platform [6].

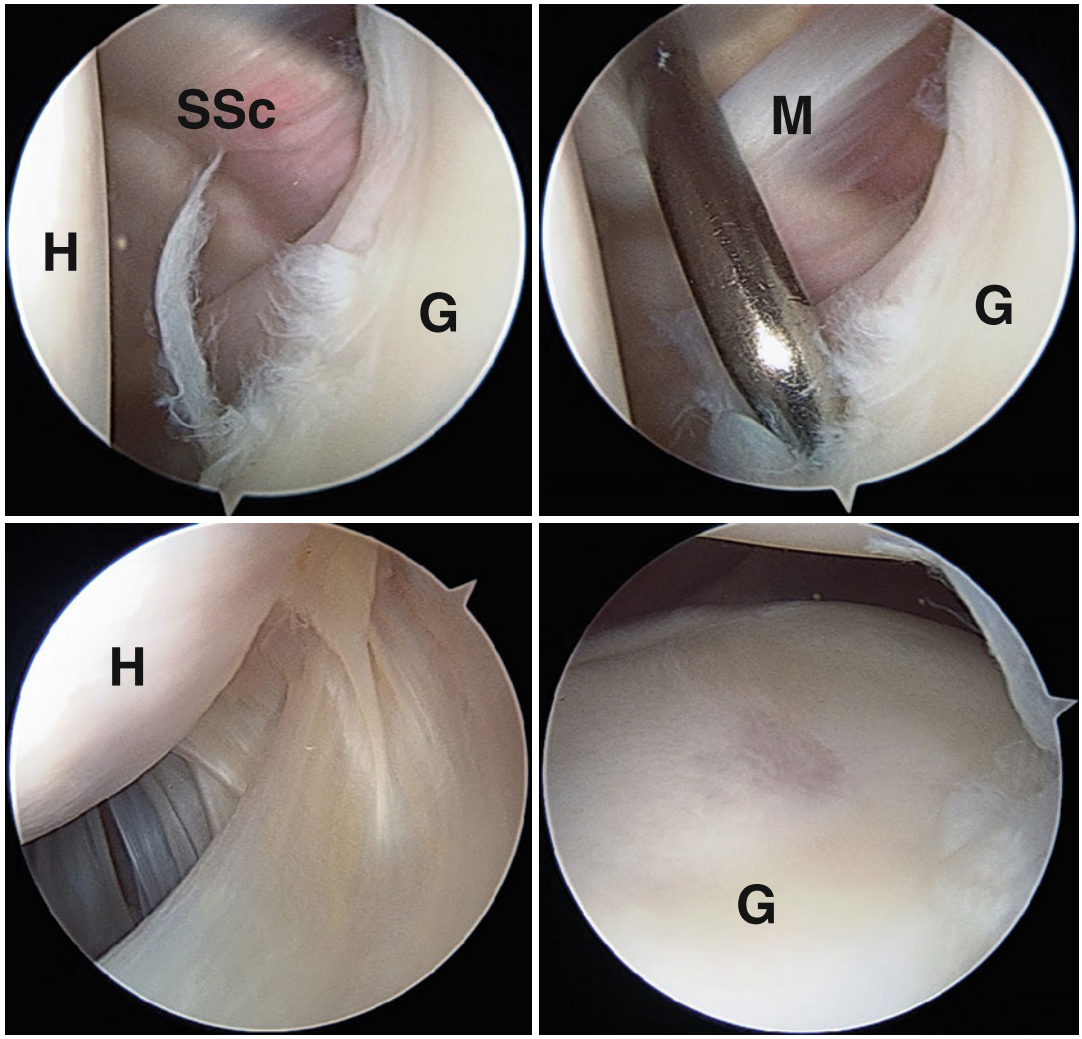

Fig. 21.7 Arthroscopic findings of traumatic shoulder instability associated with loose shoulder. The right shoulder is viewed from the posterior portal (*above left* and *right*, *below left*) and the anterior portal (*below right*). Slight disruption of the anterior labrum is observed (*above left* and *right*) along with a thin, wide capsule (*below left* and *right*). After the last traumatic event, this patient became dramatically symptomatic as she could not hold her arm without a sling until surgery despite this subtle lesion. *G* glenoid, *H* humeral head, *SSc* subscapularis, *M* MGHL

Improving the dynamic positioning of the glenoid and instituting a proprioceptive exercise program can improve the efficacy of dynamic glenohumeral stabilizers. A minimum of 6-month trial of therapy should be devoted to improving stability; however, some authors suggest that longer periods may be required [13].

Burkhead and Rockwood reported good or excellent results of nonoperative treatment in 83 % of patients with atraumatic shoulder instability [12]. However, Misamore et al. [14] reported poor outcomes in a long-term follow-up study. In a cohort of young and athletic patients, 19 of 36 were rated as having poor results with the modified Rowe grading scale, and only 8 patients were free of all pain and instability at a mean of an 8-year follow-up [14]. This study indicates that athletic patients with MDI may have a less favorable response to rehabilitation. It may be important to establish better rehabilitation program to maximize long-term outcomes in athletic patients. However, we should not hesitate

to apply surgical intervention for athletic patients who have a less favorable response to nonoperative treatment.

Surgical treatment should be considered in patients who continue to have debilitating symptoms despite of an appropriate rehabilitation. Surgical management should be individualized to address the anatomic cause of shoulder instability.

The open inferior capsular shift emerged as a successful treatment of MDI following its introduction by Neer and Foster in 1980 [2]. In this technique, the subscapularis is tenotomized, and the capsule is released from the humerus from anterior to posterior. A T-shape incision is made between the middle and inferior glenohumeral ligaments, and the inferior leaflet is shifted superiorly while the superior leaflet is shifted inferiorly. This procedure reduces posterior capsular redundancy and eliminates the inferior capsular pouch. The subscapularis is then reattached superficially to the reconstructed capsule. Neer and Foster reported that instability was eliminated in 39 out of 40 shoulders [2]. Since then, multiple other studies have shown satisfactory outcomes. However, rates of return to sports remain less than optimal. Pollock et al. reported that only 25 out of 36 athletes (69 %) were able to return to previous levels of sporting activity following the open inferior capsular shift procedure [15]. The major cause of the low return rates may be the damage to the subscapularis. Thus, arthroscopic treatment has risen, which has an advantage in the preservation of the subscapularis and the ability of better visualization of the entire capsulolabral anatomy.

At first, arthroscopic thermal capsulorrhaphy was introduced as an alternative to the open capsular shift procedure. Thermal capsulorrhaphy is a technique to shrink the capsule by applying radiofrequency or laser to the capsule resulting in cell necrosis and destruction of collagen cross-links. However, chondrolysis, thermal nerve injury, and high failure rates have been reported. The recurrence rate of instability was 12–64 % in patients with MDI, and thermal capsulorrhaphy is not a recommended procedure for management of MDI [16].

Arthroscopic capsular plication is currently the most popular technique for the management of MDI and loose shoulder. After capsular abrasion, the plication sequence begins in the direction of primary instability to enhance healing. A suture is passed through the capsular tissue and can be sutured directly to the labrum or a suture anchor can be used. These steps are repeated to complete the anterior, inferior, and posterior capsular shifts. It is necessary to avoid axillary nerve injury because the course of the nerve is in close proximity to the inferior glenohumeral pouch. Rotator interval closure can be added to this procedure. As with open inferior capsular shift techniques, arthroscopic capsular plication effectively reduces capsular volume and is dependent on plication magnitude. Cadaveric studies indicated that arthroscopic capsular plication could reduce capsular volume as effective as open capsular shift procedure [17, 18]. A systematic review suggested that clinical results of arthroscopic capsular plication for shoulders with MDI were comparable to open capsular shift with regard to recurrent instability [19]. Gartsman et al. [20] investigated a series of 47 patients with MDI who were treated arthroscopically and reported that 94 % of patients had good or excellent results. In addition, 22 of 26 patients (85 %) returned to their desired sporting level postoperatively [20]. Another study reported that 86 % of 40 patients with MDI could return to their sport with little or no limitation after arthroscopic capsular plication [21]. However, Ma et al. [22] reported that only 5 out of 23 overhead athletes could return to a full level of sports activity despite all patients satisfied with the stability postoperatively. Management of overhead athletes with MDI still remains a challenge.

21.7 Author's Preferred Treatment

Since the majority of patients respond to the physical therapy well, first of all the author sends the patients to the rehabilitation department where they can control scapular position and mobility along with the trunk and chest wall. In addition, home exercises including stretching and strengthening trunk and periscapular muscles are also encouraged. At least 3–6 months of therapy

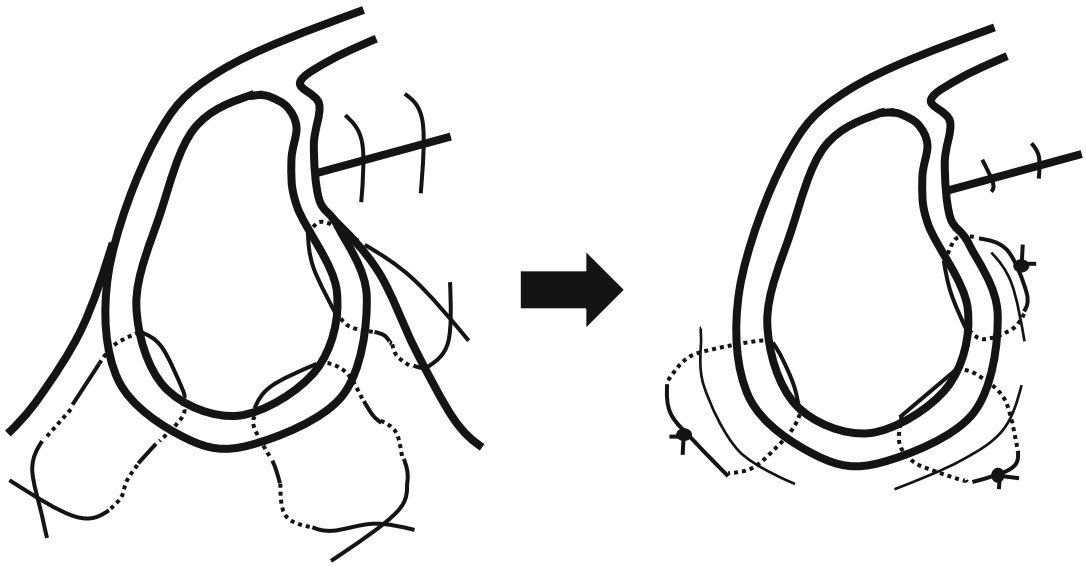

Fig. 21.8 Schematic drawings of arthroscopic "pinch-tuck" capsular plication. First, the scope is inserted to the anterosuperior portal, then a #2 high-strength suture is placed on the posteroinferior capsule and labrum using posterior portal as a working portal in a mattress fashion. Next, the scope is switched to the posterior portal, and then another #2 high-strength suture is placed on the anteroinferior capsule and labrum in a mattress fashion using the anterior portal as a working portal. Finally, the last #2 suture is placed on the anterior capsule and labrum (*left*). Then, all three sutures are tied (*right*). After knot tying, another two #2 high-strength sutures are placed on the rotator interval, the subscapularis tendon, and the superior glenohumeral ligament (SGHL), with the arm at the maximum external rotation

is required in order to eliminate or subside their symptoms.

Once conservative treatment fails, the author prefers to perform arthroscopic stabilization. Since normally it is difficult to detect anatomical disruptions only through physical examination and preoperative imaging in these patients, EUA (examination under anesthesia) and diagnostic arthroscopy are extremely important to make a final diagnosis and decision-making for surgery. The goal of the surgery is to reduce the capsular volume and induce biofeedback through the proprioception of the glenohumeral joint; therefore, capsular plication for the entire inferior glenohumeral ligament (IGHL) is basically performed. If the patient has healthy labrum, three "pinch-tuck" mattress sutures are placed on the IGHL (Figs. 21.8 and 21.9). If otherwise, suture anchor is used for capsular plication. Finally, rotator interval is closed with the arm at the maximum external rotation (Fig. 21.10).

21.8 Rehabilitation

The most common prominent physical presentation in patients with MDI and loose shoulder is protracted and downward rotated scapula (Figs. 21.11 and 21.12). This is based on the loss of flexibility of the thoracic spine and rib cage in addition to the weakness of the core strength. Therefore, rehabilitation is programmed to, first of all, resume mobility and flexibility of the thoracic spine and the rib cage in addition to posture control using correction of the pelvic tilt. Then, the scapular position should be corrected, and the mobility of the scapula also should be resumed. Sometimes trunk and core strengthening exercises are required prior to proceeding to scapular control in order to maintain optimal pelvic tilt, since the posture and thoracic spine alignment greatly affects the scapular position and mobility. Finally, strength and coordination exercises including the core and lower extremities as well

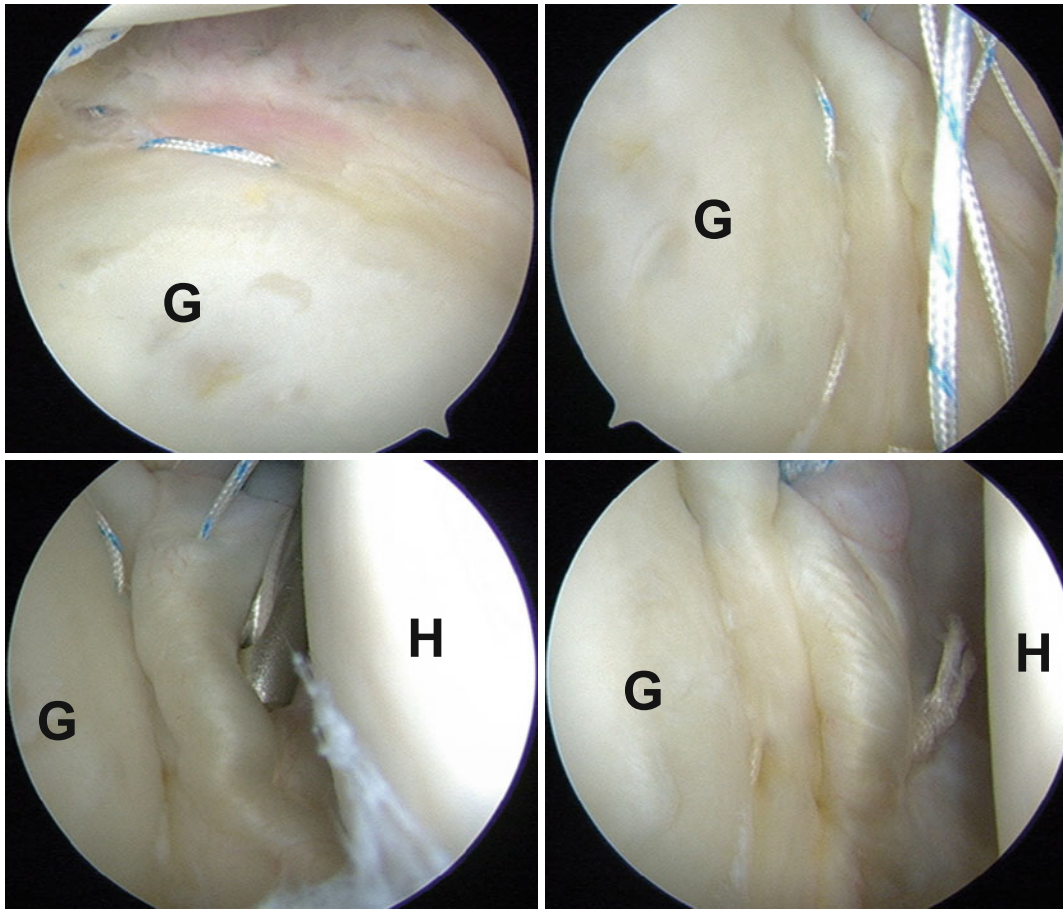

Fig. 21.9 Arthroscopic view of the "pinch-tuck" capsular plication. *Above left*: a #2 suture is placed on the posteroinferior capsule and the labrum in a mattress fashion. *Above right*: the anteroinferior and anterior sutures are placed in a mattress fashion. *Bottom left* and *right*: the anteroinferior and the anterior sutures are tied. *G* glenoid, *H* humeral head

as the rotator cuff and periscapular muscles are initiated. The main goal of rehabilitation in these patients is the correction of the scapular position and to resume normal mobility of the scapula.

21.9 Experience in Treatment of Athletes

A 21-year-old collegiate female gymnast suffered left shoulder subluxation during practice using the still rings with male gymnasts. Since then, she has been experiencing unstable sensation associated with occasional subluxation when she played the parallel bars and even during running. One year later after the initial event, she was referred to me complaining disability of her left shoulder during both practice and games. During physical examination, she did not demonstrate any loss of motion but complained apprehensive sensation when her arm was placed on the maximum external rotation, flexion, and internal rotation. Imaging study demonstrated that wide capsular volume with a slight posterior capsular injury but no apparent Bankart lesion on MRA images (Fig. 21.13). Therefore, arthroscopic stabilization was performed 6 weeks after her initial visit. During examination under anesthesia, she demonstrated hyperlaxity of the left shoulder in multiple directions. Arthroscopy revealed wide

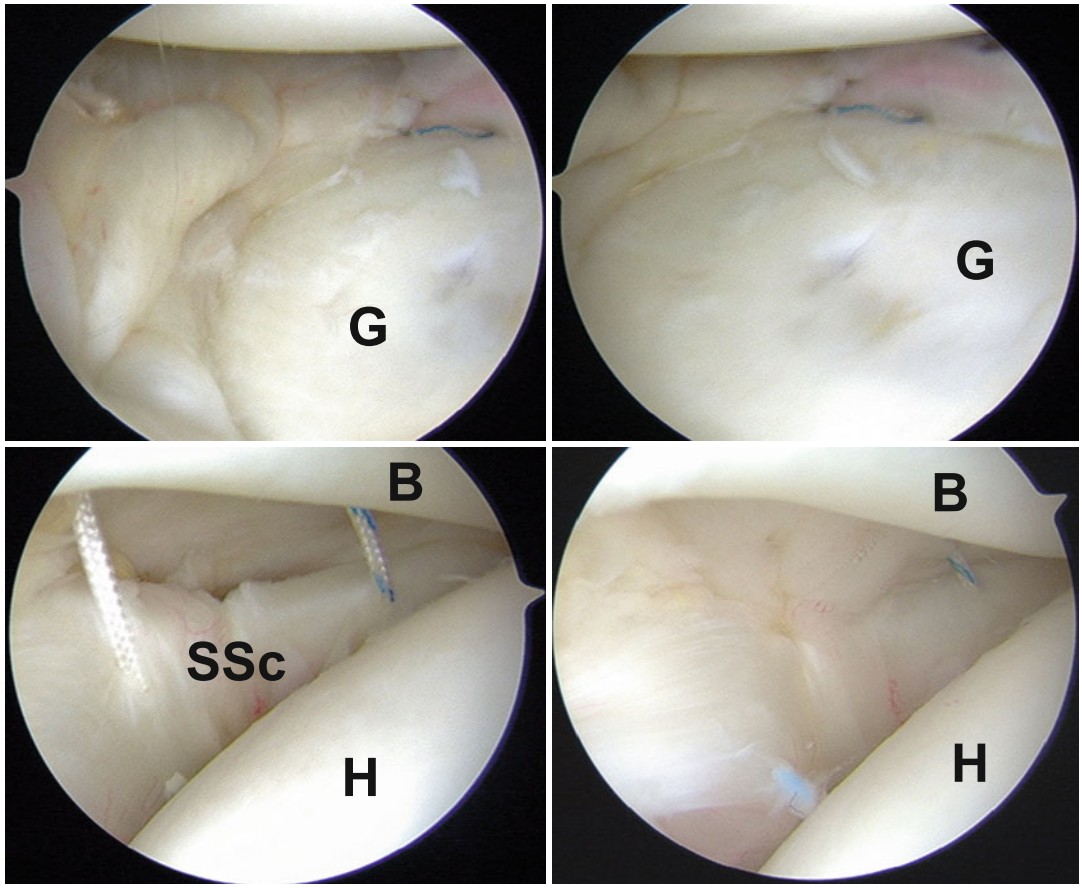

Fig. 21.10 Final view of the arthroscopic "pinch-tuck" capsular plication and rotator cuff repair. *Above left* and *right*: view from the anterior portal after capsular plication. *Bottom left*: two #2 sutures are placed on the subscapularis tendon and SGHL with the arm in the maximum external rotation. *Bottom right*: these sutures are tied with the arm in the same external rotation. *G* glenoid, *H* humeral head, *SSc* subscapularis, *B* biceps tendon

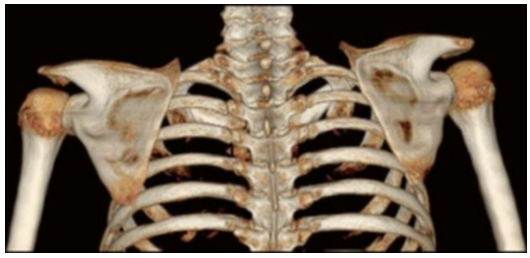

Fig. 21.11 A 3DCT image of the scapulae and rib cage in an athlete. A view from the back. Protracted scapula is observed on the affected side (*right*). Please refer to Fig. 35.3

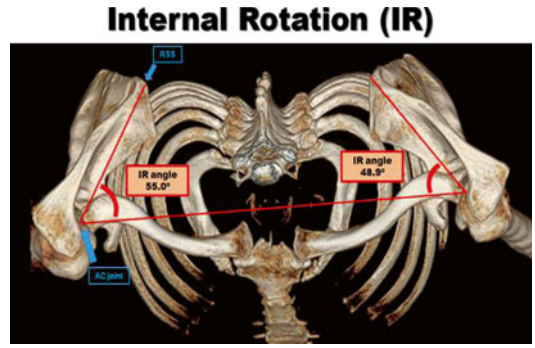

Fig. 21.12 A 3DCT image of the scapulae and rib cage in an athlete with protracted scapula (*right*). A view from the top. The right scapula rotates anteriorly relative to the rib cage compared to normal side (*left*). Please refer to Fig. 35.8

capsular volume and posterior labrum and capsular injury, but Bankart and Hill-Sachs lesions were not confirmed (Fig. 21.14). Arthroscopic posterior capsular repair and anteroinferior capsular

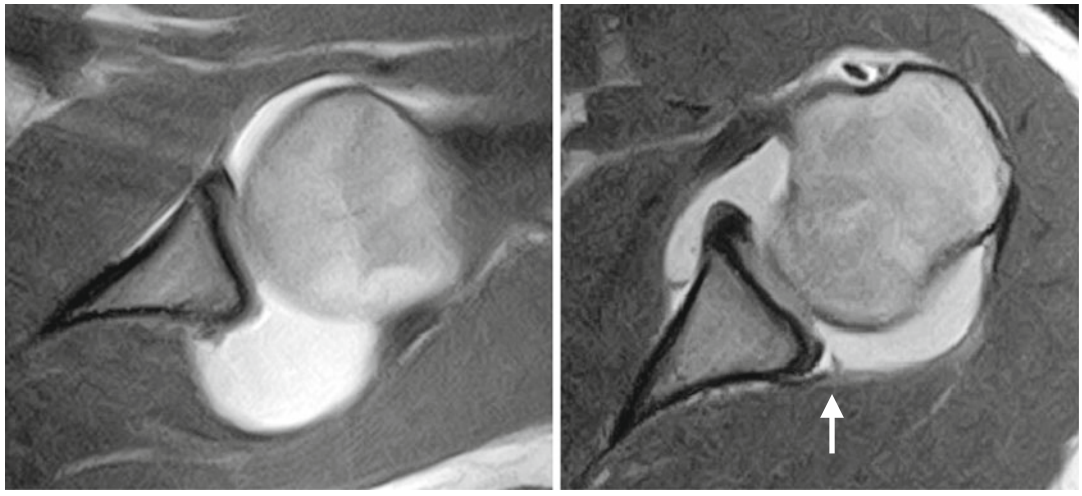

Fig. 21.13 Preoperative MRA images. The patient demonstrated wide posterior capsular volume but did not demonstrate Bankart lesion nor anterior translation of the humeral head on the ABER image (*left*). Posterior capsular injury was suspected on the axial image (*right*, *arrow*)

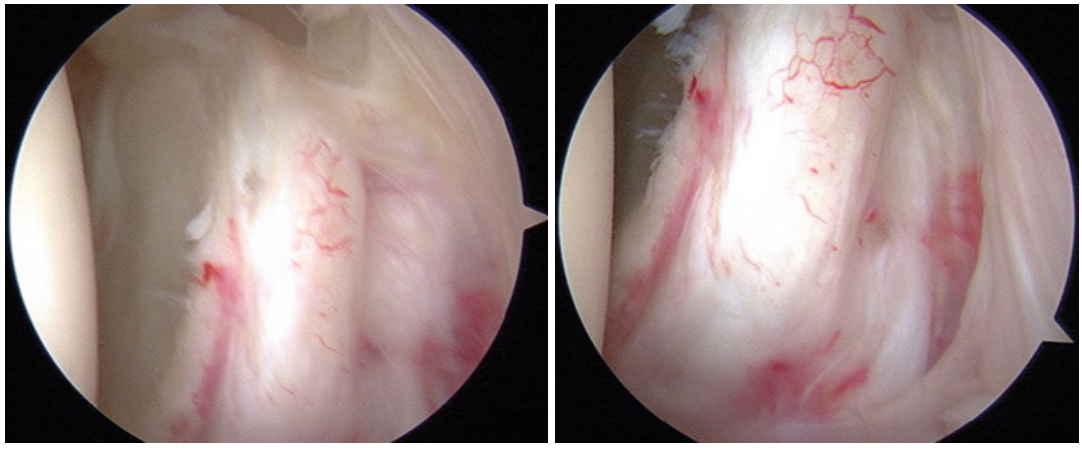

Fig. 21.14 Arthroscopic findings. Posterior capsular tear and labrum injury associated with a redundant capsular volume were confirmed

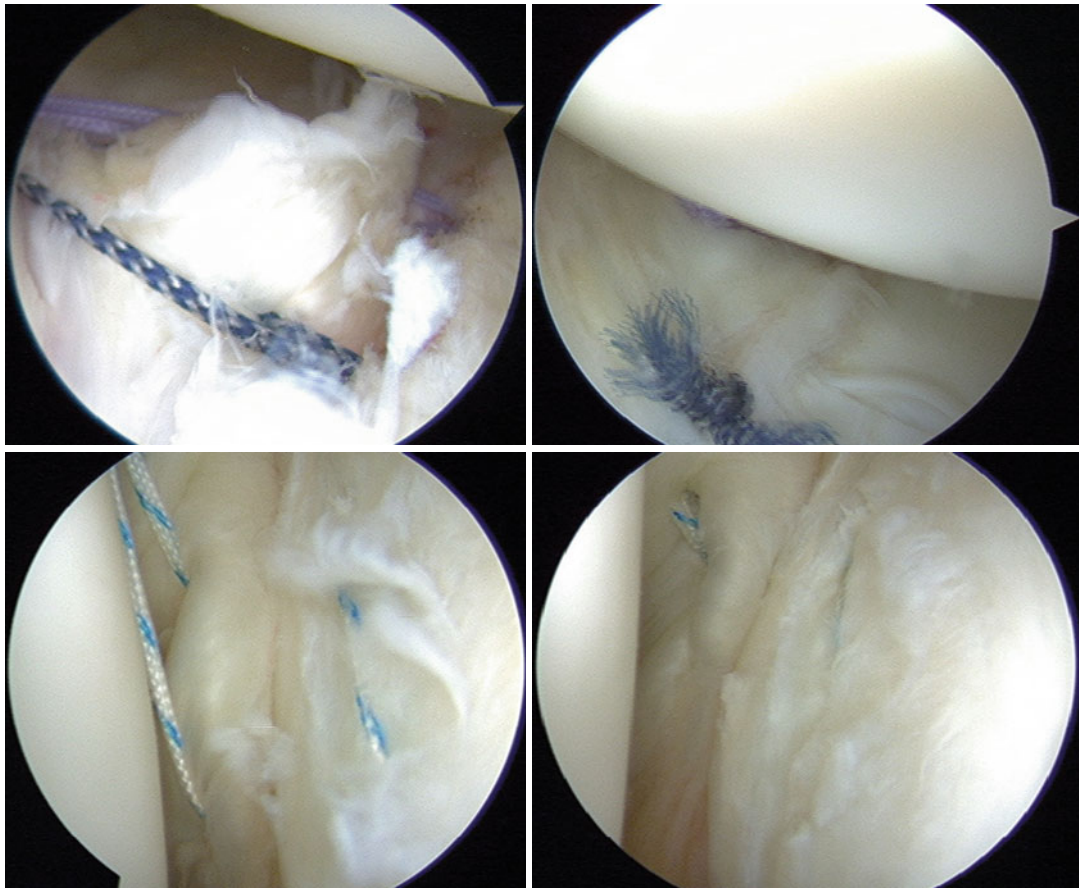

Fig. 21.15 Arthroscopic stabilization. Posterior capsular repair using two suture anchors (*above images*) and anteroinferior capsular plication (*bottom images*) were performed. *Left*: before knot tying. *Right*: after knot tying

plication in addition to the rotator interval closure were performed (Fig. 21.15). Three months later, she began to practice and returned to games 6 months after the surgery.

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Acromioclavicular Joint Problems in Athletes: Part I – Osteolysis of the Distal Clavicle

22

Yon-Sik Yoo

22.1 Introduction

Osteolysis of the distal clavicle (DCO) is an overuse injury, a sequela of repetitive microtrauma. It is essentially a stress-induced microfracture or stress reaction or stress failure syndrome resulting from intolerable exercise dose [1] leading to slow dissolution and resorption of the distal end of the clavicle. It is seen in strength and power athletes and overhead heavy job employees. It has been termed as "weight lifter's shoulder" [2].

In 1936, Dupas et al. first described osteolysis in the distal clavicle as a result of trauma [3]. In 1959, Ehricht was the first investigator to describe distal clavicular osteolysis following chronic repetitive microtrauma in an air-hammer operator [4]. Since then, distal clavicular osteolysis (DCO) has been separated into traumatic (minor trauma) and atraumatic (stress-induced) pathogeneses [5, 6]. Subsequently, it was diagnosed in a judo player, a deliveryman, and a handball player and recently in weight trainers in whom it was thought to arise from a stress failure syndrome that involves resorption of the distal clavicle [7].

Traumatic DCO usually results from acromioclavicular joint dislocations, fractures of the clavicle, and even minor contusions without demonstrable musculoskeletal injuries. Atraumatic DCO (ADCO), also called overuse or stress-induced DCO, is the most common variety. In 1982 the first series of male weight trainers who developed ADCO was reported. Excessive activities that load the outer clavicle and AC joint can lead to osteolysis, as the repetitive damage exceeds the ability for the bone to heal after loading. The most widely accepted etiology involves a connection between microfractures of the subchondral bone and subsequent attempts at repair, which is consistent with repetitive microtrauma [8]. To add further confusion to diagnosis, idiopathic DCO has been described and three cases of the same have been reported by Hawkins et al. in 2000.

In 1982, Cahill described the first series of 46 male weight trainers with emphasis on upper extremities with a mean age of 23.3 years who developed ADCO [1]. Since then, there have been more than 100 cases reported in youngsters involved in strength and power athletics and overhead heavy jobs. In many of these case reports, the upper extremity stress came from several months of intensive training and lifting [6]. In most of these case reports, there was no history of accidental trauma. Matthews et al. reported a case of ADCO in a female bodybuilder [5].

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22.2 Pathoanatomy and Biomechanics

22.2.1 Anatomy and Biomechanics of the Distal Clavicle

The flat lateral end of the clavicle functions to resist muscular and ligament forces, unlike the medial end which resists axial loading. The acromioclavicular joint (AC) is a diarthrodial joint, stabilized by the coracoclavicular ligaments (conoid and trapezoid), the superior and inferior AC ligaments, and the AC capsule. A fibrocartilaginous meniscal disk is present between the convex distal clavicle and the flat acromion incompletely dividing the joint. The joint surface usually slopes inferomedially in spite of many variations in the orientation of the AC joint demonstrated by Urist [9]. The coracoclavicular ligaments provide vertical stability to the AC joint, while the AC ligaments confer horizontal stability. Since the AC joint is superficial and a relatively weak link in the axial-appendicular interface, it is susceptible to macro- and microtrauma. The AC joint is maximally loaded with heavy overhead activities.

22.2.2 Pathogenesis

The pathogenesis of DCO has often been debated. Numerous hypotheses have been discussed without clear consensus [10, 11]. These include dysfunction of the autonomic nervous system with secondary alteration in the blood supply, catabolic hyperemia, ischemic necrosis of bone, reactive synovitis, and stress fracture [12, 13].

The most accepted etiology is repetitive microtrauma or repetitive stress injury of overtraining resulting in microfractures of the subchondral bone and subsequent attempts at repair. There will be a precarious balance between bone absorption and deposition [14]. Cahill et al. found microfractures in the subchondral bone in 50 % of the surgical specimens in their series and proposed that repetitive microtrauma caused subchondral stress fractures and attempted remodeling [1]. Hyperextension of the shoulder during weight training places excessive traction on the AC joints and leads to cumulative subchondral stress fractures with a subsequent hypervascular response [15] and ultimately contributes to ADCO pathogenesis. The surgical specimens of the patients also revealed intense osteoblastic activity of the subchondral bone, suggesting an active repair process. The articular cartilage of the lateral end of the clavicle exhibited fissuring, degeneration, and areas of complete absence [8]. Matthews et al. reported the histopathological examination of resection of the distal clavicle in a female patient. The specimen revealed subchondral microcysts, disruption of the articular cartilage, and metaplastic bone formation with increased osteoclastic activity, again consistent with a repetitive stress phenomenon [5].

The second theory of ADCO is synovial invasion of the subchondral bone as a possible cause of osteolysis as described by Brunet et al., and MRI findings have been reported to be similar to synovial proliferation [16].

Other theories include synovial hyperemia followed by osteoclastic bone resorption, neurovascular damage with autonomic nerve dysfunction, ischemic necrosis, and metabolic effects of hyperemia [17]. Roach and Schweitzer reported an association between spinal cord injury and osteolysis of the distal clavicle in seven patients [18].

22.2.3 Pathology

The consistent findings are chronic inflammation, fibrosis of synovial membrane, loss of trabecular structure, articular degeneration, and osteoblastic activity. ADCO resected specimens reveal fragments of weakly mineralized trabecular bone proximally, dense scar tissue distally, and a thin, unorganized villous hyperplastic fibrocartilaginous layer with occasional osteoclastic multinuclear giant cells. There will be active osteoblastic surfaces with abnormally large osteoid seams and hypervascular connective tissue at the sites of bone resorption. Proponents of synovial pathogenesis have shown evidence of hypertrophic synovial tissue migrating into the distal clavicle across the cartilaginous

surface, resulting in chronic degeneration of the joint [1, 10, 17].

Finally, it has been suggested that a direct communication between the lesion and the AC joint is a distinguishing pathological feature [19]. The pathological process can be divided radiographically into lytic and reparative phases.

22.3 Clinical Presentation and Essential Physical Examination

The incidence of atraumatic osteolysis has paralleled the increase in the number of athletes performing strength training. Scavenius and Iversen [20] reported a prevalence of 27 % in weight lifters. DCO often occurs concomitantly with other shoulder disorders (e.g., instability, impingement, rotator cuff tears, tendinitis, labral disease) [1].

22.3.1 Clinical Presentation

Even though DCO is a benign self-limiting process, it causes substantial morbidity among competitive sports persons and manual laborers. It may be bilateral.

Haupt HA observed that pain and discomfort in trainees is often more severe the night after a weight-lifting program [2]. This condition is commonly seen in young active males involved in competitive sports and heavy manual labor. There will be an insidious onset of dull aching pain in the shoulder region related to activities and aggravated by any activity that stresses the AC joint. The pain is exacerbated by weight training like bench presses, military press, push-ups, and dips on the parallel bars as well as sports-related activities like throwing, overhead activities, and horizontal adduction. There will not be a history of major injury to the shoulder region. There will be difficulty sleeping on the affected side. The pain may progress and radiate to the surrounding deltoid or trapezium muscles and is relieved by prolonged rest. There may be weakness of the affected shoulder and restricted mobility.

22.3.2 Essential Physical Examination

The victims are usually young with well-developed musculature and a lower than average body fat content [21]. Swelling of the AC joint and surrounding soft tissues may be present and well appreciated in unilateral cases. There will be point tenderness over the affected distal clavicle and the AC joint. The pain will be aggravated with a cross-body adduction maneuver and behind the body internal rotation, both active and passive. The AC joint will be stable in both horizontal and vertical planes. Local crepitation may be present. The range of movements of the shoulder is normal except for some discomfort in terminal ranges of adduction and internal rotation. A comprehensive examination of the shoulder should be completed to rule out concomitant pathologies. DePalma's type I and II AC joints with more vertical than horizontal orientation (more forces concentrated at the distal clavicle) appear to be more susceptible for DCO. Cervical spine and neurovascular evaluations are also important to rule out as potential sources of referred pain. An AC joint injection can be both a diagnostic and a treatment modality in the management of DCO.

22.4 Essential Radiology

22.4.1 Plain Radiographs

It is difficult to diagnose the DCO on plain radiographs in the early stages due to considerable age- and activity-related as well as radiographic technique-related variation in the radiographic appearance of the distal clavicle of the individual [6]. Radiographs of both AC joints AP view with 30° cephalic tilt will reveal very subtle changes months to years after the onset of symptoms. Zanca AP view with 15° cephalic tilt will better visualize the AC joint without overlapping the spine of the scapula [22]. Although soft tissue swelling of the AC joint is the earliest finding, it is least specific [17]. The early signs in radiographs include focal loss of subchondral bone in a typical

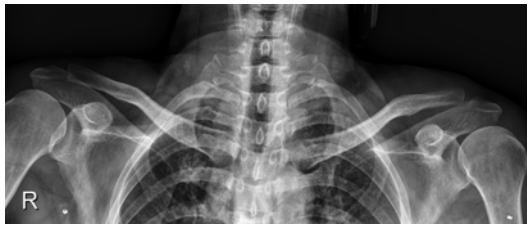

Fig. 22.1 Posttraumatic osteolysis of the distal clavicle on the right side

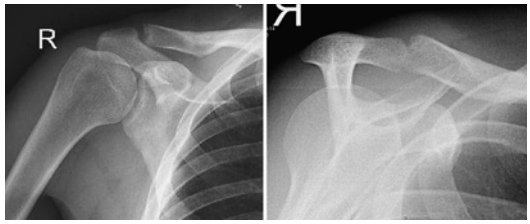

Fig. 22.2 Zanca view and scapular Y view showing subtle erosions along the subchondral bone of distal clavicle

"flame-shaped" pattern without evidence of osteoarthritic changes [11], microcystic changes in the distal clavicle, and widening of the AC joint without involvement of the acromion [1]. The presence of panarticular involvement should prompt us to consider other differential diagnoses like AC joint arthritis. Better appreciated findings include varying degrees of distal clavicle osteopenia early in the disease with progressive erosion, resorption, and tapering of the distal clavicle, cupping of the acromion, dystrophic calcification [12], and AC joint widening late in the disease [5].

During lytic phase which may last for 12–18 months, pathological features include osteopenia, cortical margin resorption, subchondral cystic changes, and subsequent joint space widening resulting in progressive radiolucency of the distal clavicle up to 0.5–3.0 cm and tapering of the distal clavicle. The reparative phase lasting for a 4–6-month period is characterized by evidence of healing like reconstitution of the distal clavicular cortex with a reduction in subchondral cysts, but the AC joint classically remains permanently widened (Figs. 22.1, 22.2, and 22.3).

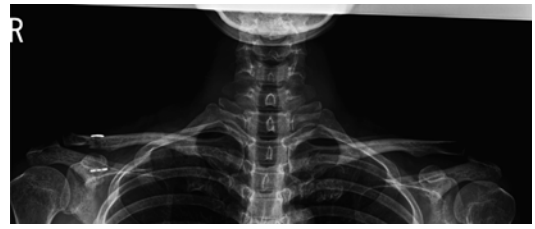

Fig. 22.3 Distal clavicular osteolysis after fixation of fracture of distal clavicle

22.4.2 Scintigraphy

Early in the course of DCO, Tc-99 scintigraphy will show marked increase in uptake in angiographic and blood pool phases in the distal clavicle. In addition there will be increased focal distal clavicular uptake in the delayed phase.

At times, there is also increased activity in the adjacent acromion [1]. But the difficulty with this bone scan is high sensitivity and very low specificity, which needs to be interpreted in the background of clinical findings. Some critics have pointed out that the metaphyseal end of all long bones demonstrates an increase in the uptake on scintigraphy, and the clavicle is no exception. A further increase in the uptake of that area can represent a simple increase in bone turnover, due to the stress applied by young individuals, and is a normal phenomenon [23]. It may be related to an increased blood flow and blood pooling.

22.4.3 Magnetic Resonance Imaging (MRI)

MRI demonstrates increased signal intensity associated with T2-weighted images, most notably on the fluid-sensitive STIR and fat-suppressed spin echo on the T2-weighted sequences.

Bone marrow edema in the distal clavicle is the most common manifestation of DCO and it has a high correlation to patient symptoms [12], but edema may also be seen in the acromion.

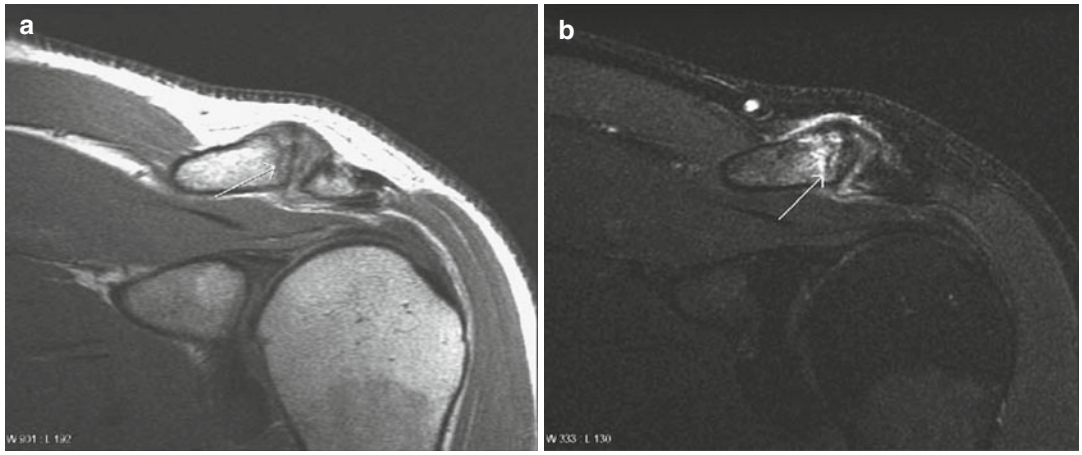

Fig. 22.4 Coronal oblique proton density (**a**) and fat-suppressed intermediate (**b**) MR images show extensive edema in the distal clavicle. Note the clearly visualized hypointense line (*arrow*), centered within the edema in the distal clavicle and consistent with a subchondral fracture. Also, there is minimal edema in the tip of the acromion. Fluid is present in the acromioclavicular joint

Other common findings are osseous fragments and osseous irregularity [12], bulbous prominence of AC joint capsule, minimal joint effusion, and intra-articular bone fragmentation [12].

Undisplaced subchondral fracture in the distal clavicle (hypointense subchondral line centered within edema of distal clavicle) [24] is the most recently established finding (Fig. 22.4).

22.4.4 Ultrasound- or CT-Guided Injections of the AC Joint

Injection of Xylocaine or Sensorcaine (local anesthetics) into the AC joint may temporarily relieve the pain and can be used as a diagnostic tool. The greatest benefit may be that a positive temporary relief of pain can be seen as a diagnostic tool for confirming that the pain is indeed localized in the AC joint. Intra-articular corticosteroids with or without Hyaluron can be considered for short-term symptom relief, if patients do not respond to conservative treatment for 3 months [6]. However, they provide little long-term relief. Image-guided injection allows location of the best point on the skin, appropriate depth and needle inclination, and correct positioning of the needle tip [6].

22.5 Disease-Specific Clinical Pathology

22.5.1 Differential Diagnosis

Although DCO is a benign condition and straightforward to diagnose, some of the more important conditions should be excluded at the time of diagnosis. These include glenohumeral and subacromial injuries, multiple myeloma, rheumatoid arthritis, hyperparathyroidism, gout, scleroderma, septic or tubercular arthritis, osteoarthrosis of the AC joint, corticosteroid-induced arthropathy, primary and metastatic neoplasms, massive essential osteolysis (Gorham's disease), rickets, progeria, eosinophilic granuloma, and cleidocranial dysplasia [25].

The diagnosis of DCO is usually made by history, physical examination, and plain radiographs.

Bone scans, MRI, and differential injections are helpful in patients with equivocal findings or other shoulder problems [21].

22.5.2 Natural History

The osteolytic process is variable in time but consistent in evolution. Lytic phase may continue for 12–18 months, resulting in 0.5–3 cm of bone loss from the distal clavicle and rarely the acromial end. Calcification of ligaments and subperiosteal reaction may occur. During the reparative phase, the distal clavicle becomes smooth and tapered with reconstitution of cortices but the AC joint remains permanently widened [17, 26].

DCO is a self-limiting disorder, with resolution within 1–2 years with activity modification. Although most patients respond to conservative management, with relief of symptoms along with partial or complete osseous restoration of the clavicle, symptoms often return with resumption of previous activity level [27]. There will be gradual cessation of symptoms from active lytic phase to burnt-out phase [17].

22.6 Treatment Options

Treatment methods for DCO are generally driven by symptomatology and disease stage. However, controversy does exist on the course of the disease, with some authors concluding that insufficient treatment predisposes and exaggerates the osteolytic process, whereas others believe that the eventual severity of bony damage is predetermined and directly related to the severity of the inciting event [16, 25].

22.6.1 Nonsurgical Treatment

Early diagnosis and treatment have been shown to successfully decrease clinical symptoms, halt the osteolysis process in some cases, and result in varying degrees of reversal and healing. A delayed diagnosis typically results in a permanently widened AC joint with varying degrees of mechanical dysfunction and pain [26].

Avoidance of provocative maneuvers, modification of weight-training techniques and behavior, immobilization of the affected extremity, cryotherapy, and nonsteroidal anti-inflammatory drugs (NSAIDs) constitute the basis of initial treatment. Corticosteroid injections have been used with limited success. Haupt has suggested several modifications in the young athlete's weight-training program [2]. Most of the specific modifications of weight-training techniques involve narrowing the hand spacing on the barbell (less than 1.5 times the biacromial width) and controlling the descent phase of the bench press to end approximately 4–6 cm above the anterior chest. The narrower handgrip allows the athlete to make adjustments to the component angles of the bench press by maintaining shoulder abduction at less than 45° and shoulder extension at less than 15°. This then decreases the compressive force on the distal clavicle [28]. Haupt HA promotes a routine program in which the bench press, dips, and push-ups are eliminated. Alternative recommendations are the cable crossover, dumbbell decline press, and incline press with straight bar [2]. All pressing motions are performed with a narrow grip, no greater than 1.5 times the biacromial width.

The power clean, although a rather full-body functional exercise, does place significant stress on the AC joint during the "racking" phase. In this part of the exercise, the shoulders are shrugged, the elbows flexed, and then the shoulders are abducted to bring the bar up into a "racked" position. If the athlete is suffering from an AC joint injury, the power clean should be modified to allow only the pulling portion of the lift without racking the bar—an exercise termed a "power clean high pull" or "power pull." The key to this motion is that the athlete still gains a lower extremity benefit but avoids additional AC trauma that can be associated with a mistimed lift. The preferred way to perform the exercise is to adjust the exercise machine or starting position so that the elbows are even with or above the frontal plane when beginning the lift and during repetitions (honing technique) [28].

Dietary supplementation of multiminerals, glucosamine, and chondroitin sulfate has also

been recommended [26]. Alendronate (bisphosphonate) along with other conservative modalities [29] has been tried in a case report with successful resolution of symptoms and imaging findings. Conservative treatment options should be used as long as the patient will comply, with consideration of the patient's symptoms and functional status [26].

CT-guided injections of the AC joint allow for selection of location of best access points on the skin, appropriate depth, and inclination of the needle to inject local anesthetic mixed with depot preparations of corticosteroids. Conservative care stresses the use of NSAID and cryotherapy of the AC joint after all workouts [2]. In a sense, continued physical activity and pathogenesis will result in a "self-surgery"; that is, the clavicle will be resected on its own.

Appropriate educations should be guided to at-risk individuals like collision sports athletes and contact sports athletes; using proper techniques when falling, tackling and weight-lifting. Also it is important to ensure proper use and fit of protective equipment. Finally the rehabilitation program should be completed with strength and endurance training for the rotator cuff muscles, deltoid, trapezius, and other scapulothoracic muscles.

Even immediate immobilization after the injury in traumatic DCO does not seem to decrease the early manifestations of osteolysis. Early treatment may, however, decrease the total amount of bone loss, help decrease the time of the lytic phase, initiate repair, and decrease clinical symptoms [30].

22.6.2 Surgical Treatment

General indications for surgery include recalcitrant and isolated acromioclavicular joint pain with point tenderness of the AC joint, evident abnormal signs with AC joint imaging, lack of response to conservative treatment, and an unwillingness to give up or modify weight or sports training or manual labor [1, 5, 16]. Distal clavicle resection (DCR) is the most common type of surgery for DCO. Once patients have been selected strictly for surgery based on appropriate indications, decision must be made whether to perform open DCR or arthroscopic DCR and how much of the distal clavicle should be resected.

Both open and arthroscopic distal clavicle resection have been successful in alleviating pain and returning patients to previous activity levels [1, 31]. While Cahill et al. reported excellent results with an open approach resecting 1–2 cm of the distal clavicle, where 37 of 40 patients returned to weight training or competitive sports [1], Ague et al. reported that arthroscopic resection of only 4 mm was effective [21]. Arthroscopic DCR is a minimally invasive procedure with maximum visualization, associated with less surgical morbidity and early rehabilitation; associated intra-articular injuries can be diagnosed and treated simultaneously.

It can be difficult to determine whether pain after a history of trauma to the acromioclavicular joint is caused by osteolysis of the distal clavicle or by subtle instability. Therefore, a careful assessment of acromioclavicular stability is mandatory before recommending surgery. This should include translation testing of the distal clavicle in the anteroposterior and superoinferior planes, as well as careful scrutiny of preoperative radiographs for evidence of prior low-grade acromioclavicular separation. In this setting, arthroscopic distal clavicle resection is a reasonable initial treatment option with low morbidity. However, the patient should be warned that open stabilization of the acromioclavicular joint may be necessary should pain continue after arthroscopic resection of the distal clavicle.

22.6.2.1 Open DCR

The rationale of the open DCR is that the pathological articular surfaces can be resected under direct vision in order to create a wide enough margin to prevent further acromion abutment on the clavicle. Either of the two skin incisions can be used, the strap and the horizontal [2]. The junction of the deltoid and trapezius fascia must be split to provide proper exposure of the AC joint. Classic Mumford procedure is carried out; that is, 1–2 cm of the distal clavicle as well as the acromion is resected. The inferior AC joint capsule can be incorporated into the repair of the deltoid and trapezius fascia in order to eliminate any potential

dead space. Flatow and colleagues support the transferring of the coracoacromial ligament to cover the outer end of the clavicle in order to provide additional stability to the weight lifter [31].

The majority of follow-up studies have reported positive results when considering pain as a major indicator of success. Slawski and Cahill treated 12 active weight lifters and two manual laborers with open DCR. They reported that all patients returned to full sports activity and employment by an average of 9 weeks postoperatively and ultimately returned to a level of competition or productivity as good as or better than when they had been symptomatic. There were eight excellent and nine good results based on UCLA Shoulder Rating Scale [10].

Although the open procedure has been shown to produce good to excellent results clinically, the extensive tissue damage required to gain access to the AC joint has been linked to resulting muscle weakness [5, 32, 33] and disruption of the AC ligaments with abutment of the distal clavicle stump on the acromion with arm motion and instability of the AC joint with limited range of motion [31].

22.6.2.2 Arthroscopic DCR

Due to very obvious advantages of arthroscopic shoulder surgeries, it is not wise to compare arthroscopic DCR with open DCR and it is like comparing apples to oranges. Evidence has shown that 0.5–1.0 cm arthroscopic resections are comparable to the 1.5–2.0 cm resections performed during an open procedure [5, 34], suggesting optimal bone removal during arthroscopic DCR. There are two approaches—indirect (subacromial) or direct (superior). Both the approaches offer the advantages of decreased morbidity, with fewer postoperative restrictions on motion, earlier return to normal activity, and improved cosmesis. These early results are encouraging and warrant further examination of the treatment of this disease entity, an entity with a low prevalence as an isolated condition and which perhaps is becoming more prevalent in our society [21].

Subacromial (Indirect) Approach

The subacromial approach, first described by Ellman and Esch, preserves the superior AC joint ligaments and provides less chance for postoperative horizontal instability. It can be done in either lateral decubitus or beach chair sitting positions, depending on training and comfort of the surgeon. The technique uses anterior instrumental, posterior scope, and lateral inflow portals. A shaver is used to debride initially any obscuring bursa. Electrocautery is used to clearly demarcate the distal clavicle and minimize bleeding. Great care should be used not to disrupt the supporting ligaments and capsule. Once good visualization is obtained, a burr (usually 5–6 mm) is used through the anterior portal to clear any remaining osteophytes and to resect the distal clavicle from anterior to posterior. Modifications of this technique include burring from both the posterior and the lateral portals, as well as visualization through the three standard portals. Bone depth can be gauged using the known diameter of a burr; however, Tolin and Snyder recommend the routine use of two needles to demarcate the orientation of the joint, as well as to gauge the amount of bone resected, by measuring the distance between the two needles on the skin. Although some investigators recommend resecting a small portion of the medial acromion, most find it unnecessary. To aid in resection of the superior portion of the distal clavicle, manual pressure can be applied to bring the clavicle into the subacromial space. It has been suggested that failure of this technique is not due to the amount of bone removed but rather the result of uneven resection or disruption of the AC ligaments. This would lead to translation of the clavicle, resulting in an abutment on the acromion and cause recurring symptoms [35, 36]. This problem often happens after aggressive arthroscopic resection when care is not taken to preserve the stabilizing ligamentous envelope. Morrison and colleagues recommend beveling the posterior edge of the distal clavicle if this instability is recognized intraoperatively to avoid the resultant painful impingement [37].

Kay et al. treated ten patients with DCO in lateral decubitus position with traction. Distal clavicle resection was done via a bursal approach in conjunction with subacromial decompression. All patients had satisfactory outcome and returned to their sports at or above their pre-injury

level [38]. It has often been believed that the bursal approach may not allow easy access of the clavicle in a tight joint with medial inclination, especially in osteoarthritic joints, even with direct superior pressure on the clavicle [39]. An open incision has been proposed for these cases by surgeons favoring the bursal approach. Tolin and Snyder believed they overcame this problem by using a lateral position with 10–15 lb traction on a 70° abducted arm [40].

Superior (Direct) Approach

First described by Lanny Johnson and later championed by Flatow and associates [35], the superior approach offers a direct approach to the AC joint. While some investigators recommend routine arthroscopic examination of the subacromial space for potential pathology, others believe that there is no reason to violate the bursa in isolated AC problems [35]. A superior approach also allows resection of the outer end of the clavicle under direct visualization, without the edema and bleeding of a bursal approach. Two small-bore needles (22 gauge, 1.5 in.) are used to determine the location and orientation of the joint so as to allow precise introduction of the instruments. This is critical, because otherwise variations in joint inclination may be hard to appreciate. A 4.0 mm 30° arthroscope and necessary instruments are placed into the AC joint via direct anterosuperior and posterosuperior portals. A 2.7 mm arthroscope may be placed initially if the joint space is narrow. The capsule and ligaments of the AC joint are subperiosteally elevated to expose the distal clavicle, allowing direct visualization of the clavicle during the resection. The meniscus and intra-articular soft tissues are resected with a 5.0 mm motorized full-radius resector. An electrocautery unit may be used to shell out the outer end of the clavicle in order to preserve the soft tissue containing the AC ligaments and capsule. After this, if the joint space is large enough, a 6.0 mm burr is introduced. If the space is too small, more room can be created using smaller burrs first until the 6.0 mm burr can be accommodated. Approximately 4–7 mm of the distal clavicle is removed. After resection, the joint should be carefully examined arthroscopically from both the anterior and posterior portals to ensure adequate bone removal and to check for loose fragments. It is essential to probe the edges to be sure that no overhanging ridges remain [35, 41].

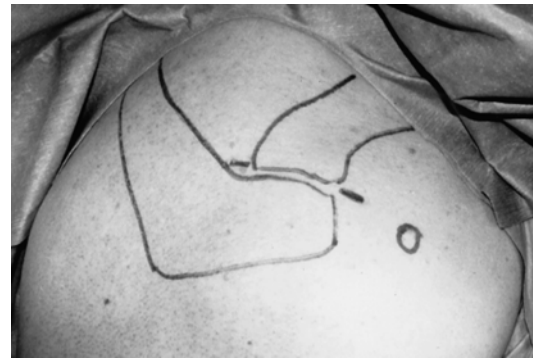

Fig. 22.5 Skin markings outlining the distal clavicle, acromion, acromioclavicular joint, and coracoid process are made. The anterosuperior portal is made 3–5 mm anterior and in line with the acromioclavicular joint. The posterosuperior portal is made posterior to and in line with the acromioclavicular joint in the notch formed by the acromion and distal clavicle

This approach preserves joint stability and prevents excessive posterior translation and painful abutment of the distal clavicle against the acromion (Fig. 22.5).

Flatow et al. reported a 91 % success rate with the superior approach [35], while Zawadsky et al. determined that all results of superior arthroscopic DCR were either good or excellent [41]. Bigliani et al. [42] have proposed a more limited resection of the clavicle to improve cosmesis and function. Limited (less than 1–2 cm) arthroscopic DCR, an average of 4.5 mm specifically in weight lifters, has shown promising results [21]. The surgical approach consists of a superior arthroscopic approach to the AC joint with two portals [42]. Standard arthroscopic instrumentation (30° camera and 4.0 mm arthroscope), an arthroscopic shaver, and a 4 mm motorized burr should be used. The AC joint is debrided of material such as meniscal remnants and cartilaginous debris. The distal 4 mm of the clavicle is resected with the burr, using the diameter of the burr as a guide. The outer cortical shell of the distal clavicle is addressed with the burr after elevating the capsule from the clavicle with electrocautery, sparing the superior AC ligament. A rasp can be used through the portals to complete the distal clavicle contouring [21].

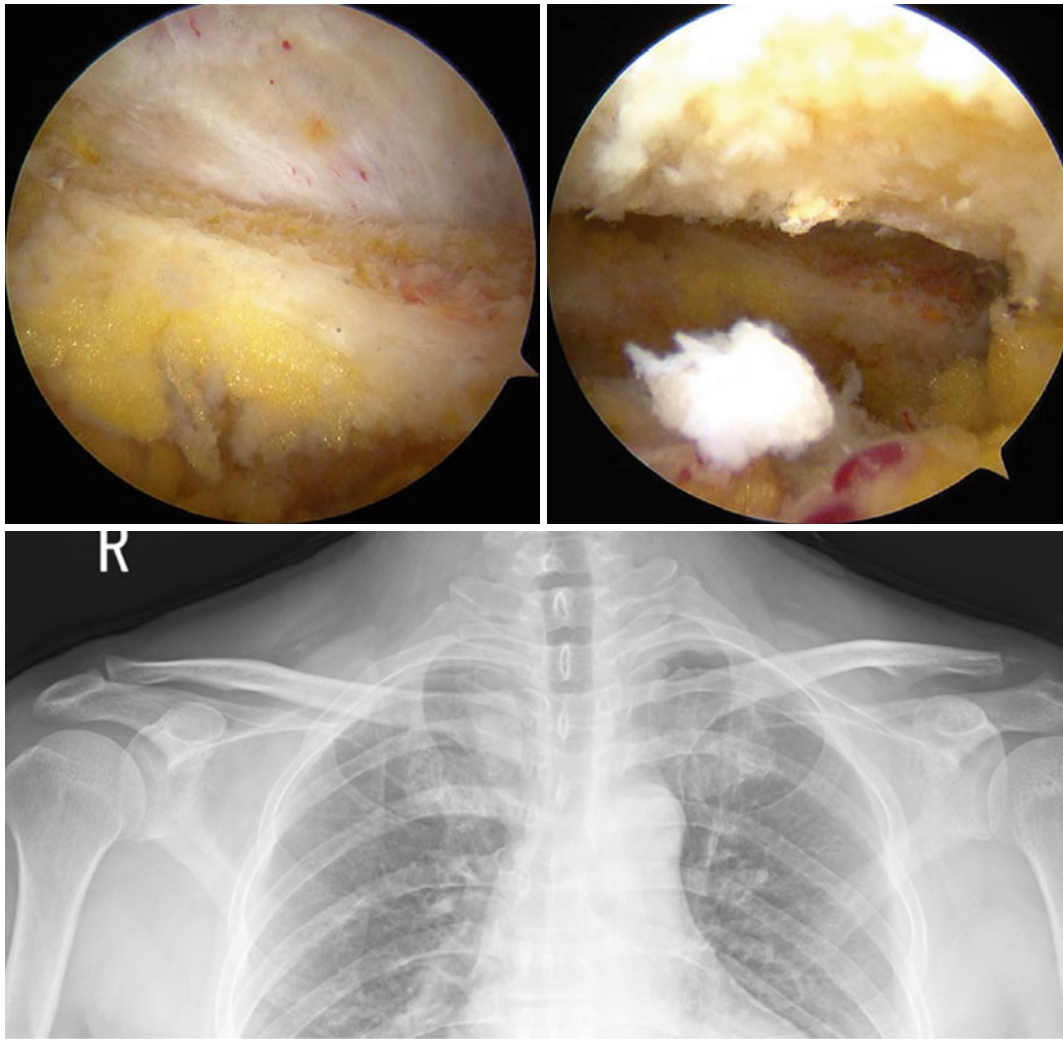

Fig. 22.6 Arthroscopic view and postoperative X-ray of distal clavicular resection

Branch et al. showed that a 5 mm resection is adequate (Fig. 22.6).

Branch et al. showed that a 5 mm resection is adequate to prevent bony abutment in both rotationally and axially loaded shoulders if the coracoclavicular and acromioclavicular ligaments are intact [43].

22.7 Author-Preferred Treatment and Literature Review

Song et al. treated a series of 17 patients diagnosed with painful residual instability resulting from conservative management of grade II AC joint injuries over a period of 8 years, with an average injury-surgery interval of 12 months treated with arthroscopic DCR. In the initial four cases, subacromial bursal approach was used, and in the rest of the patients, superior approach was done using 2.7 mm arthroscope. There was radiological evidence of DCO in 4 patients. Two of 17 patients (11.8 %) required additional surgery because of persistent pain and further instability. Of the remaining 15 patients, 11 showed good to excellent subjective results (visual analog scale and constant shoulder score) at final follow-up, whereas 4 judged the result to be fair or poor. For subjective satisfaction, 6 of 17

patients (35.3 %) rated a poor result. However, none of the patients showed any demonstrable clinical instability or symptoms at the time of final follow-up. They concluded that arthroscopic distal clavicle resection statistically improved the pain score and constant score and seemed to be a reasonable initial treatment option with lower morbidity [44]. All these previous works show the efficacy and safety of arthroscopic DCR.

Ague et al. treated ten weight lifters with mean age of 30.4 years suffering from isolated ADCO. Follow-ups at an average of 18.7 months of limited DCR done on outpatient basis showed that they can resume their training within the first week postoperatively (average, 3.2 days; range, 1–6 days). Preoperative training levels can be reached by the second week postoperatively (average, 9.1 days; range 7–12 days), and all of them remained asymptomatic. Very few patients will lose strength in the military press or the incline press. At final follow-up, the radiographic appearance of the distal clavicle had not changed [21].

In a systematic review of English literature published in peer-reviewed journals, Rabalais R. David and McCarty Eric concluded that arthroscopic DCR has provided more good or excellent results than has the open DCR but is comprised of level III or IV evidence consisting largely of retrospective case series [45]. Again in another systematic review of literature, Michael Pensak et al. concluded that patients treated with arthroscopic superior (direct) ACDR can be expected to have a faster return to activities while obtaining similar long-term outcomes compared with the open procedure [46].

Although distal clavicle resection has been shown to be a successful procedure, some failures have been reported. One of the least recognized reasons of failure may be heterotopic bone formation. Thus, most investigators recommend removal of all bone and fragments within the joint in order to avoid a nidus for new bone formation. Berg and Ciullo suggested that it might be a more common cause of failure of both acromioplasty and distal clavicle resection [47]. They suggested the use of prophylactic measures with patients considered at risk like long-standing smokers and chronic pulmonary diseases with hypoxemia.

Resection of the distal clavicle and disruption of the AC articulation create the potential for instability of the distal clavicle. Blazar et al. evaluated 17 isolated distal clavicle patients (open and arthroscopic) and discovered that the average anterior plus posterior translation was 8.7 mm (range, 3–21 mm), which was significantly greater than the contralateral shoulders (mean, 3.2 mm; range, 1–6 mm). The amount of pain determined by a questionnaire, correlated with the amount of translation and showed that excessive anteroposterior instability of the distal clavicle can cause postoperative pain and lead to poor surgical outcomes [48]. As suggested by Branch et al. [43], instability may cause postoperative symptoms by means of increased tension on the soft tissues of the acromioclavicular joint and trapezius attachment, not by continued bony abutment. This is supported by the fact that the presence of instability can also produce unsatisfactory results in open procedures where a larger resection is performed. The complications of DCR other than instability include underlying muscle injury, excessive bleeding, lateral clavicle fracture, and infection.

22.8 Summary

Distal clavicle osteolysis is a unique disease most likely due to an overuse phenomenon. When activity modification and conservative treatment fails to provide relief in an active patient, distal clavicle resection has provided good results. In isolated DCO, there is scarcely any indication for an open procedure, while the superior and subacromial approaches have their pros and cons. The subacromial approach offers certain advantages, including (1) assessing for other pathology or working through established portals if other pathology is already being addressed, (2) less injury to the capsule, and (3) no need for smaller instruments. Disadvantages include (1) violating an area with potentially no pathology, (2) more portals, and (3) more bleeding and fluid extravasation. The merits of a direct approach should not be discounted.

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Acromioclavicular Joint Problems in Athletes

23

Yon-Sik Yoo

23.1 Introduction

The injury has a very interesting background. Hippocrates (460–377 BC) said that physicians at the time were very liable to be deceived by the injury taking it to be glenohumeral injury. Galen (129–199 AD) diagnosed his own acromioclavicular joint injury which he obtained from wrestling. He tried treating himself in the manner of Hippocrates by tight bandages holding the clavicle down but was not able to continue it due to the discomfort [1]. It is apt to say that one of the oldest cases of acromioclavicular joint injury was related to sports.

Acromioclavicular (AC) joint injuries are very common and occur usually in people in their second and third decade, especially in active sporting individuals. Athletes and contact sports players, in particular, are more prone to getting these injuries relating to the mechanism which causes the injury. Acromioclavicular joint afflictions account for approximately 9 % of all shoulder injuries [2] and are as high as 40–50 % of athletic shoulder injuries [3, 4]. These injuries are overwhelmingly more common in men than in women and the male to female ratio is 5:1. More often these dislocations are incomplete. The problem statement can be estimated from the fact that a study dealing with the pattern of injuries in ice hockey teams described the acromioclavicular injury as the third most common injury after concussions and knee medial collateral ligament sprains [5]. Also worth mentioning is that approximately 15 % of injuries in the study were inflicted upon shoulder joint which was again the third most common body part affected after the knee and leg (together) and head.

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23.2 Pathoanatomy, Biomechanics, and Preferred Classification

23.2.1 Joint Anatomy and Biomechanics

The acromioclavicular joint is a diarthrosis or a synovial joint permitting free movements. The joint permits rotations as well as translation in the anteroposterior as well as superioinferior planes. It is surrounded by a joint capsule with synovium and has an articular surface made up of hyaline cartilage containing and an intra-articular disc which is a meniscus-type structure [2]. Many studies have demonstrated that this structure involutes with age and is completely degenerated by 40 years of age [6–8]. The actual function of this meniscoid structure in the joint is negligible. The joint is innervated by the lateral pectoral, suprascapular, and axillary nerves.

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The articular surface of the AC joint line is oblique and slightly curved. The curvature of the joint permits the acromion to glide forward or backward over the lateral end of the clavicle. This movement of the scapula keeps the glenoid fossa continually facing the humeral head. The oblique nature of the joint is such that forces transmitted through the arm will tend to drive the acromion process under the lateral end of the clavicle with the clavicle overriding the acromion. The joint is important because it contributes to total arm movement in addition to transmitting forces between the clavicle and the acromion. The acromioclavicular joint has three degrees of freedom. Movement can occur between the acromion and lateral end of the clavicle, about a vertical axis, around a frontal axis, and about a sagittal axis.

Multiple studies have concluded that the clavicle rotates approximately 40–45° with full shoulder abduction and elevation; however the movement is only 5–8° as reported by Rockwood et al. when compared with the acromion because of the synchronous scapula-clavicular motion. Although the rotation motion at the AC joint is still being debated, a principal rotational function of the AC joint in the abduction of the arm is to permit continued lateral rotation of the scapula after about 100° of abduction when sternoclavicular movement is restrained by the sternoclavicular ligaments.

Other important functions of the acromioclavicular joint are transmission of force from the appendicular skeleton to the axial skeleton and suspension of the upper extremity.

The acromioclavicular joint is stabilized by both static and dynamic stabilizers. The static stabilizers are the AC joint capsule and the ligamentous supports to the AC joint which include the acromioclavicular (AC) ligament, the coracoclavicular (CC) ligament, and the coracoacromial (CA) ligament.

Dynamic stabilization is provided by the muscles – the origin of the deltoid muscle from the clavicle and the trapezius through its fascial insertion into the acromion.

The AC joint capsule and the AC ligament are the primary constraints to anteroposterior translation of the AC joint [9]. The AC ligament is comprised of four parts – superior, inferior, anterior, and posterior. Of these the posterior and the superior AC ligaments together are the main contributors to the horizontal stability of the joint. These (posterosuperior AC ligaments) are the strongest and are invested by the deltotrapezial fascia.

The coracoclavicular ligaments are the primary vertical stabilizers of the AC joint. The coracoclavicular ligaments are comprised of the conoid and the trapezoid ligaments. These two components, functionally and anatomically distinct, are united at their corresponding borders. In up to 30 % of subjects, these bony components may be opposed closely and may form a coracoclavicular joint. These ligaments suspend the scapula from the clavicle and transmit the force of the superior fibers of the trapezius to the scapula.

The normal span of the CC ligaments (coracoclavicular space) is 1.1–1.3 cm as reported by Bearden et al. [10]. Fukuda et al. [9] advocated that the conoid ligament was the primary constraint to the superior displacement of the clavicle and the trapezoid ligament was found to be the primary restraint to the compression of the AC joint. A number of studies have been done on the function of conoid and trapezoid ligaments and have found the conoid ligament to be extremely important and the structure to be meticulously repaired in a case of acromioclavicular dislocation with coracoclavicular ligament disruption [9, 11].

23.2.2 Mechanism of Injury

Acromioclavicular injuries are very common in athletes especially those involved in contact sports. The usual mechanism of injury is direct trauma to the point on the shoulder and infrequently indirect trauma. The classical history for an injury to the acromioclavicular joint is the force applied on the acromion with the arm held in a position of adduction.

A direct injury results from a direct force to the acromion with the shoulder adducted, resulting in movement of the acromion inferiorly and medially while the clavicle is stabilized by the

sternoclavicular joint ligaments [12]. This mechanism is involved in most injuries and is usually the result of a fall on the superolateral portion of the shoulder. The force results in systematic failure of the stabilizing ligaments with the propagation of increasing force. Failure of the AC ligaments and capsule is followed by failure of the CC ligaments and deltotrapezial fascia. Indirect force injury is less common. Indirect injury occurs by falling on adducted outstretched hand or elbow where the humerus translocates superiorly pushing the humeral head into the acromion. The subcutaneous position of the joint with very little muscle coverage makes it prone to injuries. In this situation, the coracoclavicular ligaments are usually not damaged because of a decrease in the coracoclavicular space.

23.2.3 Classification

The classification of the acromioclavicular joint dislocations was initially given by Tossy et al. [13] wherein they had classified the injury into three types – type I, II, and III. However to this classification, modification was done by Rockwood [14] wherein they added three more grades of injuries – type IV, V, and VI – in the year 1984. This classification encompasses all the injuries ranging from acromioclavicular ligament sprains to frank and severe acromioclavicular joint dislocations with the rupture of the acromioclavicular ligaments, coracoclavicular ligaments, as well as the trapezial and deltoid fascia. It is the most commonly used classification of acromioclavicular injuries (Fig. 23.1, Table 23.1).

23.2.3.1 Type I

Grossly type I injuries represent minor strains of the acromioclavicular ligament and joint capsule. Type I injuries are synonymous with grade I injuries. These injuries commonly result from direct force to the shoulder. The AC ligaments and the coracoclavicular ligaments are both intact although the AC ligaments are sprained. The deltotrapezial fascia is intact. Pain is minimal. The AC joint is stable and the radiographs at the time of injury are negative though periosteal calcification at the distal end of clavicle may be apparent later. The treatment is essentially conservative.

23.2.3.2 Type II

More significant forces cause type II or grade II injuries. In this type of injury the acromioclavicular ligaments are disrupted but the coracoclavicular ligaments are intact. However some degree of sprain of the CC ligament is present. The deltotrapezial fascia is also intact. AC joint instability is present especially in the anteroposterior plane. Vertical stability is present due to the intact coracoclavicular ligaments. Considerable pain and tenderness are present. The radiographs show slight elevation of the clavicle as compared to the acromion, even on stress x-rays. Due to some element of medial rotation of the scapula at the AC joint, there can be widening of the AC joint. The deformity and the instability become apparent on application of stress. These injuries are also managed conservatively with good results and only sparingly needing surgery.

23.2.3.3 Type III

This injury is characterized by rupture of both the acromioclavicular and the coracoclavicular ligaments. These are caused by forces which are strong enough to cause injury and disruption of both ligaments. Literature mostly says that the deltoid and trapezius muscles are intact and there is no significant disruption of the deltoid or trapezial fascia. Pain is severe on movements. The acromioclavicular joint is disrupted and the clavicle is displaced superiorly. There is gross instability of the acromioclavicular joint in both horizontal and anteroposterior planes. The stress views demonstrate that the distal clavicle is separated from the acromion and displaced superiorly. The radiographs show 25–100 % increase in the coracoclavicular space as compared to the normal side. The superior displacement of the clavicle is due to inferomedial drooping of the shoulder complex and the scapula. There are two schools of thoughts on the treatment of these injuries. Some surgeons advocate operative treatment but many orthopedicians give trial of a conservative treatment and go for surgery only when there are residual or persistent symptoms after

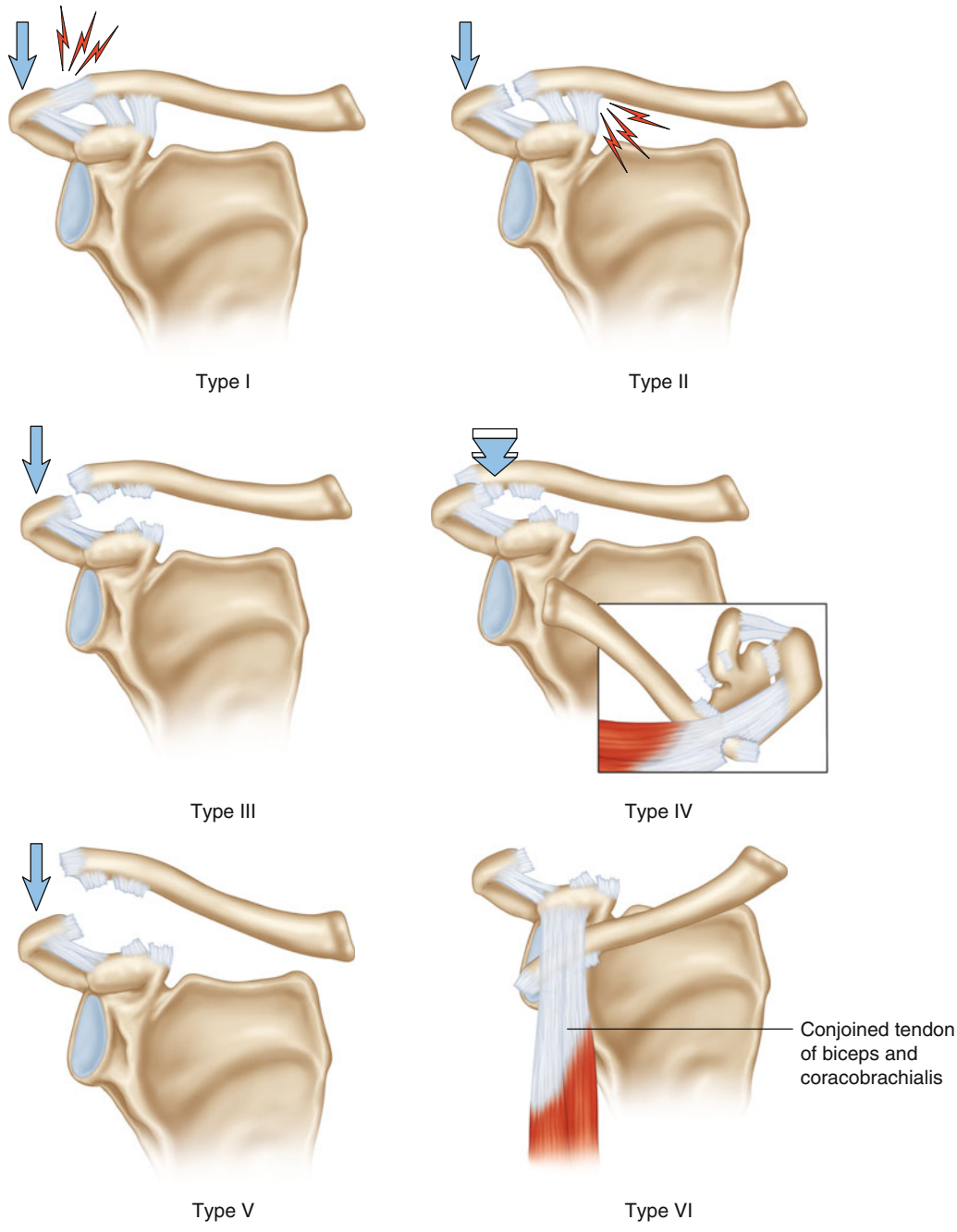

Fig. 23.1 Acromioclavicular joint injuries. *Type I*: AC sprain, few fibers torn. *Type II*: disruption of the acromioclavicular ligaments with coracoclavicular ligaments intact. *Type III*: disruption of the AC and coracoclavicular ligaments. *Type IV*: disruption of both ligament complexes with posterior clavicular displacement. *Type V*: disruption of both ligament complexes with marked superior clavicular displacement. *Type VI*: disruption of the ligament complexes with anterior entrapment beneath the coracoid

Table 23.1 Injury pattern of acromioclavicular joint according to the Rockwood classification

Type of injury	AC joint	AC ligament	CC ligament	Deltoid and trapezius muscles	Displacement of the clavicle
Type I	Intact	Sprain	Intact	Intact	Undisplaced
Type II	Unstable in horizontal direction	Torn	Sprain/intact	Intact	Slight superior displacement
Type III	Disrupted	Torn	Torn	Usually intact	Superior displacement
Type IV	Disrupted	Torn	Torn	Detached	Posterior displacement
Type V	Disrupted	Torn		Detached	Severe superior displacement with more than 100 % increase in the coracoclavicular space
Type VI (rare) Subcoracoid	Disrupted	Torn	Torn	Variable	Inferiorly
Type VI (rare) Subacromial	Damaged partially or completely	Torn	Intact	Variable	Inferiorly

3–6 months. The physical status and the patient demands are also an important factor in deciding the treatment of these injuries. The author's preferred treatment is also conservative initially with a close watch on the condition. In athletes also the same protocol is practiced by the author as the rehabilitation phase is very difficult in athletes after surgery.

23.2.3.4 Type IV

This injury is characterized by posterior displacement of the clavicle through the trapezius muscle. The force acting on the acromion drives the scapula anteriorly and inferiorly causing the posterior displacement of the clavicle. Both the acromioclavicular and the coracoclavicular ligaments are torn. The trapezial and the deltoid fascia are disrupted with the detachment of the deltoid and trapezius muscles. The clavicle may tent the posterior skin sometimes. The AP x-rays may be misleading as they may appear normal, although the axillary x-rays demonstrate the posterior displacement of the clavicle. CT scan is often required for delineation of these injuries. An important point to be noted with these injuries is that they are associated with the anterior displacement of the sternoclavicular joint. Thus in every case of type IV acromioclavicular injury, the sternoclavicular joint should be evaluated and imaging done. These injuries are relatively rare.

23.2.3.5 Type V

These injuries are severe forms of type III injuries when the deforming force is of a high amplitude. The acromioclavicular and the coracoclavicular ligaments are disrupted and the acromioclavicular joint is extremely unstable in both directions. The deltoid and the trapezius muscles are detached. The clavicle goes superiorly and the displacement is extreme. There is severe superior migration of the distal clavicle due to the unopposed action of the sternocleidomastoid along with drooping of the shoulder complex and scapula leading to marked disfigurement of the shoulder. The radiographic coracoclavicular distance is increased more than 100 % in comparison to the normal side. Some authors have suggested that there is a change in acromioclavicular distance of 100–300 % on radiographs [2] as compared to 25–100 % increase in the distal acromion-clavicle distance as seen in type III injury.

23.2.3.6 Type VI

These injuries are extremely rare. Gerber and Rockwood [15] have reported three cases and this series is the largest one reported in the literature.

The injury is characterized by inferior dislocation of the clavicle. The injury represents severe trauma and is frequently associated with a number of other injuries. The injury is thought to be caused by hyperabduction and external rotation of the arm along with retraction of the scapula. The clavicle is inevitably found in either a subacromial or subcoracoid position. The ligament status and muscle injury depend on the displacement of the clavicle. In a subcoracoid position both the acromioclavicular and the coracoclavicular ligaments are disrupted and there is variable degree of damage to the deltoid and the trapezius muscles. The clavicle dislodges behind an intact conjoint tendon. In the subacromial position the acromioclavicular ligaments are torn but the coracoclavicular ligaments are intact. Most patients have associated paresthesia with the injury which resolved on relocation of the clavicle.

23.3 Clinical Presentation

The main symptom is pain and the main sign is tenderness located at the AC joint though swelling and deformity often accompany pain and tenderness. Any attempt to move the shoulder causes pain. The pain and tenderness increase with increase in the grade of dislocation. Abnormal protuberance of the distal end of the clavicle can be found with grade III or grade V acromioclavicular joint dislocations. Instability in the horizontal plane as well as the vertical plane can be assessed depending upon the grade of dislocations (described with the grades of dislocations) (Fig. 23.2).

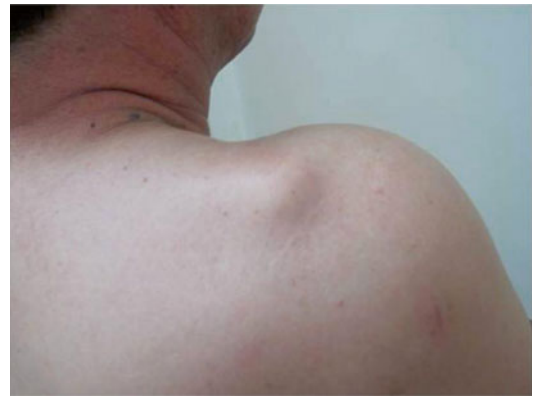

Fig. 23.2 Clinical photograph of a patient with an acromioclavicular dislocation

23.4 Essential Radiology

23.4.1 The AP View and the Zanca's View

The imaging of the acromioclavicular joint is slightly tricky. The x-rays taken for the shoulder usually have a high penetration for proper visualization of the glenohumeral structures. However the acromioclavicular joint gets overpenetrated in the process and is improperly visualized or seen more dark. In order to have a proper visualization of the AC joint, the penetration of the beam or the voltage is reduced by 50 % when compared to the x-ray being taken for the glenohumeral joint.

The other point of importance is that in a normal AP x-ray the distal clavicle and the acromion are superimposed by the spine of the scapula and proper visualization is not present. Zanca evaluated 1,000 x-rays of patients with shoulder pain and finally recommended the Zanca's view in which the x-ray beam was given a 10–15° cephalic tilt to give an unobscured view of the acromioclavicular joint.

Also important is the comparison with the normal side to be certain not to miss the subtle changes indicative of an acromioclavicular injury. So it is recommended to take both the acromioclavicular joints imaged on the same film.

23.4.2 Lateral View/Axillary View

Only AP or Zanca's views are not enough. Subtle anteroposterior displacements may be detected on lateral/axillary views. So it is always recommended to include the axillary views when imaging a case of suspected acromioclavicular dislocation. Also type IV injuries where the clavicle is displaced posteriorly may only be detected by the axillary views as the anteroposterior views may appear surprisingly normal.

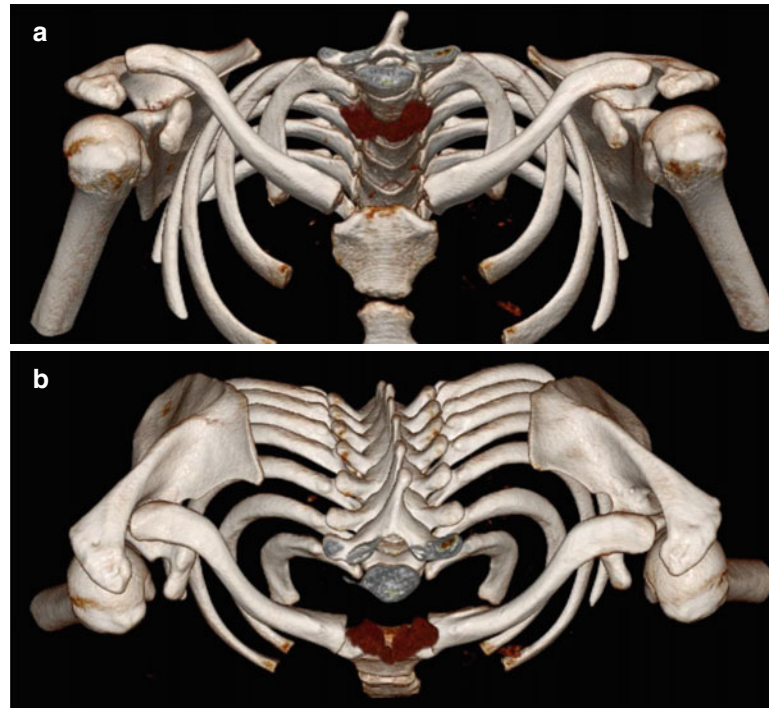

Fig. 23.3 (**a**) CT scan view with a 3-D image showing a right-sided acromioclavicular joint dislocation with superior migration of the clavicle. (**b**) CT scan top view with a 3-D image showing a right-sided acromioclavicular joint dislocation with posterior displacement of the clavicle on the right side

23.4.3 Stress Views

With modern imaging and CT scans available, a lot of orthopedic surgeons do not prefer the stress views nowadays. The stress views have been conventionally used to differentiate between incomplete and complete AC joint disruptions (type II and type III injury). They are taken with a weight of 10–15 lbs (4.5–6.8 kg) that are suspended from both wrists of the patient and the AP x-rays of both sides AC joints taken and compared. In significant subluxations or dislocations, the lateral end of the clavicle is displaced superiorly. However the stress views cause significant discomfort to the patient and rarely provide any additional information. So they are rarely used nowadays.

23.4.4 Stryker Notch View

This view is taken with the patient supine and the hand of the patient is positioned on top of his/her head. X-ray beam is directed 10° cephalad centered on the coracoid process. This x-ray is able to give the best coracoid profile and identify all coracoid fractures which may be associated with AC joint dislocations. This image is to be taken in suspicion of a coracoid fracture when the AP projection shows an acromioclavicular dislocation but the coracoclavicular distance is normal [16] or comparable to the uninvolved opposite side.

23.4.5 CT Scan

A 3-dimensional CT scan is the preferred imaging technique for AC joint evaluation by the author. The 3D computed tomographic scan is very sensitive and specific in the detection of all acromioclavicular injuries, dislocations, and other pathologies. All displacements of the lateral end of the clavicle – superior, posterior, subcoracoid, subacromial, or inferior – can be easily delineated by a CT scan (Fig. 23.3).

23.5 Treatment Options

The treatment of acromioclavicular joint injuries varies according to the severity or grade of the injury and patient requirement.

The objective of treatment – operative or nonoperative – is to attain a pain-free shoulder with full range of motion, full power, and no limitation of activities. The demands differ from the general population to athletes and recreational athletes to professional athletes and these demands play an important role in deciding the management of the injury. However, there are few peer-reviewed studies of the treatment of AC joint injuries in athletes. No prospective studies compare the operative and nonoperative treatment in type III AC injuries in the group of patients. Thus there are no absolute indications for either type of treatment of this injury in athletes.

23.5.1 Nonoperative Treatment

Nonoperative treatment is almost always the rule for type I and type II injuries. General consensus is towards the conservative treatment of these injuries set aside special circumstances. For type I injury rest and immobilization in simple sling, strapping, or shoulder immobilizer for 1–2 weeks with ice application and pain management by NSAIDs lead to resolution of discomfort. For type II injuries this time is slightly longer and the immobilization is continued for 2–3 weeks with symptomatic and supportive treatment. Full return to activities is not started till the patient has resumed full range of painless motion. Sports activities can be resumed when all the symptoms have resolved and this is generally 6–8 weeks and this duration is even longer for type II injuries. Operative intervention is left for those who have persistent symptoms or unfavorable outcome with conservative treatment. However there has been increasing consciousness about the outcome of type I and type II injuries with conservative management. Moushine et al. [17] in their study had found out that 27 % of the conservatively managed type I and type II AC joint separations required further surgery at 26 months of injury. Also voices have been frequently raised regarding scapular dyskinesis arising as a sequelae to acromioclavicular joint dislocations [18]. With all the other facts taken into consideration, conservative management still remains the treatment of choice for type I and type II injuries.

Nonoperative treatment of the type III injuries entails the use of sling, ice, and analgesics. The sling can be discontinued and light activities resumed when pain subsides. Return to sports activity is allowed when there is full strength and range of motion of the injured shoulder. This may take 8–12 weeks.

23.5.2 Surgical Management

Surgical management is kept reserved for high-grade AC joint dislocations or the dislocations which fail to show improvement with conservative management. Generally type IV, V, and VI dislocations are almost always managed operatively and previously there was a split in the management of grade III dislocations. Many surgeons including the author currently prefer giving a conservative trial for the management of type III AC joint injuries and then turn to operative management in case of no improvement or inadequate outcome. This also holds true for the athletes. Athletes in a type III acromioclavicular injury are almost always managed by conservative treatment including sling, ice application and NSAIDS. McFarland et al. [19] in the year 1997 had published the results of survey of major league baseball physicians, evaluating the treatment modalities for these injuries (type III acromioclavicular dislocations) in pitchers. He had found that 69 % of physicians went for nonoperative treatment. Additionally the results of nonoperative treatment was comparable with the results obtained with the operative treatment with 80 % of the nonoperatively managed athletes having complete pain relief and normal function.

Also take into account the problems in athletes with operative treatment. The time for rehabilitation is long and mostly involves restoration of the coracoclavicular distance by means

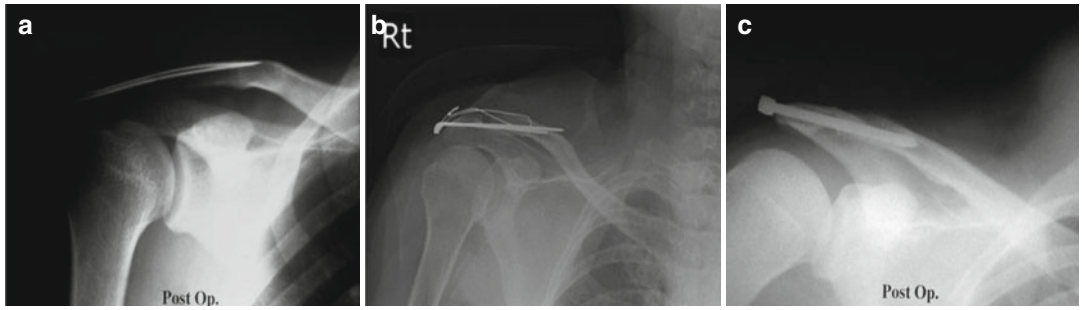

Fig. 23.4 Different methods of primary fixation of the AC joint. *From left to right*: (**a**) AC joint fixed with K-wires, (**b**) AC joint fixed with tension band wiring, (**c**) AC joint fixed by Knowles pin insertion

of tightrope fixation or graft fixation by making tunnels into the clavicle and the coracoid process. Because of the increased functional demands in the athletes and the increased stress on the acromioclavicular joint, there is high risk of pathological fracture through the tunnels. Also the general complications with any surgery including infection, stiffness, noncompliance for rehabilitation, etc. can also lead to altered results. So currently the treatment of choice for type III AC joint dislocation in an athlete is conservative.

There are a number of operative procedures described for the treatment of AC joint dislocations. The list is endless and growing fast. We will here discuss only the important and the most commonly practiced surgical techniques with a special mention of the author's preferred technique.

All techniques have the common goal of obtaining and retaining anatomic joint reduction. The main principles of surgical treatment include:

1. Restoration of the coracoclavicular (CC) interval
2. Restoration of the congruity of the acromioclavicular joint
3. Anatomic reconstruction of the ligaments

23.5.2.1 Primary Fixation of the AC Joint

Primary fixation of the AC joint has been historically done with either smooth pins or threaded pins or K-wires spanning the acromioclavicular joint. The procedure has virtually been given up now because of the catastrophic side effects seen with the pins. The pins migrated and were found all over the viscera – in the lungs, the heart, the major vessels, etc. [20, 21] (Fig. 23.4).

However an alternative technique is very frequently done across the world, in Europe and Asia in particular. This is the hook plate placement. The hook plate is spanned across the joint with the hook going just under the acromion and levering the clavicle downwards and fixed to it by *screw*s. Many surgeons also advocate ligament reconstruction along with hook plate application. People have obtained good results with the hook plate. But there are both the dark sides and the bright sides attached to it. The advantages of the hook plate include relatively easy implantation procedure and early postoperative mobilization. However, known complications of the hook plates are very severe and include recurrent dislocations [22] by fixation failure, distal clavicle stress fracture medial to the plate, and skin/wound complications [23]. Folwaczny et al. [24] noted only a 63.2 % patient satisfaction rate postoperatively. The main disadvantage is the necessity of implant removal and more so before mobilization at approximately 2–3 months. Common complications are infections, plate bending, slippage of plate, clavicular fractures, and migration of hook into the subacromial bone.

The major disadvantage with the primary fixation of the AC joint is that it is an unnatural fixation and theoretically would disturb the biomechanics of the shoulder movements.

Even with all the discussion the hook plate remains the first choice implant for many surgeons and institutes for AC joint dislocations.

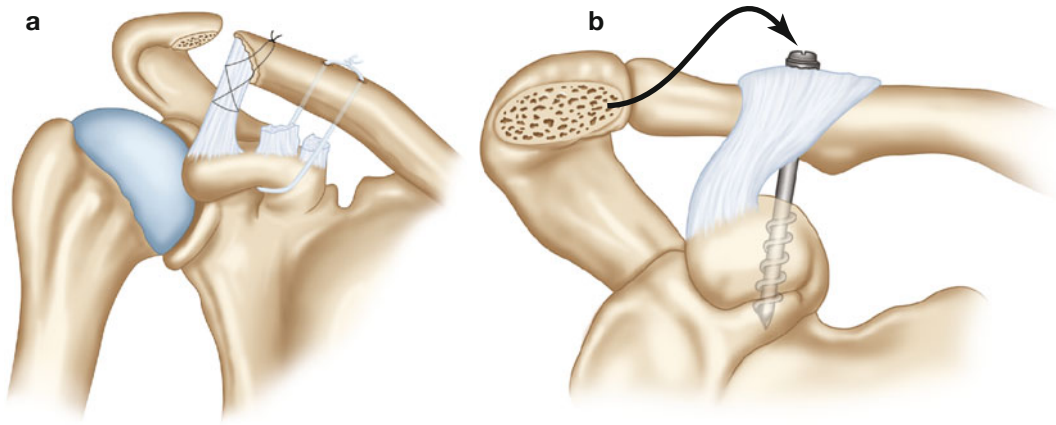

Fig. 23.5 (**a**) Weaver-Dunn procedure with resection of the distal clavicle and transfer of the coracoacromial ligament to the medullary canal of the clavicle. The construct has been augmented with a suture loop around the clavicle and coracoid. (**b**) Transfer of the coracoacromial ligament from acromion to clavicle and augmenting it with a coracoclavicular screw

23.5.2.2 Ligament Reconstruction

Weaver and Dunn [25] were the first to describe the coracoacromial ligament transfer to the clavicle. In their procedure they combined resection arthroplasty of the acromioclavicular joint with fixation of the distal end of the clavicle by suturing the acromial end of the shortened coracoacromial ligaments into the medullary canal of the clavicle. This construct can be augmented with a suture loop or screw that provides protection while the reconstructed ligament heals (Fig. 23.5).

An alternative technique for ligament reconstruction is the use of a semitendinosus tendon autograft. This technique is combined with resection of the distal clavicle. Jones et al. [26] described the use of a looped semitendinosus graft around the coracoid process and clavicle in a revision AC joint reconstruction. Yoo et al. [27] recently have described a new arthroscopically assisted mini open double-bundle, three-tunnel method for anatomical reconstruction of CC ligaments using a semitendinosus tendon autograft. Biomechanical testing of this construct has been favorable (Fig. 23.6).

Recently Lafosse et al. [28] described all arthroscopic techniques for CA ligament transfer in the setting of acute or chronic dislocations.

23.5.2.3 Fixation Between the Coracoid Process and Clavicle

Coracoclavicular screw fixation is an old technique which has been used for a very long time in the treatment of acromioclavicular separations. Bosworth [29], as early as 1941, popularized this technique and in many places it is still quite a popular technique for treatment of AC joint separations.

The screw was placed by percutaneous technique, using local anesthesia and fluoroscopic guidance. With the patient in sitting position, a stab wound was made on the superior aspect of the shoulder, approximately 3.8 cm medial to the distal end of the clavicle. A drill hole was made in the clavicle. After this an assistant reduced the AC joint by depressing the clavicle and elevating the arm using a special clavicle-depressing instrument. An awl was then used to develop a hole in the superior cortex of the base of the coracoid process, which was visualized using fluoroscopic imaging. A regular bone screw was inserted. Recently, a 3.5 or 4.5 mm cancellous screw is used with a washer. Originally, the screw was recommended to leave indefinitely unless specific indications emerged for removal developed. Bosworth did not recommend either repair of the CC ligaments or exploration of the AC joint.

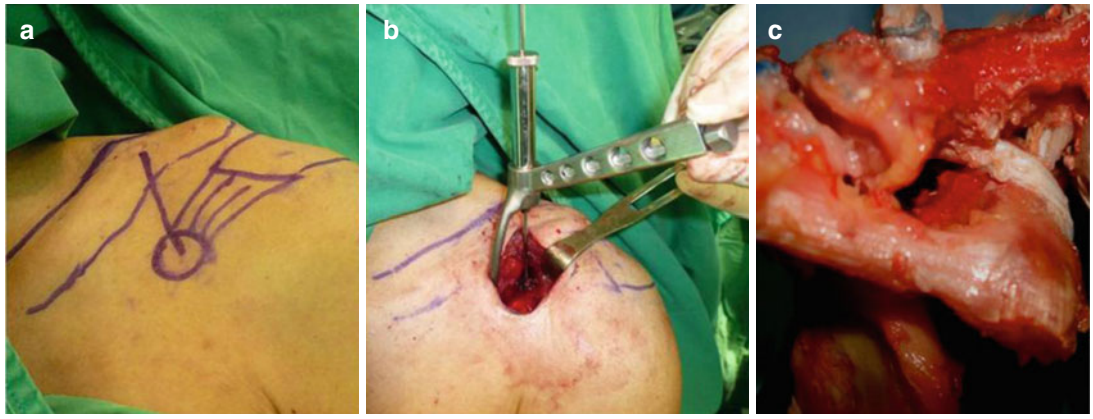

Fig. 23.6 (**a**) Surface marking of the coracoid process, clavicle, acromion, and coracoacromial ligament. Note that the tentative direction of the bone tunnel to be drilled from the clavicle to coracoids has been also marked. (**b**) Guide wire being inserted from the clavicle to the coracoid process. (**c**) Cadaveric specimen showing anatomic double-bundle coracoclavicular ligament reconstruction with a semitendinosus graft with the tunnels made in the clavicle and coracoid process

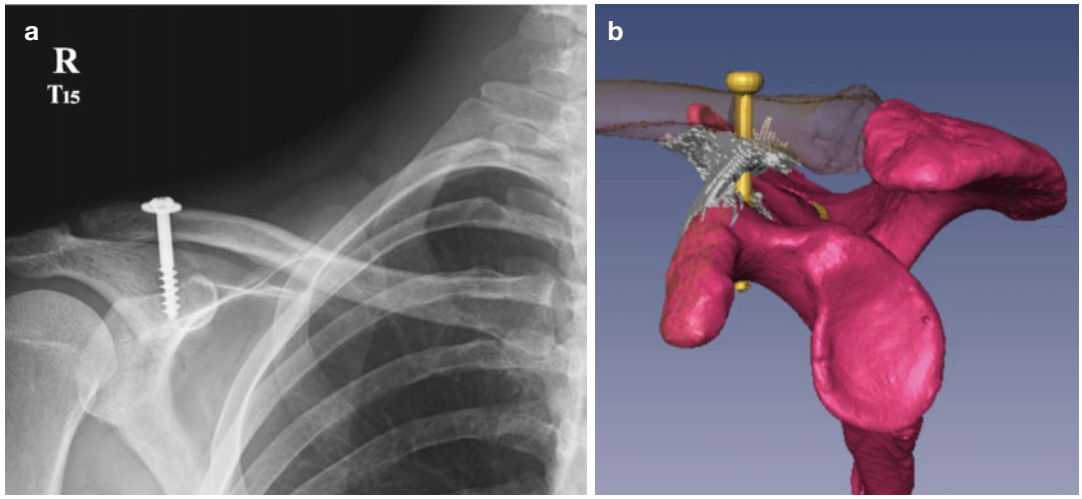

Fig. 23.7 (**a**) X-ray showing coracoclavicular screw fixation with a washer. (**b**) Illustration showing the screw fixation of the coracoclavicular joint – screw put in from the clavicle to the coracoid base

However current recommendations are doing CC ligament repair along with coracoclavicular cancellous screw fixation with a washer and screw removal at 8 weeks postoperatively. Before screw removal elevation of the arm beyond 90° is not permitted.

Major complications with the coracoclavicular screw include infection, screw back out, and redislocation after screw removal (Fig. 23.7).

23.6 Author's Preferred Treatment

The author's preferred treatment in acute cases (<4 weeks post-injury) is coracoclavicular fixation with a tightrope application done with an arthroscopic-assisted mini open technique. For delayed or chronic cases we do a double-bundle

anatomic coracoclavicular ligament reconstruction with a mini open technique. The technique has been done for quite a long period now and has good results in our institute. The technique is primarily indicated for type V AC joint separations and in some cases of type III AC joint dislocations. For type IV injuries we prefer an open reduction and hook plate fixation. We do not have any experience with type VI injuries.

23.6.1 Surgical Technique of Double-Bundle Coracoclavicular Ligament Reconstruction [27]

The entire process is done arthroscopically but for a small incision given over the posterior aspect of the distal clavicle. The patient is taken in a lateral position as for routine arthroscopic procedures. A standard posterior portal is created 1 cm inferomedial to the posterolateral corner of the acromion. After diagnostic arthroscopy of the glenohumeral joint to look for associated injuries, the subacromial space is entered. An anterolateral portal is made approximately 1.5 cm lateral to and in line with the anterior border of the acromion. A 3.5-mm right angle radiofrequency ablation device is introduced through the anterolateral portal and the subacromial bursa is removed for better visualization and ease of tissue dissection at the base of the coracoid.

The coracoid base is identified and the soft tissue around the coracoid base is removed via the anterior portal to facilitate easy graft passage.

Next, a 2-cm-long incision is given over the posterior margin of the distal clavicle at its most convex point. The trapezius muscle is dissected off the clavicle. Approximately 1 cm anterior to the conoid tubercle a unicortical hole is made in the clavicle to mark the conoid tunnel on the clavicular side. Under arthroscopic vision, an AC TightRope® drill guide (Arthrex, Inc., Naples, FL, USA) is then introduced through the anterior portal and placed under the base of the coracoid arch, while its other end (the drill sleeve) is placed in the pre-drilled hole on the clavicle. The drill guide is held in this position, and under clear arthroscopic view, a 2.4-mm guide pin is drilled from the superior surface of the clavicle to the undersurface of the coracoid. Next, a 5.5-mm tunnel is made based on the 2.4-mm pre-drilled tunnel, using a reamer guide pin. The superior positioning of the tunnel on the clavicle for the conoid ligament permits automatic anatomical reduction of the joint while fixing the conoid ligament.

For the trapezoid ligament, the guide pin is placed 20 mm lateral from the center of the conoid tunnel and slightly more anteriorly and then reamed with a 5.5-mm reamer. The trapezoid tunnel is confined to the clavicle and does not span the coracoid process. After both the tunnels are reamed, a double-loop semitendinosus tendon graft or an allograft 5.5 mm in diameter is whipstitched into place with sutures at each end. One limb of the graft is threaded through the conoid clavicular tunnel in the clavicle and followed through the base of the coracoid. The free end of the graft, exiting the coracoid at its undersurface, is then threaded through the clavicular trapezoid tunnel. Two PEEK (8 × 5.5 mm) screws (Arthrex, Inc., Naples, FL, USA) are used to fix each end of the graft to the clavicle.

The focus in this procedure is to create ligament footprints, both on the coracoid process and on the undersurface of the clavicle, with bony tunnels rather than a loop/sling around the coracoid process. In this way, the reconstructed ligaments replace the torn ligaments in a natural and more anatomical fashion.

23.6.2 Surgical Procedure of CC Tightrope Fixation

For tightrope fixation all the steps followed are the same except for the fact that no trapezoid tunnel is drilled and the tightrope is passed under arthroscopic guidance through the conoid tunnel and fixed to the undersurface of the coracoid on one side and the superior surface of the clavicle on the other side.

The entire process is done arthroscopically but for a small incision given over the posterior

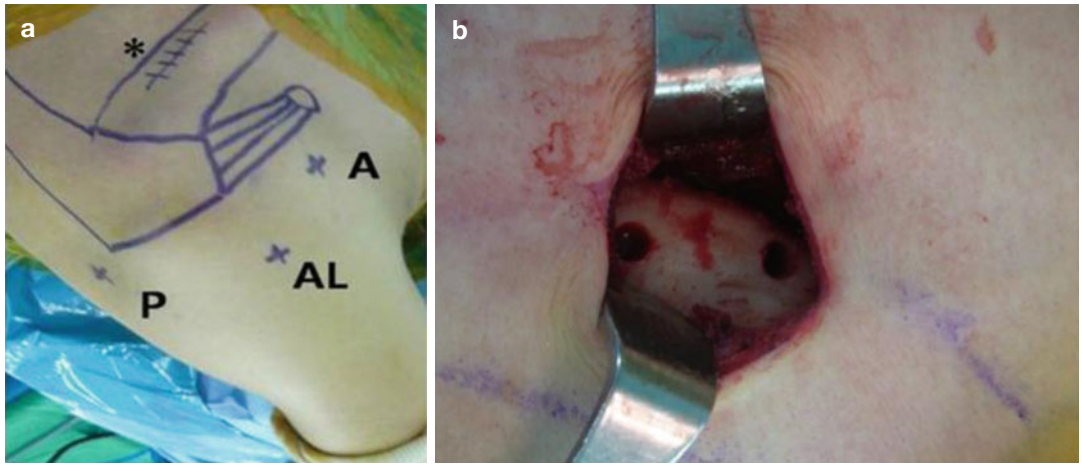

Fig. 23.8 (**a**) The standard portals used *P* to represent the site for the posterior portal, *A* is the anterior portal, and *AL* is the anterolateral portal. *Asterisk* additional 2.0 cm-long incision over distal clavicle. (**b**) *Top view* of the clavicular orifices of the conoid and the trapezoid tunnels. The anterior tunnel is for the trapezoid portion and the posterior orifice is the conoid tunnel in the clavicle

aspect of the distal clavicle. The patient is taken in a lateral position as in the case for double-bundle reconstruction and similar portals are created. After diagnostic arthroscopy of the glenohumeral joint to look for associated injuries, the subacromial space is entered. The coracoid base is identified and the soft tissue around the coracoid base is removed via the anterior portal to facilitate easy graft passage.

Next, a 2-cm-long incision is given over the posterior margin of distal clavicle at its most convex point. The trapezius muscle is dissected off the clavicle. Approximately 1 cm anterior to the conoid tubercle a unicortical hole is made in the clavicle to mark the conoid tunnel on the clavicular side. Under arthroscopic vision, an AC TightRope® drill guide (Arthrex, Inc., Naples, FL, USA) is then introduced through the anterior portal and placed under the base of the coracoid arch, while its other end (the drill sleeve) is placed in the pre-drilled hole on the clavicle. The drill guide is held in this position, and under clear arthroscopic view, a 2.4-mm guide pin is drilled from the superior surface of the clavicle to the undersurface of the coracoid. Following this, a 5.5-mm tunnel is made based on the 2.4-mm pre-drilled tunnel, using a reamer guide pin. Then the tightrope is introduced from the clavicular side to the coracoid base and locked in situ by flipping the coracoid endobutton making it horizontal. At this point the assistant reduces the AC joint by downward pressure over the distal clavicle and the surgeon tightens the tightrope and locks the clavicular endobutton by knot placement. Slight overcorrection is attempted and desirable to compensate for the loss or reduction in the postoperative period. The tightrope is secured on the clavicular side by a number of knots, and then after thorough washing of the wound, it is closed in layers taking uttermost care to repair the deltotrapezial fascia (Figs. 23.8, 23.9, 23.10, 23.11, and 23.12).

23.7 Rehabilitation

Postoperatively, the arm was placed in a Kenny Howard brace for 3–4 weeks. Then, patients were allowed to perform pendulum exercises and to use their arm for waist-level activities of daily living. Activity above shoulder level was prohibited for the first 2 postoperative months.

Stretching exercises for overhead activity was started after 2 months, and return to contact or overhead sports was allowed at 6 months.

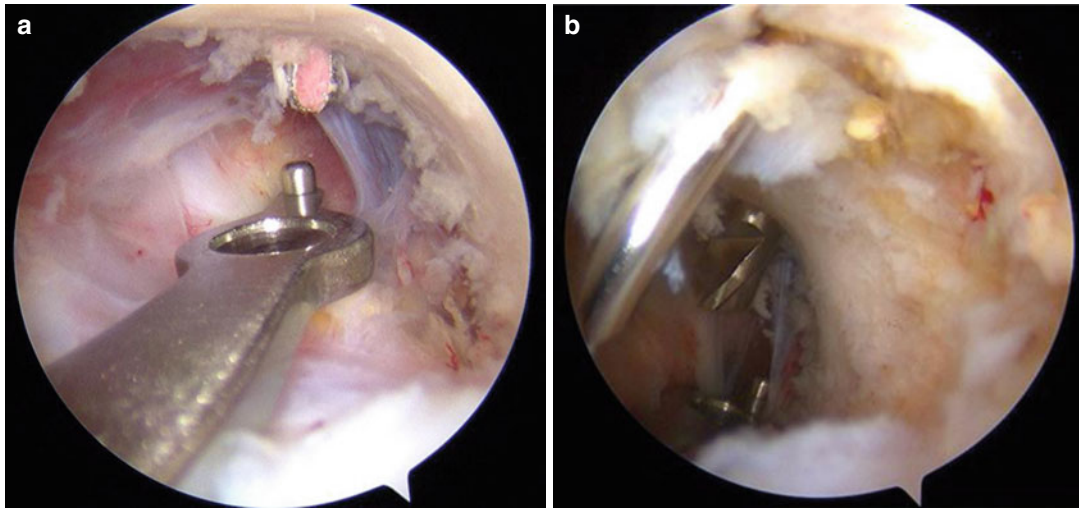

Fig. 23.9 (**a**) Placement of AC tightrope drill guide for the conoid tunnel in the coracoid base. (**b**) The distal hole with the drill bit in situ represents the orifice of the conoid tunnel in the coracoid base. The proximal hole with the guide wire in situ denotes the trapezoid tunnel orifice on the undersurface of the clavicle

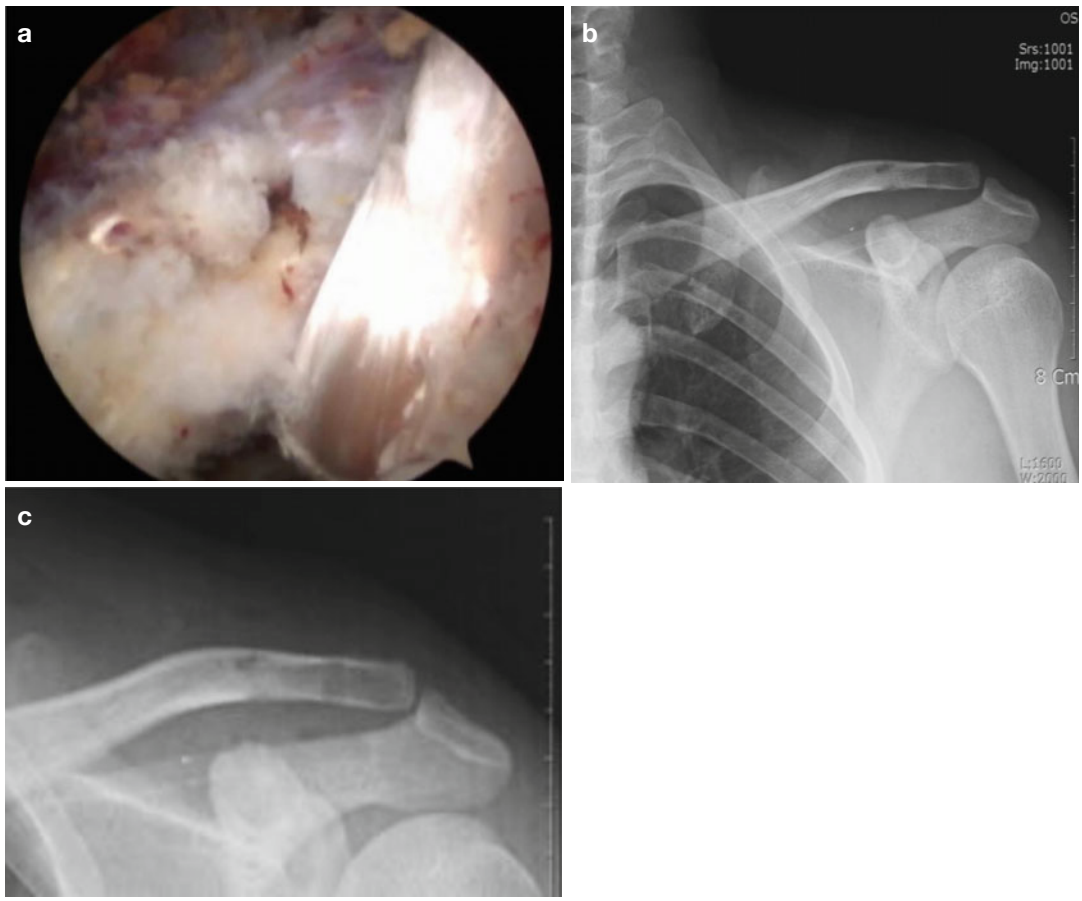

Fig. 23.10 (**a**) The figure shows an arthroscopic view wherein the semitendinosus tendon can be seen looped from the conoid tunnel into the trapezoid tunnel. (**b**, **c**) Postoperative radiologic findings after double-bundle CC ligament reconstruction showing reduced AC joint. Note the tunnel marks in the clavicle and coracoid

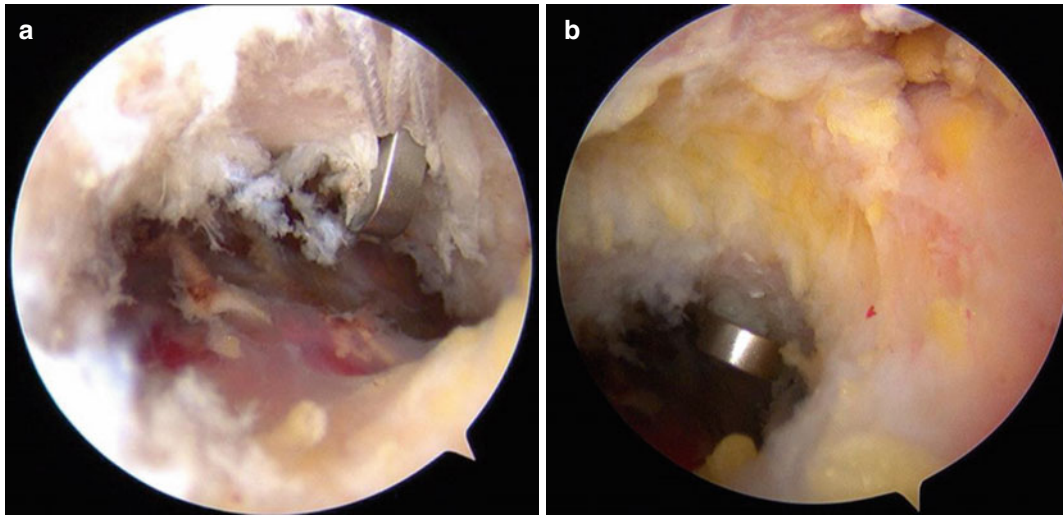

Fig. 23.11 (**a**) Arthroscopic view or the coracoid base with the tightrope being introduced from the tunnel. (**b**) Arthroscopic view of the tightrope flipped and put in position in the coracoid base

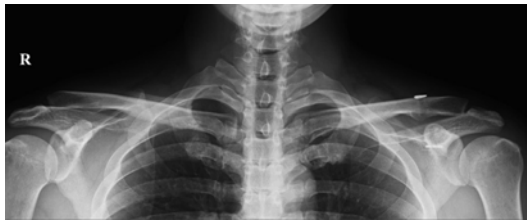

Fig. 23.12 Postoperative x-rays showing tightrope in situ over the left side with slight overcorrection of the AC joint displacement to compensate for the delayed loss or reduction

23.8 Complications of the Procedure

We have had very good results in our institute with the procedures described with very few complications. However complications include lack of anteroposterior reduction, tunnel malposition, and delayed loss of reduction. Theoretically, allograft rejection, graft breakage due to tension, and clavicular fractures are also possibilities.

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Biceps Instability: With Versus Without Rotator Cuff Lesions

24

Brody A. Flanagin, Kelly Fitzpatrick, Raffaele Garofalo, Gi-Hyuk Moon, and Sumant G. Krishnan

24.1 Introduction

Disorders of the biceps tendon are a well-recognized cause of shoulder pain/dysfunction. They can be broadly classified according to the following etiologies: (1) traumatic, (2) inflammatory, or (3) instability. Instability of the LHBT, first described by Meyer in 1926, can be further subdivided into two categories: instability with or without concomitant rotator cuff pathology [1]. In the following chapter, we provide a concise review of the pathoanatomy, clinical presentation, diagnosis, and treatment of instability of the LHBT.

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24.2 Pathoanatomy and Biomechanics

The LHBT originates within the glenohumeral joint, consisting of a broad origin from the supraglenoid tubercle and superior glenoid labrum. There is some variation regarding its superior glenoid labral attachment with a predominance toward the posterior labrum [2]. The intra-articular portion of the biceps tendon traverses through the glenohumeral joint in the rotator interval posterior to the coracohumeral ligament (CHL) and anteroinferior to the supraspinatus tendon. Upon exiting the glenohumeral joint, the biceps is surrounded and stabilized by a coalescence of structures that make up the biceps pulley. These structures include the CHL, superior glenohumeral ligament (SGHL), and fasciculus obliquus along with contributions from the adjacent supraspinatus and subscapularis tendons [3, 4] (Fig. 24.1). The anterior biceps pulley is made of the medial/deep fibers of the CHL and the SGHL, while the posterior biceps pulley consists of the lateral/superficial fibers of the CHL as they blend with the anterior fibers of the supraspinatus [3–5]. The floor of the pulley consists mainly of fibers from the SGHL which blends with the upper subscapularis tendon, while the roof of the pulley is made up of fibers from the SGHL, CHL, and fasciculus obliquus [4, 5]. The LHBT then exits the glenohumeral joint and travels within the intertubercular

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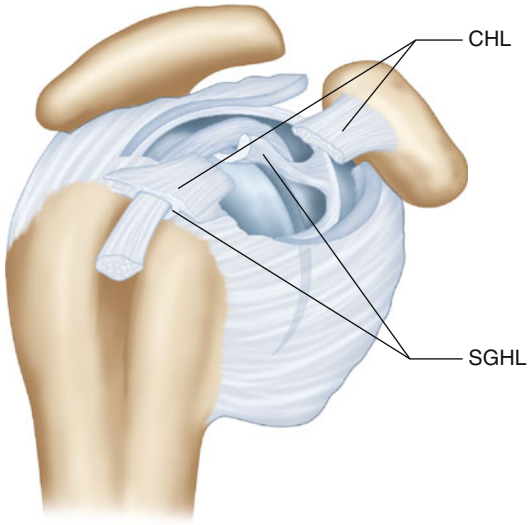

Fig. 24.1 Anatomy of the biceps pulley. The CHL and SGHL blend together with the upper fibers of the subscapularis anteriorly, while the CHL blends with the anterior fibers of the supraspinatus posteriorly. The fasciculus obliquus (not pictured here) forms the roof of the sling (Reprinted with permission Habermeyer et al. [13])

(bicipital) groove under the transverse humeral ligament and down the anterior aspect of the arm before merging with the biceps muscle belly. The biceps is innervated by a network of sensory sympathetic fibers and derives its blood supply from branches of the thoracoacromial and brachial arteries [6, 7]. There is a hypovascular region of the intra-articular biceps tendon extending from 1.2 to 3 cm distal to its origin, which may predispose this area to rupture [7].

While there is a general consensus that the biceps is a common pain generator in the shoulder, the function of the biceps has been debated in the literature and is not completely understood. It has been reported to serve as a dynamic stabilizer in the unstable shoulder as well as a dynamic depressor and restraint to external rotation of the humeral head during abduction in the stable shoulder [8–10].

Biceps pulley lesions can result from either a degenerative or traumatic process, which typically results from a fall on the outstretched arm (in combination with full external or internal rotation), a fall backward on the hand or elbow, or a forcefully stopped overhead throwing motion [11]. Gerber and Sebesta first described the concept of anterosuperior impingement of the shoulder whereby there is contact between the upper subscapularis, biceps pulley, and LHBT with the anterosuperior glenoid rim [12]. They suggested that this phenomenon can lead to lesions of the upper subscapularis (involving the articular fibers) and/or anterior biceps pulley that can result in biceps instability, a finding supported by Habermeyer et al. [13]. Boileau et al. have suggested that a hypertrophic "hourglass" biceps may lead to pathologic stretching of the biceps pulley, thereby causing symptomatic instability of the tendon at its entrance to the bicipital groove [14]. Baumann et al. have suggested that biceps pulley lesions are progressive in nature and ultimately lead to adjacent rotator cuff tearing at the rotator interval [11].

Depending on the location of the pulley lesion, instability of the biceps tendon may be present anteromedially, posterolaterally, or anteroposteriorly [15, 16]. While dislocations of the LHBT have been described in either an anteromedial or posterolateral direction, subluxations can occur anteromedially, posterolaterally, or anteroposteriorly [15, 16]. When the tendon sits or can be manipulated out of its normal position as it enters the bicipital groove without passing over the greater or lesser tuberosity, it is defined as a subluxation. Conversely, when the tendon rests or can be manipulated completely out of the bicipital groove and over the greater or lesser tuberosity, it is defined as a dislocation. Furthermore, biceps instability can be present either with or without concomitant rotator cuff lesions. While anteromedial subluxation can be secondary to an isolated lesion of the anterior biceps pulley (i.e., SGHL), anteromedial dislocation has only been reported in the presence of associated tearing of either the supraspinatus or subscapularis [3, 15–17]. Partial tearing of the superior fibers of the subscapularis appears to be more commonly associated with anteromedial dislocation versus subluxation of the LHBT [15, 16]. Slätis and Aalto demonstrated that disruption of the CHL was the key anatomic finding that led to anteromedial instability of the biceps tendon in the

presence of a supraspinatus tear [17]. All cases of anteromedial dislocation were found to have an associated full-thickness supraspinatus tear, while all cases of anteromedial subluxation were found to have a partial-thickness tear of the anterior supraspinatus. In all of their cases of anteromedial dislocation, they noted the biceps tendon was dislocated along the ventral (anterior) surface of the subscapularis. No associated lesions were reported in the SGHL or subscapularis. However, their anatomic description of the CHL included all tissues between the superior border of the subscapularis and anterior border of the supraspinatus and therefore likely included some portion of what is now termed the SGHL. Walch et al. described anteromedial instability of the LHBT in the presence of a full-thickness supraspinatus tear and an associated "hidden lesion" of the deep fibers of the CHL, SGHL, and upper subscapularis [3]. In all of their cases, the LHBT was located along the deep (posterior) surface of the upper subscapularis tendon. It is reasonable to deduce that the location of an anteromedial biceps tendon dislocation (ventral or deep surface of the subscapularis) is dictated by the integrity of the deep fibers of the CHL, SGHL, and superior fibers of the subscapularis.

Isolated posterolateral instability of the LHBT is generally secondary to disruption of the posterior biceps pulley consisting mostly of the CHL as it blends with the anterior fibers of the supraspinatus [5, 15]. This can be seen in association with either a partial- or full-thickness tear of the supraspinatus involving the far anterior tendon footprint [15]. Anteroposterior biceps instability occurs as a result of a lesion to the anterior and posterior biceps pulley and is highly associated with tears of both the anterior supraspinatus and subscapularis involving at least the upper one-third of the tendon [15, 16].

24.3 Classification

Several authors have proposed classification systems for biceps pulley lesions. Habermeyer et al. described lesions of the biceps pulley leading to anteromedial instability of the biceps tendon in association with anterosuperior impingement of the shoulder (Fig. 24.2) [13]. Group 1 consisted of isolated lesions of the SHGL; group 2 consisted of SGHL and partial articular-sided tears of the supraspinatus; group 3 involved the SGHL and partial articular-sided tears of the subscapularis; and group 4 involved the SGHL and partial articular-sided tears of both the subscapularis and supraspinatus.

Bennett proposed a slightly more detailed classification of rotator interval lesions in the presence of an anterosuperior (supraspinatus and subscapularis) rotator cuff tear that allows for anteromedial biceps subluxation/instability (Fig. 24.3) [18]. Type 1 lesions involve tears of the subscapularis without involvement of the medial fibers of the CHL. Type 2 lesions result from tearing of the medial fibers of the CHL without a tear of the subscapularis. Type 3 lesions result from concomitant tears of the subscapularis and medial fibers of the CHL. Type 4 lesions involve the posterior biceps pulley consisting of tears of the supraspinatus and the lateral fibers of the CHL. Type 5 lesions result from tearing of the anterior and posterior pulley and involved the subscapularis, with medial and lateral fibers of the CHL and the leading edge of the supraspinatus tendon.

More recently, Lafosse et al. described an arthroscopic classification of LHBT instability in patients with rotator cuff tears [15]. Their classification scheme allows for stratification according to the degree of instability (subluxation vs dislocation), direction of instability (anterior, posterior, or combined anteroposterior), macroscopic appearance of the LHBT, and integrity of the adjacent rotator cuff tendons. This was the first proposed classification system to highlight the importance of posterolateral instability of the LHBT.

24.4 Clinical Presentation and Essential Physical Exam

As with all patients presenting with a chief complaint of shoulder pain, a careful history is essential to accurately determine the proper diagnosis. Patients with LHBT instability can be challenging to evaluate as there is no one specific

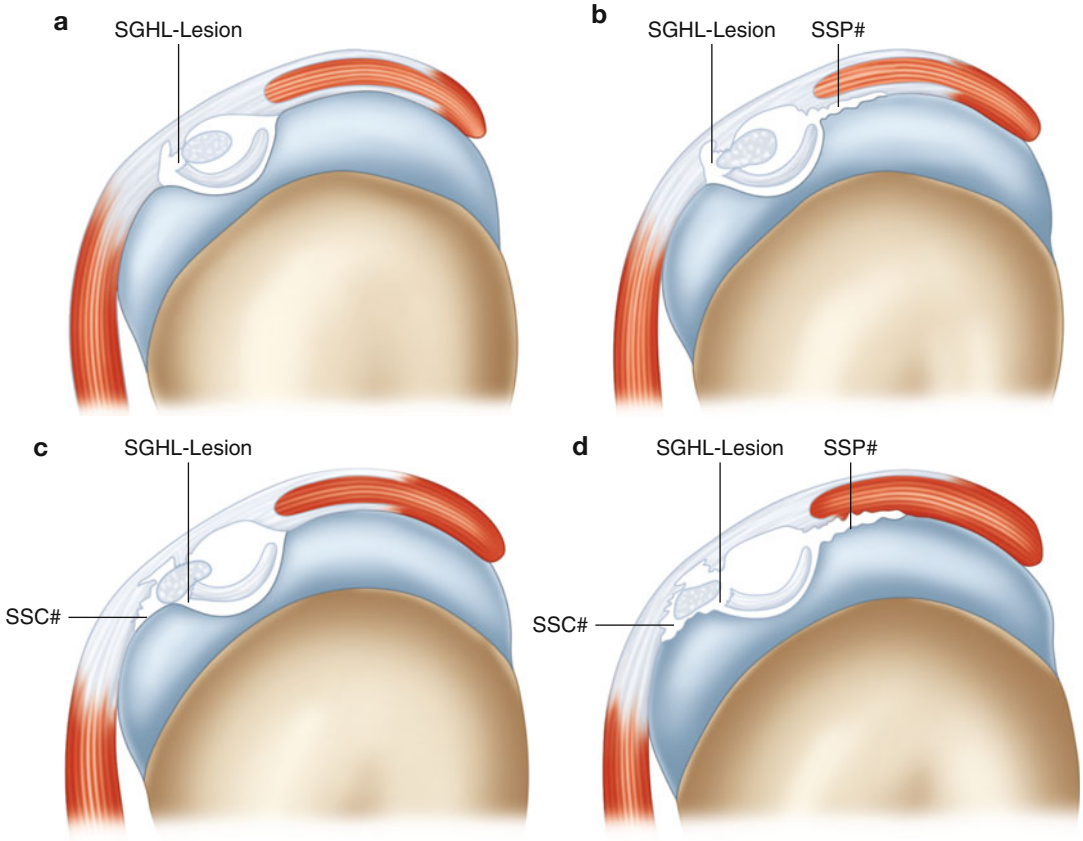

Fig. 24.2 Classification of biceps pulley lesions according to Habermeyer et al. (**a**) Isolated SGHL lesion. (**b**) SGHL lesion and partial articular-sided supraspinatus tear. (**c**) SGHL lesion and partial articular-sided upper supraspinatus tear. (**d**) SGHL lesion and partial articular sided tear of the upper subscapularis and supraspinatus. (Reprinted with permission Habermeyer et al. [13])

component of the history we are aware of that is unique to this population. As previously noted, biceps pulley lesions can be caused by either trauma or degenerative changes. If the onset is traumatic in nature, particular attention should be paid to the mechanism of injury. Pulley lesions can be a result of a fall on an outstretched arm (in combination with internal or external rotation), a fall backward on the hand or elbow, or a forcefully stopped overhead throwing motion of the arm [13]. Pain can often be diffuse and nonspecific or located in the anterior or anterolateral aspect of the shoulder. Some patients may complain of a "deep" pain in the front of the shoulder. Associated clicking or popping of the shoulder may or may not be present.

Physical examination of patients with suspected biceps pathology can be particularly challenging. Numerous tests have been described in the literature, but currently there is no single one that serves as the gold standard. Pain may be present when palpation is done along the bicipital groove, but this can be nonspecific. O'Brien's, Speed's, and Yergason's tests have not been shown to have very good sensitivity or specificity in differentiating biceps pathology from SLAP tears [15, 19]. However, Kibler et al. have demonstrated that the combination of a positive Speed's and upper cut tests is significantly better at detecting isolated biceps pathology separate from SLAP tears [20]. Rotator cuff strength testing may reveal pain

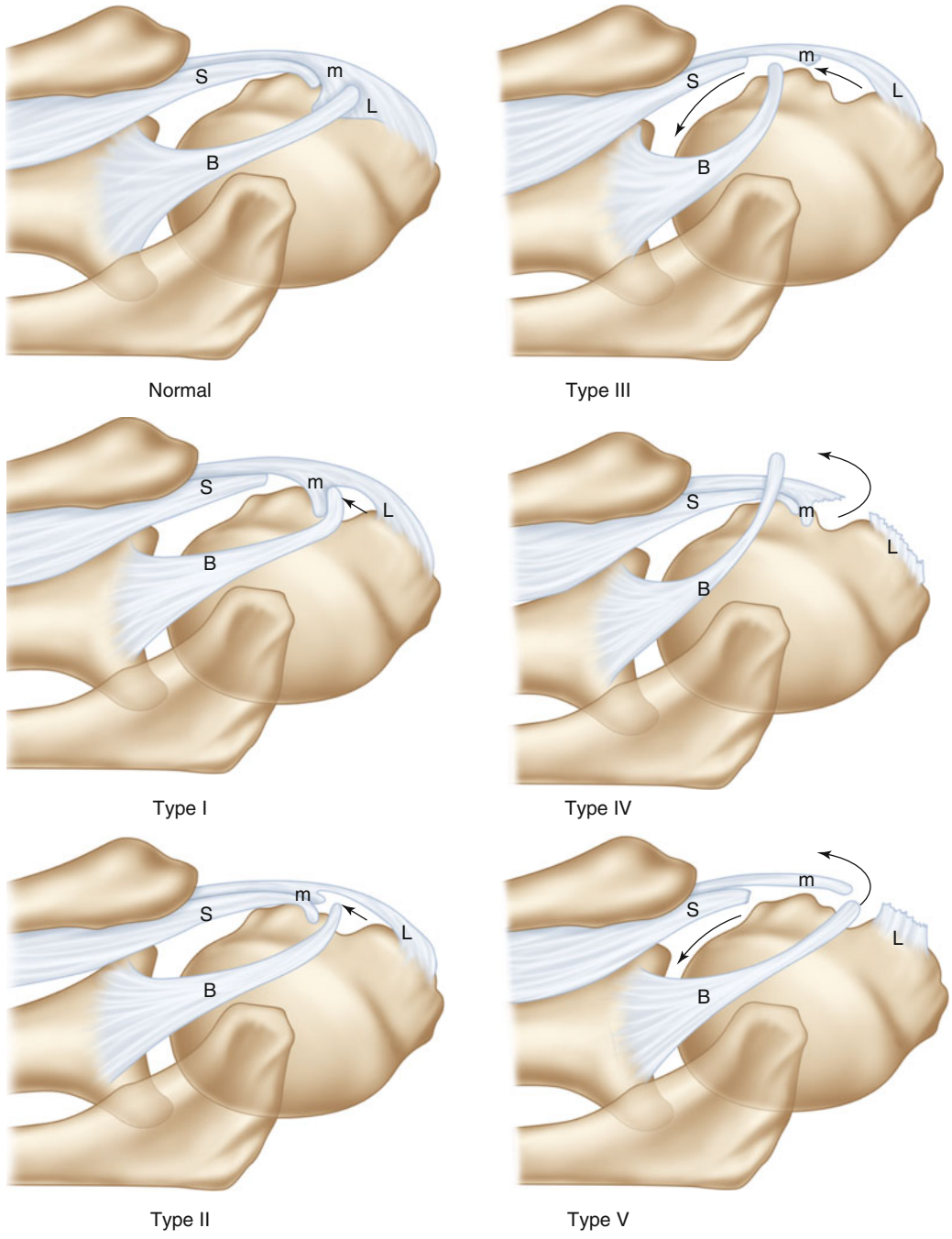

Fig. 24.3 Classification of rotator interval lesions according to Bennett (Reprinted with permission Bennett [18])

and/or weakness if associated tearing is present. In particular, the belly-press test may be positive if the SGHL/upper subscapularis is involved. However, positive provocative tests for rotator cuff pathology do not definitively rule in a biceps pulley lesion.

Bennett has previously described a provocative maneuver called the biceps subluxation test in order to better delineate lesions resulting in biceps instability [18, 21]. To perform this test the patient's arm is held in 90° of abduction and full external rotation. The arm is then passively brought into full cross-body adduction and internal rotation in an effort to elicit subluxation of the biceps tendon within the sheath. The test is considered positive if the patient relates the sensation of pain, slipping, catching, or popping during the passive range. We are not aware of any studies in the literature that serve to validate this test.

Currently there is no literature present on the diagnosis of biceps pulley lesions specifically in the overhead athlete. However, when treating such patients, we feel it is important to take time to review pitch count, types of pitches being thrown, and how often they are throwing/pitching throughout the year. It is also important to determine which phase of throwing and at what arm position their pain ensues.

We prefer to perform a diagnostic injection with 1 % lidocaine into the glenohumeral joint in the office setting for patients with suspected biceps pathology based on history and physical examination. This injection is done under sterile conditions from an anterior approach halfway between the tip of the coracoid process and acromion. After a few minutes, the amount of relief reported by the patient is noted and can serve as a helpful tool for either raising or lowering the index of suspicion for biceps pathology.

24.5 Essential Radiology

Despite the inherent challenges noted above, a through history and physical examination should provide an accurate level of suspicion for biceps pathology/biceps pulley lesion. We feel that the radiographic examination should be obtained to confirm the appropriate diagnosis and rule out any additional pathology that may also need to be addressed during surgery. We begin with plain radiographs on all patients consisting of anteroposterior (AP) views in internal and external rotation along with axillary lateral and outlet views. Subtle cystic erosion involving the lesser tuberosity has been previously reported in patients with "hidden lesions" of the rotator interval; however, we are not aware of any other plain radiographic findings specific for lesions of the biceps pulley [3].

Magnetic resonance imaging (MRI) is a helpful tool to assess for a biceps pulley lesion, LHBT instability, as well as associated rotator cuff pathology. Magnetic resonance arthrography (MRA) has been shown to be more sensitive and specific for rotator cuff and rotator interval pathology, and this remains our study of choice in evaluating for a biceps pulley lesion [22, 23]. Optimal MR imaging should include images obtained in all three planes and aligned with the glenohumeral joint. Since MRA is a static image, it may not demonstrate clear evidence of biceps tendon instability depending on the extent of the pathology and the position of the arm at the time of the study [15, 21]. We pay particular attention to the T1- and T2-weighted axial and sagittal oblique image sequences to evaluate the contents of the rotator interval and biceps pulley for pathology that would be suggestive of biceps instability (Fig. 24.4). We find the axial and coronal sequences more helpful for evaluating associated tearing of the rotator cuff. The axial images are principally used to evaluate for anteromedial biceps tendon subluxation or dislocation in conjunction with an upper subscapularis lesion and should be carefully considered when deciding on proper surgical treatment (Fig. 24.5). Of note, if patients cannot undergo MRA for any reason, then we use CT arthrogram as our preferred imaging test.

Ultrasound has recently gained increased popularity amongst orthopedic surgeons in the diagnosis of shoulder pathology. This modality has shown good utility in diagnosing rupture, subluxation, or dislocation of the biceps tendon but poor accuracy in diagnosing small partial-thickness tears or fraying of the intra-articular LHBT [24, 25].

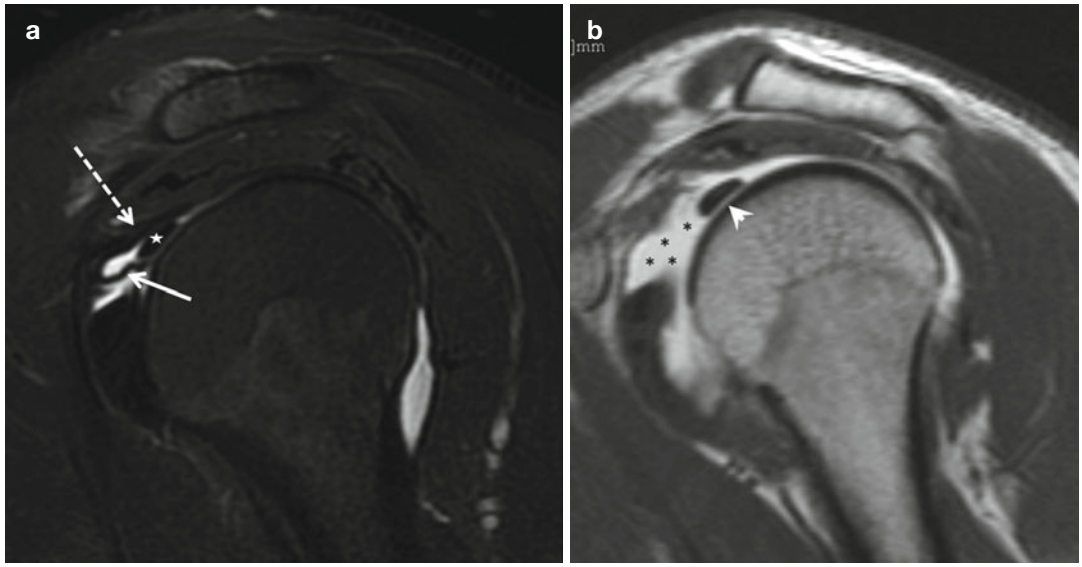

Fig. 24.4 (**a**) Oblique sagittal fat-saturated T2-weighted images showing the biceps tendon (*star*), SHGL (*white arrow*), and CHL (*dotted white arrow*). (**b**) Oblique sagittal fat-saturated T1-weighted images demonstrating biceps tendon (*white arrowhead*) with a tear of the SGHL (*black stars*) (Reprinted with permission Zappia et al. [26])

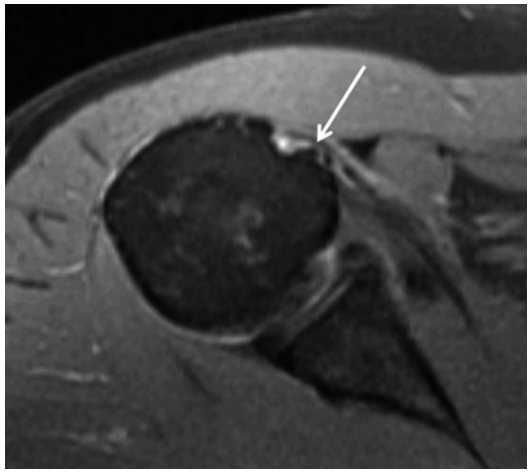

Fig. 24.5 Axial T2-weighted MRI of a patient with anteromedial biceps instability. The long head of the biceps tendon is flattened and perched on the lesser tuberosity (*white arrow*). The subscapularis origin showed mild tendinosis

24.6 Disease-Specific Clinical and Arthroscopic Pathology

It is our feeling that diagnostic arthroscopy is the "gold standard" for accurate diagnosis of biceps pulley lesions and biceps instability. Despite the high sensitivity and specificity of MR arthrography for lesions of the biceps pulley, it represents a static image for a potentially dynamic problem and in our experience can be inadequate if the images are not perfectly aligned with the glenohumeral joint. We begin with visualization of the biceps tendon and its position within the glenohumeral and entrance to the bicipital groove. The ramp test is then performed as described by Motley et al. to assess for anteromedial instability of the biceps tendon [27]. We then visualize the anterior and posterior biceps pulley for any tearing and use an arthroscopic probe to manually test for anteromedial or posterolateral biceps subluxation/instability (Fig. 24.6). This is followed by dynamic evaluation of the biceps tendon as described by Lafosse et al. in slight abduction with external and internal rotation to test for anteromedial and posterolateral biceps instability, respectively [15]. In the presence of what appears to be an isolated supraspinatus tear, careful attention must be directed to the rotator interval for evidence of a "hidden lesion" involving the CHL, SHGL, and upper subscapularis as described by Walch et al. [3]. This can result in anteromedial instability of the biceps tendon and should be treated with biceps tenodesis and

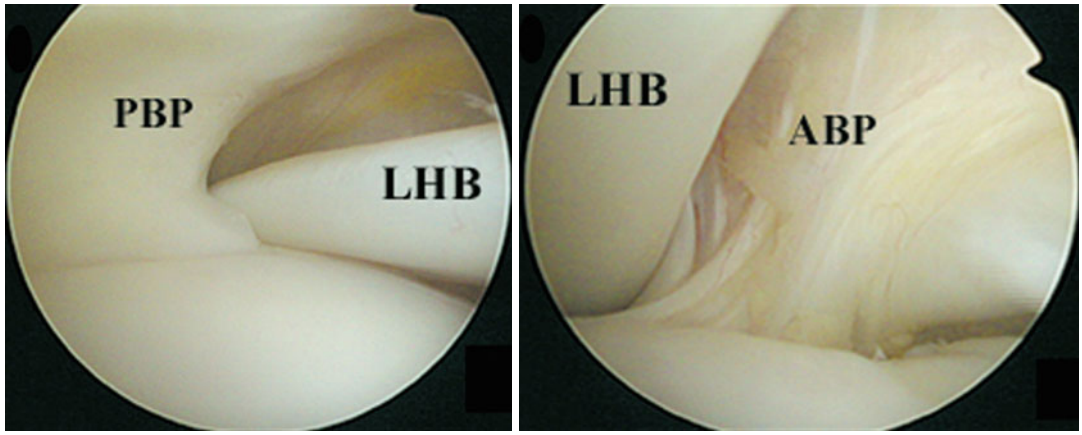

Fig. 24.6 Arthroscopic images demonstrating the relationship of the biceps tendon (*LHB*), anterior biceps pulley (*ABP*), and posterior biceps pulley (*PBP*) (Reprinted with permission [15])

possible subscapularis repair depending on the extent of its involvement. In patients with an anterosuperior (subscapularis and supraspinatus) rotator cuff tear, careful attention should be directed to the anterior and posterior biceps pulley for evidence of biceps instability.

24.7 Treatment Options

There are a number of ways to address biceps instability. Treatment of this problem should be directed specifically toward patient goals and expectations. Treatment goals, whether nonoperative or operative, are directed toward decreasing pain and restoring motion and strength.

As with most shoulder injuries, initial treatment should consist of activity modification and nonsteroidal anti-inflammatory medications. While there is literature supporting the efficacy of physical therapy for rotator cuff tears, there are no studies discussing the efficacy of physical therapy for a specific diagnosis of biceps instability [28]. While therapeutic injections with cortisone and some other medications are commonly performed for rotator cuff tears and may provide temporary pain relief, we do not feel that these injections are likely to provide much relief for an isolated biceps pulley lesion with a symptomatic, unstable biceps tendon.

If nonoperative treatment fails to provide significant relief, surgical treatment is generally indicated. Surgery may be performed either arthroscopically, arthroscopic-assisted, or open. Treatment begins with careful examination under anesthesia. Though it may be difficult to recreate a palpable click of the biceps tendon, the patient must be assessed for occult instability of the shoulder.

A diagnostic arthroscopy is performed in the standard fashion by viewing the shoulder from the posterior portal. An anterior portal is established via an outside-in technique with a spinal needle, and a probe is used to evaluate the biceps tendon. The ramp test allows for the biceps tendon to be pulled into the joint to assess for degenerative changes or tenosynovitis at the level of the groove as well as to assess the integrity of the anterior pulley. Further direct inspection of the LHBT and rotator interval should be performed to assess for a "hidden lesion" and/or posterior pulley lesion. Associated tearing of the rotator cuff can be performed using arthroscopic, mini-open, or open techniques. Arthroscopic subscapularis repairs can be performed from either an intra-articular or extra-articular approach depending on the extent of the tear and surgeon preference. Additional rotator cuff pathology involving the supraspinatus and/or infraspinatus can be repaired from the subacromial space in the standard fashion.

Biceps instability with or without a rotator cuff lesion can be treated with either tenodesis or tenotomy using arthroscopic or open techniques. The technique chosen should be based on surgeon preference and does not necessarily depend on the presence of a rotator lesion that requires repair. We do not advocate pulley repair and biceps recentering as this leads to a high rate of secondary rupture/failure [3]. There are no randomized controlled studies to support tenodesis over tenotomy [29]. Patients younger in age and/or with high activity demands (i.e., athletes) may have better outcomes after tenodesis due to the higher risk of cosmetic deformity and fatigue/cramping after tenotomy [30]. Arthroscopic or open suprapectoral biceps tenodesis can be performed using soft tissue techniques, via suture anchors, and using an interference screw [31–33]. Open subpectoral biceps tenodesis can be performed via bone tunnels, suture anchors, or with interference screw fixation [34, 35]. Clinical and biomechanical studies have not demonstrated a clear "gold standard" with respect to either location or technique for biceps tenodesis [33, 35–37]. Good clinical outcomes have been reported for combined arthroscopic rotator cuff repair and biceps tenodesis using a suture anchor technique, but this needs to be further evaluated as well [38].

24.8 Author's Preferred Treatment

The senior author's preferred treatment of an isolated biceps pulley lesion/biceps instability is to perform an arthroscopic biceps tenodesis in a similar fashion as previously described by Boileau et al. [32]. After confirming biceps instability during diagnostic arthroscopy via the previous methods, the biceps tendon is percutaneously pierced with an 18-gauge spinal needle to prevent proximal retraction. The LHBT is tenotomized at its origin along the supraglenoid tubercle using an arthroscopic biter through the anterior portal. The arthroscope is placed in the anterolateral portal, which is placed 2–3 cm distal from the anterolateral corner of the acromion.

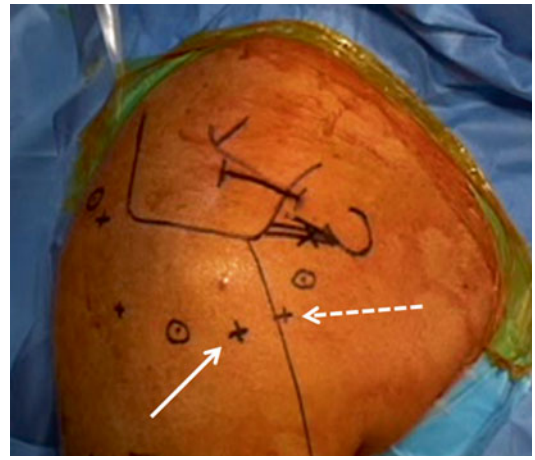

Fig. 24.7 Anterolateral viewing (*white arrow*) and biceps working portal (*dashed white arrow*)

It is critical to place the shoulder/arm in relative flexion (~30°) in order to adequately visualize and work in the anterior subacromial space. After adequate bursectomy, we establish a biceps portal approximately 2 cm distal from the anterolateral corner in line with the biceps tendon and transverse humeral ligament (i.e., straight anterior with the arm in 10° of internal rotation) (Fig. 24.7). The transverse humeral ligament is then completely released through the biceps portal, and the LHBT is exteriorized through this incision after removal of the 18-gauge needle (Fig. 24.8a). The elbow is hyperflexed past 90° to take tension off the LHBT, and it is then doubled over on itself using a high-tensile-strength suture after resecting the intra-articular portion of the LHBT. A 2.4 mm pin is then impacted with a mallet by hand through the anterior cortex of the humerus starting 1 cm below the top of the bicipital groove until it engages the posterior cortex of the humerus (Fig. 24.8b). A humeral socket roughly 30 mm in depth is created by drilling over the pin with a 7 mm reamer (Fig. 24.8c). The tendon is manually placed into the socket and seated appropriately. A Milagro® BR (DePuy Synthes, Raynham, MA) interference screw (generally of 7×23 mm diameter and length) is then placed over a nitinol wire to secure the tendon into the humeral socket to create an interference fit

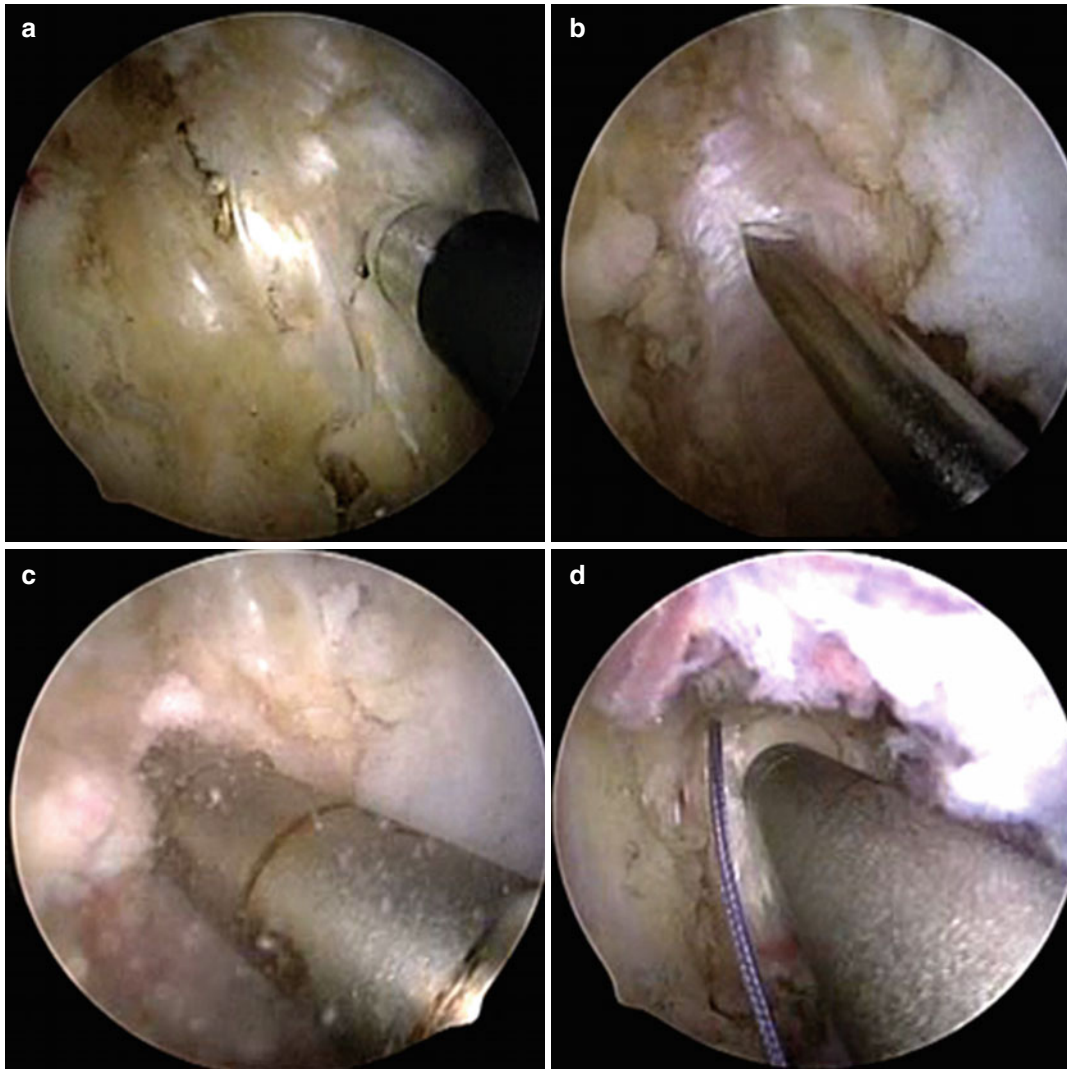

Fig. 24.8 (**a**) Identification of the biceps tendon after releases of the transverse humeral ligament with an electrocautery device. (**b**) Placement of a 2.4 mm pin 1 cm below the top of the bicipital groove. (**c**) Drilling of the humeral socket over the 2.4 mm pin to a depth of 30 mm with a 7 mm reamer. (**d**) Seating of the interference screw over a nitinol wire to secure the biceps tendon within the humeral socket and complete the tenodesis

(Fig. 24.8d). The elbow is gradually straightened to just short of full extension while advancing the screw to ensure the tendon is not overtensioned during the tenodesis (Fig. 24.9). The interference screw is left flush with the anterior cortex of the humerus. The elbow is then passively ranged to ensure there is no excessive tension on the tendon at the tenodesis site.

If biceps instability is present with a rotator cuff tear, the rotator cuff tear is performed using an all arthroscopic transosseous technique as previously described [39]. The biceps tenodesis is performed using either an interference screw technique method as described above or with a lasso loop technique using the transosseous sutures passed through the rotator cuff.

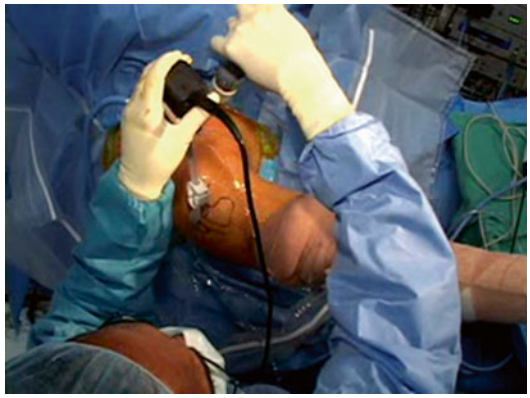

Fig. 24.9 The elbow is straightened to near full extension as the interference screw is advanced to ensure the LHBT is not overtensioned during the tenodesis

24.9 Rehabilitation

Rehabilitation of patients with biceps instability can be highly variable and typically depends on the severity of any associated rotator cuff pathology. All shoulder-specific rehabilitation is performed under the supervision of a physical therapist. For the rare patient who undergoes an isolated biceps tenotomy, sling immobilization is carried out for comfort only for 1–2 weeks, and the patient is allowed to return to all activities as tolerated without restriction when their pain level allows.

If an isolated arthroscopic suprapectoral or open subpectoral biceps tenodesis is performed, we place the patient in a Velpeau sling with the arm at the side for 2 weeks and allow no biceps or rotator cuff resistance exercises for 6 weeks postoperatively. We begin immediate scapular isometrics and passive and active-assisted elbow, wrist-hand motion. During weeks 0–3 we allow full passive and active forward flexion and external rotation only. We then begin passive and active internal rotation from weeks 4–6. Full passive and active motion in all planes is allowed at week 7, and we begin gentle strengthening exercises during weeks 7–9. Patients are released to all activities as tolerated at week 10. Full recovery can be expected between 4 and 6 months postoperatively.

If a biceps tenodesis is performed with a rotator cuff repair, it is our opinion that appropriate rehabilitation of the rotator cuff takes precedence. No specific changes are implemented that differ from our standard protocol that is individualized based on the size of the tear, number of tendons involved, amount of retraction, overall tissue quality, and security of the repair at the time of surgery.

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Elbow Arthroscopy and Related Anatomy

25

Edward S. Chang, Rachel Schneider, and Christopher C. Dodson

25.1 Introduction and Brief History

The roots of arthroscopy can be traced back to the early twentieth century. In 1931, Burman [1] described his experience with arthroscopy on cadaveric specimens. Based on earlier work by Nordentoft [2] and Bircher [3] on knee arthroscopy, a 3-mm laparoscope (developed by Han Christian Jacobaeus [4] 20 years earlier) was employed, and with the aid of distention via nonirritant gas or liquid, he was able to visualize the articular anatomy. Burman noted that the anatomy and biomechanics of certain joints precluded it from being amenable to safe arthroscopy, in particular the elbow. His work drew international interest, and among his foreign visitors was a Japanese surgeon, Dr. Watanabe [5].

While arthroscopic techniques of the shoulder and knee continued to evolve, elbow arthroscopy was largely neglected. In 1985, Andrews and Carson [6] published on a series of 12 patients that underwent elbow arthroscopy and concluded that arthroscopy of the elbow is a safe diagnostic tool as well as an effective treatment option of certain disorders, such as removal of loose bodies.

More recently, elbow arthroscopy has expanded its indications greatly—from the treatment of lateral epicondylitis to the debridement of osteochondritis dissecans of the capitellum to capsular contracture release, with more techniques constantly being innovated and refined. The potential advantages of treating elbow disorders arthroscopically include a better view of the intra-articular anatomy and less capsular disruption, leading to decreased stiffness. The main disadvantage of elbow arthroscopy is the increased risk of iatrogenic injury, owing to the close proximity of the neurovascular structures.

As the indications and use of elbow arthroscopy continue to expand and become more widespread, it is imperative the surgeon understands the relevant surgical anatomy, indications and contraindications, portal placement, and technique to ensure the best possible outcome for his or her patients.

25.2 Relevant Surgical Anatomy

As with any surgical procedure, an in-depth knowledge of the elbow anatomy must be mastered. Superficially, the landmarks can be palpated and used as reference (Fig. 25.1a, b). On the lateral aspect, the lateral epicondyle, radial head, and olecranon tip form the borders of the

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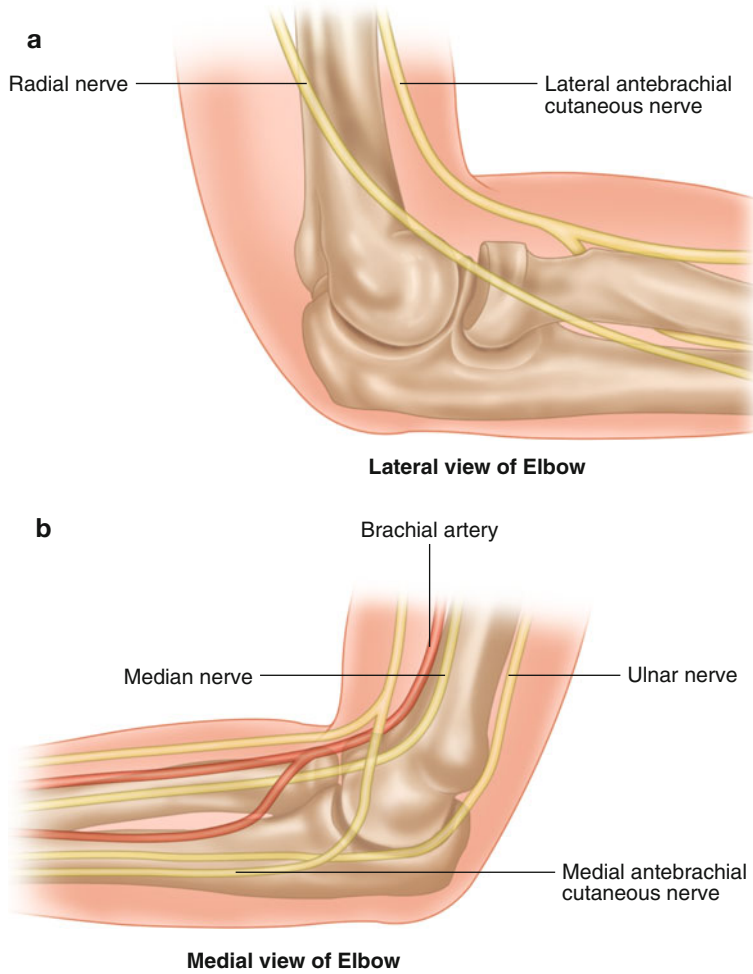

Fig. 25.1 Relevant elbow anatomy. (**a**) Lateral view of the elbow. (**b**) Medial view of the elbow

triangular shape interval, in which the "soft spot" can be palpated. Posteriorly, the triceps tendon and olecranon are identified. Anteriorly, the antecubital fossa can be palpated. Medially, the medial epicondyle and the course of the ulnar nerve should be identified.

Superficially, the nerves that one must be aware of are the medial and lateral antebrachial cutaneous nerves. The medial antebrachial cutaneous nerve (MABC) originates off the medial cord of the brachial plexus and travels along the ulnar aspect of the arm, along with the basilic vein. It pierces the deep fascia at the middle of the arm, where it branches and provides sensation to the ulnar aspect of the forearm. The lateral antebrachial cutaneous nerve is the termination branch or continuation of the musculocutaneous nerve. Proximal to the antecubital fossa, it emerges from the distal aspect of the biceps brachii and courses lateral along the brachioradialis. It then divides into volar and dorsal branches, supplying sensation to the radial forearm.

The deep neurovascular structures along the elbow include the median, radial, and ulnar nerves and the brachial artery. The brachial artery can be found proximally between the brachialis and biceps brachii. It courses medial to the biceps tendon and branches at the level of the radial head. The median nerve travels with the brachial artery on its medial side. It crosses the joint and enters

the forearm deep to the pronator teres. Proximally, the ulnar nerve courses posterior to the intermuscular septum. It continues to travel posterior to the medial epicondyle at the elbow joint and enters the forearm between the flexor digitorum superficialis and profundus. The radial nerve pierces the lateral intermuscular septum approximately 7.5 cm proximal to the joint line. It then courses through the anterior compartment between the brachialis and brachioradialis. At the elbow joint, the radial nerve bifurcates into the superficial sensory branch and the deeper posterior interosseous nerve. The superficial branch enters the forearm deep to the brachioradialis, while the posterior interosseous nerve pierces the supinator muscle and curves around the radial head.

25.3 Clinical Presentation and Physical Examination

25.3.1 Patient History

A comprehensive history is important in the formation of a differential diagnosis of the elbow. Mechanism of injury, signs, and symptoms should be elicited to determine whether this is acute in nature or chronic from repetitive trauma. Other important information to aid in the decision-making includes: patient age, hand dominance, activity level, and occupation.

Understanding the location of injury can also help narrow the differential diagnosis [7]. Pain along the medial aspect of the elbow carries a wide differential including: cubital tunnel syndrome, ulnar neuritis, ulnar nerve subluxation, ulnar collateral ligament (UCL) injury, and medial epicondylitis. Symptoms in the lateral region of the elbow can be due to: lateral epicondylitis, radial head fracture, osteochondritis dissecans (OCD) lesions, and lateral collateral ligament injury following a simple dislocation. The differential diagnosis for symptoms at the anterior elbow in includes: distal biceps rupture and anterior capsular strain. Posterior elbow pain can result from olecranon bursitis or fracture, triceps tendon pathology, loose bodies, and valgus overload extension.

25.3.2 Physical Examination

As with all upper extremity evaluations, a thorough cervical spine examination should be performed first. Findings of radiculopathy and myelopathy should warrant further workup, including cervical spine imaging. It is also important to examine the ipsilateral shoulder and contralateral elbow.

The physical examination of the elbow is no different from any other examination of a joint: inspection, palpation, range of motion, and joint-specific special testing. Upon inspection, one should first look for swelling or fullness of the joint. Depending on the location, the differential can include: radial head or olecranon fracture, olecranon bursitis, triceps avulsion, synovitis, ulnar collateral ligament injury or avulsion, and distal biceps rupture. Erythema of the joint should prompt the physician to be wary of an infectious or inflammatory process.

Although the location of the patient's symptoms may lead the physician to focus on the afflicted area, it is important to palpate all four regions of the elbow. This helps ensure that coexistent injury or pathology will not be missed.

Range of motion is evaluated in two planes: flexion and extensions and pronation and supination. Active and passive range of motion should be recorded accurately. Morrey et al. [8] evaluated 33 normal patients and the amount of elbow motion needed to carry out activities of daily living. Most activities can be accomplished with 100° of elbow flexion (30–130°) and 100° of forearm rotation (50° of pronation to 50° of supination). Range of motion outside of these parameters can indicate a significant functional disability.

Specific testing of the elbow generally revolves around assessing stability. The two most common forms of instability are valgus instability and posterolateral rotatory instability (PLRI). Valgus instability is generally seen following ulnar collateral ligament insufficiency, in particular the anterior bundle [9]. The moving valgus stress test as described by O'Driscoll et al. [10] is commonly used to assess valgus stability. This is performed by having the examiner apply and

maintain a valgus load to the flexed elbow and then quickly extending it. The test is considered positive if this reproduces the patients medial elbow pain and is generally seen between 70° and 120° of flexion.

Posterolateral rotatory instability can be assessed by the lateral pivot-shift test. With the patient supine and affected extremity over head, the forearm is supinated, and valgus to varus stress is applied while the elbow is flexed. A positive test is defined by reproduction of symptoms, signs of apprehension, or subluxation.

25.4 Essential Radiology

Three views (anteroposterior, lateral, and oblique) of the elbow are evaluated. These are useful in identifying fractures, loose bodies, osteophytes, and osteochondral lesions.

Advanced imaging studies such as MRI are useful to evaluate soft tissue structures about the elbow. In particular, the collateral ligaments and chondral injuries can be closely scrutinized. Nondisplaced fractures and common extensor tendons tears can also be assessed. Arthrography can be a useful adjunct in diagnosing collateral ligament injuries.

25.5 Indications and Contraindications

Classic indications for elbow arthroscopy included diagnostic arthroscopy and removal of loose bodies. As instrumentation and techniques continue to evolve, indications for elbow arthroscopy have expanded to: capsular release, synovectomy, lateral epicondylitis, treatment of OCD lesions of the capitellum, and certain intraarticular fractures.

The primary contraindication for elbow arthroscopy is any change in the patient's normal anatomy in whom arthroscopy carries a high risk of neurovascular injury [11]. Other contraindications include prior ulnar nerve transposition and erythema or soft tissue infection around the elbow.

25.6 Surgical Considerations

25.6.1 Anesthesia

Either general or regional anesthesia may be used for elbow arthroscopy. The advantages of general anesthesia include various options for patient positioning and total muscle relaxation. Disadvantages include the potential for greater postoperative pain and a longer post anesthesia recovery.

Regional anesthesia, with or without intravenous sedation, includes interscalene block and axillary block. The advantage of regional anesthesia is that it optimizes postoperative pain control, minimizes postoperative nausea, and facilitates positioning of the patient. The main disadvantage of regional anesthesia is the inability to perform a postoperative neurologic examination to determine whether nerve injury has occurred. At our institution, we typically use general anesthesia and reserve a regional block postoperatively, once the patient completes a full neurologic exam.

25.6.2 Instrumentation

The arthroscopic systems used in the larger joints (4.0-mm, 30° offset arthroscope) allow good visualization of the elbow. The smaller 2.7-mm arthroscope is typically not employed but may be useful to visualize smaller spaces such as the lateral compartment from the direct lateral portal. It is important to use elbow cannula systems that are compatible with both the 4.0- and 2.7-mm arthroscopes to enable switching between viewing and working portals without repeated injury to the elbow capsule and risk to the neurovascular structures. Non-vented cannulas should be used in order to decrease fluid extravasation into soft tissue, and trocars should be conical and blunt tipped in order to decrease the risk of neurovascular injury.

Arthroscopy pumps control irrigation and distention of the joint by maintaining a selected pressure (typically 35 mmHg) and fluid flow rate. The ideal arthroscopy pump will sustain a

constant pressure by automatically decreasing flow when the pressure increases over the preset value. When the pressure returns to the desired range, the pump will automatically start fluid flow to avoid loss of distension and visibility.

We typically use a combination of handheld instruments (e.g., probes, graspers, switching sticks, pointed awls, curved osteotomes) as well as motorized instruments (e.g., synovial resectors, radial end-cutting shavers, burrs, electrocautery devices).

25.6.3 Patient Positioning

A variety of patient positioning has been described, each with their inherent advantages and disadvantages.

25.6.3.1 Prone Position

The patient is positioned prone on chest rolls, and the arm is stabilized by an arm holder and allowed to hang off the table. The shoulder is abducted to 90°, and the elbow is flexed to 90°. Advantages of this position include easy manipulation of the elbow from flexion to full extension and increased safety of anterior portals due to anterior sagging of neurovascular structure. The main disadvantage of the prone position is that general anesthesia is required due to poor access to the airway.

25.6.3.2 Lateral Decubitus

The patient is positioned lateral, with the shoulder flexed forward at 90° over a padded bolster and the elbow allowed to hang freely flexed. The lateral decubitus position affords the same advantages of the prone position: improved arm stability and posterior joint access, while allowing easier access to the airway. The main disadvantage of this position is that repositioning may be necessary in order to access the anterior compartment or to transition to an open procedure.

25.6.3.3 Supine/Supine-Suspended (Author's Preferred Position)

Initially, elbow arthroscopy was performed with the patient in the supine position, with the arm placed on an arm board and laid across the body. However, this has been largely replaced by the supine-suspended position. In this position, the patient's shoulder is placed in 90° of abduction, with the elbow flexed 90° and the forearm, wrist, and hand suspended by a mechanical traction device [6]. We use a modification of this position, with the shoulder flexed 90° such that the forearm is suspended over the chest (Fig. 25.2a, b).

Advantages to this position include easy access to both the anterior and posterior compartments due to the ability to adjust the position of the arm in space, excellent medial and lateral access, and safety to anterior neurovascular

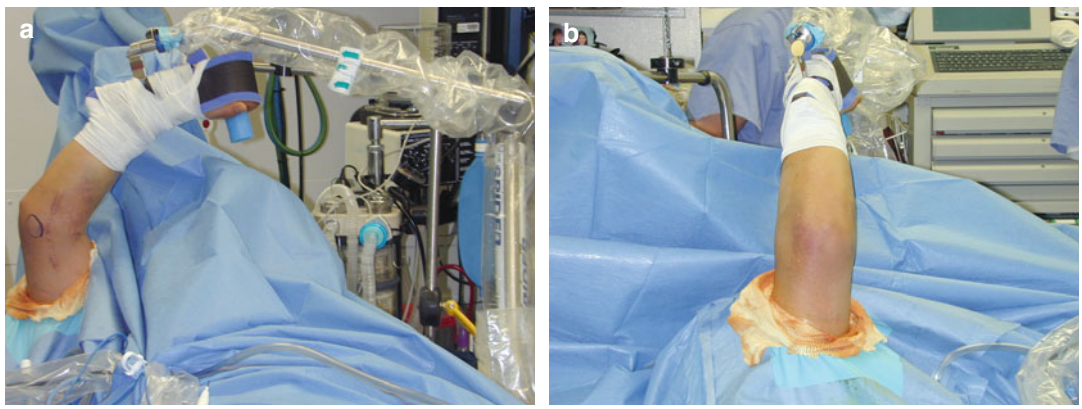

Fig. 25.2 Surgical setup for the modified supine position. The arm is placed in a holder (spider limb positioner). (**a**) Front view. (**b**) Side view

structures, which drop away from the anterior capsule with the arm flexed over the chest. Furthermore, with the patient positioned supine, the anesthesiologist has excellent access to the airway. If conversion to an open procedure is necessary, the arm can be removed from the holder and placed across the arm board.

25.6.4 Portal Placement

A number of different portals have been described in elbow arthroscopy. We most commonly employ the midlateral, proximal lateral, proximal medial, posterolateral, and transtriceps portals.

25.6.4.1 Midlateral Portal (Direct Lateral Portal)

The midlateral portal is located on the lateral "soft spot", which is bounded by the lateral epicondyle, radial head, and olecranon process which is often used initially to distend the joint. This portal can also be used as a viewing portal for work in the posterior compartment.

25.6.4.2 Proximal Medial Portal

The proximal medial portal is located just anterior to the intermuscular septum and 2 cm proximal to the medial epicondyle. This portal allows excellent visualization of the anterior compartment, especially the radiocapitellar joint, and is often the first working portal established. The portal must be placed anterior to the intermuscular septum to avoid injury to the ulnar never, which lies 3–4 mm posterior to the intermuscular septum [12].

25.6.4.3 Proximal Lateral Portal

The proximal lateral portal can be placed anywhere from the sulcus between the radial head and capitellum to a point 2 cm proximal to the later epicondyle along the anterior aspect of the humerus. This portal is used to visualize the medial aspect of the elbow, the radiocapitellar joint, and the lateral recess. The risk of radial nerve injury decreases as the portal is placed more proximally.

25.6.4.4 Posterolateral Portal

The posterolateral portal is located 2–3 cm proximal to the tip of the olecranon at the lateral border of the triceps tendon. This portal is used to visualize the olecranon fossa, olecranon, and posterior trochlea. The medial antebrachial cutaneous nerve is approximately 25 mm from this portal [13].

25.6.4.5 Straight Posterior Portal (Transtriceps Portal)

The straight posterior portal is located 3 cm proximal to the tip of the olecranon in the midline. This portal is introduced through the triceps just above the musculotendinous junction. Although typically used as a working portal, it can also provide excellent visualization of the posterior compartment.

25.7 Author's Preferred Surgical Technique

Following anesthesia administration and patient positioning, the bony landmarks, neurovascular structures, and the portal sites are marked (Fig. 25.3a–c). The elbow joint is then distended with 20–30 mL of saline, typically through the soft spot in the midlateral portal. Distending the joint shifts facilitates the safe entry of the instruments; however, overdistension of the capsule can lead to capsular rupture and an inability to maintain adequate fluid pressure during the procedure.

It is important to remember that while capsular distention increases the distance between the joint and the neurovascular structures, the relationship between the neurovascular structures and the capsule remain the same. We also employ switching sticks when changing from one portal to another. This minimizes repetitive capsular disruption as well as risk of neurovascular damage.

25.7.1 Anterior Arthroscopy

We introduce the arthroscope through the proximal lateral portal into the anterior compartment,

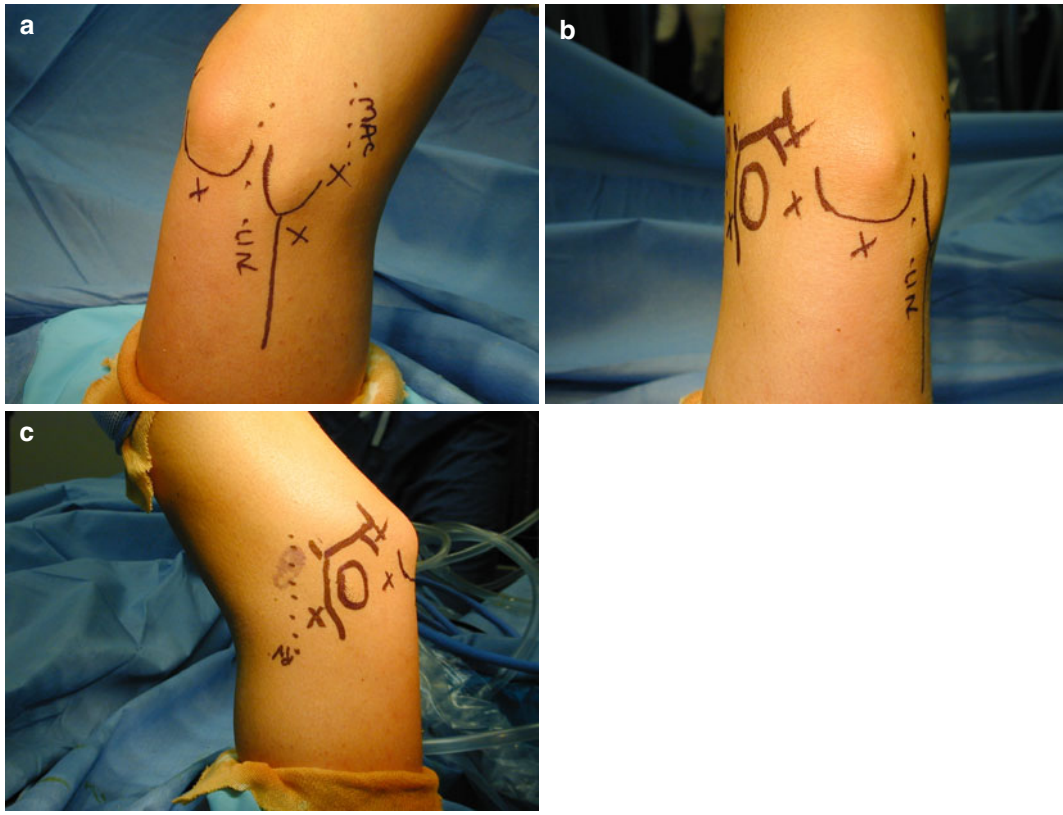

Fig. 25.3 Elbow portal anatomy. Common portals marked with "X." (**a**) Medial view demonstrating proximal medial portal. It is important to stay anterior to the intermuscular septum. The ulnar nerve (UN) and medial antebrachial cutaneous nerve (MAC) are represented by the dotted lines. (**b**) Posterior view demonstrating the transtriceps portal and the posterolateral portal. (**c**) Lateral view demonstrating the proximal lateral portal. The dotted line represents the radial nerve

and a systematic diagnostic arthroscopy of the anterior compartment is performed. The articular cartilage and synovium are evaluated for the presence of loose bodies, the coronoid process is examined for osteophytes, and the anterior trochlea and coronoid fossa are examined for cartilage lesions. The anterior radiocapitellar joint is evaluated for osteochondral lesions of the capitellum and any matching pathology of the radial head. Importantly, the radial nerve lies on or within a few millimeters of the anterolateral joint capsule, so debridement in this area requires extreme caution.

In cases in which ulnar collateral ligament insufficiency is suspected, the arthroscopic valgus stress test is done during assessment of the anterior compartment. With the arthroscope in the proximal lateral portal visualizing the medial aspect of the ulnohumeral joint, valgus stress is applied while the elbow is in 70° of flexion. Ulnar collateral ligament insufficiency is present if this maneuver produces an opening between the ulna and the humerus of >3 mm [14]. When intervention is warranted in the anterior compartment such as with the removal of loose bodies (Fig. 25.4a–d), capsular release, synovectomy, or debridement, a proximal medial portal is established under direct visualization.

25.7.2 Posterior Arthroscopy

Following completion of the anterior arthroscopy, a posterolateral portal is established. The camera is switched from the anterior cannula and

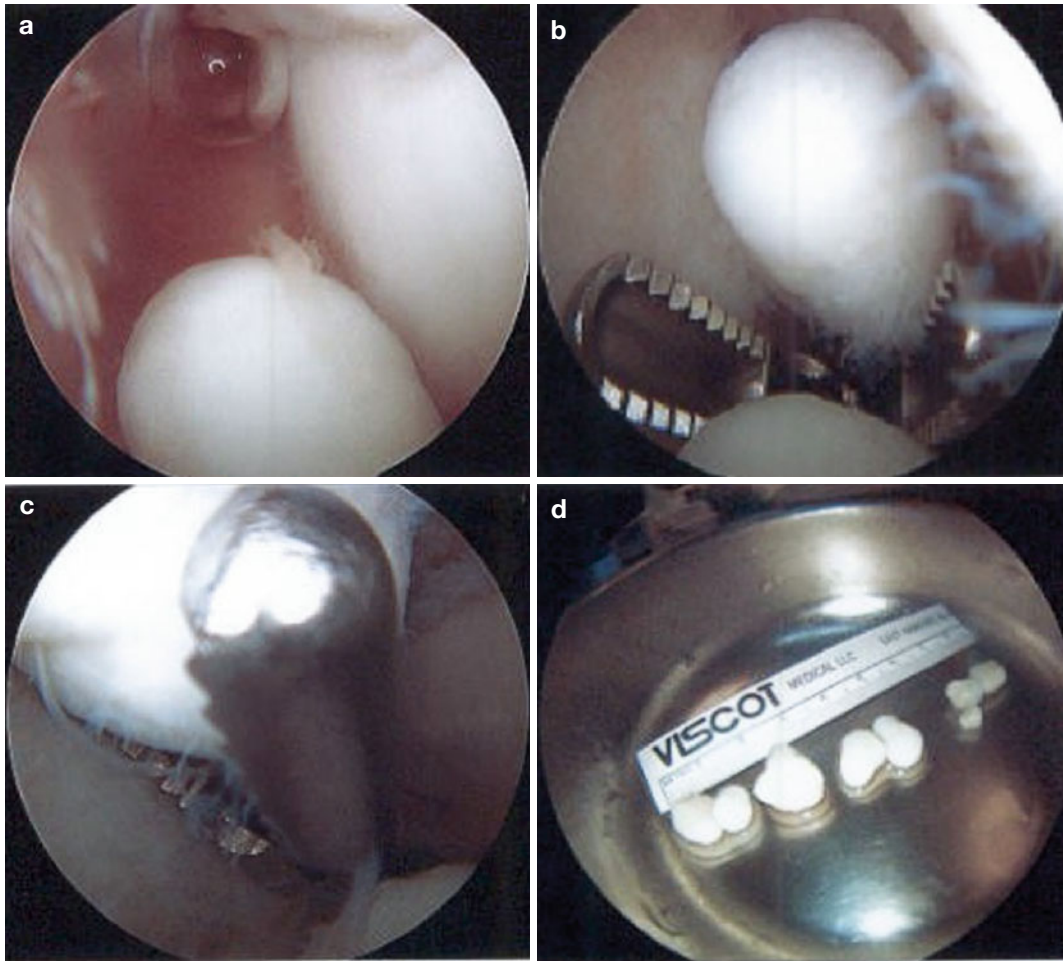

Fig. 25.4 Elbow arthroscopy for loose bodies. (**a**, **b**) Demonstrating intra-articular loose bodies. (**c**) Retrieval of loose bodies with an arthroscopic grabber. (**d**) Intraoperative photograph using the arthroscope demonstrating the loose bodies retrieved, using a ruler as reference

inserted into this portal. We typically maintain the anterior cannula to facilitate reentry into the anterior compartment, if necessary. The olecranon is evaluated for the presence of osteophytes. The corresponding olecranon fossa and posteromedial aspect of the humeral condyle are evaluated for matching chondral defects. The posterior radiocapitellar joint and the lateral aspect of the ulnohumeral joint are evaluated by advancing the arthroscope down the lateral gutter.

Conclusion

Elbow arthroscopy is an evolving tool in the diagnosis and management of elbow pathology. With evolving techniques, the indications for elbow arthroscopy have expanded considerably over the past few decades. Due to the proximity of neurovascular structures, it is critical that the surgeon has a thorough understanding of the relevant anatomy to ensure a safe and successful procedure.

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Arthroscopic Treatment of Lateral Epicondylitis

26

John Jennings, Rick Tosti, and J. Milo Sewards

26.1 Introduction

Runge first described lateral epicondylitis in 1873, and the term "tennis elbow" was attributed to a description by Morris about 10 years later with reference to a small group of symptomatic tennis players [1]. Since its initial description, the understanding of the pathoanatomy, histology, and treatment strategies continues to evolve.

Lateral epicondylitis is a common cause of elbow pain, affecting 1–4 % of the general population and up to 20 % of those engaging in manually intensive occupations. Workers engaging in repetitive movements requiring large handgrip forces or utilizing vibrating tools are particularly susceptible [2–4]. While tennis players account for fewer than 10 % of those with this condition, over 50 % of players will experience some degree of symptoms in their lifetime, which is often attributed to excessive eccentric loading of the wrist extensors particularly during the one-handed backhand stroke [5–7].

Most cases of tennis elbow are self-limiting, although up to 8–10 % will eventually require surgery [8, 9]. Although several variations of surgical techniques have been described, which include open, arthroscopic, and percutaneous approaches, none have proven superiority in outcome studies [9]. Recently, arthroscopic treatment has gained popularity as reports have favored the approach to the undersurface of the extensor carpi radialis brevis (ECRB); advantages include decreased dissection, concurrent access to intra-articular pathology, and possibly decreased recovery time [10].

26.2 Pathoanatomy, Biomechanics, and Preferred Classification

The extensor tendon origin, particularly the ECRB, is the chief location of pathology. Cadaveric studies highlight the deep position of the ECRB relative to the rest of the common extensor origin and postulate that this anatomic position accounts for the localization of degenerative change as it contacts the lateral edge of the capitellum during elbow motion [11, 12]. Tanaka et al. quantified these contact forces showing maximal contact with elbow extension, forearm pronation, and varus stress to the elbow [12].

In athletes, cyclical eccentric loading of the wrist extensors is thought to be responsible for stress overloading of the proximal extensor

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tendon origin. Studies utilizing electromyography, grip pressures, wrist kinematics, and computer simulations have shown that when novice players make ball contact during a backstroke, the wrist is often deviating into flexion, which requires an eccentric forearm muscle contraction in order to stabilize the wrist at the time of impact [13, 14]. In contrast, experienced players make contact with the wrist moving into extension, which is more energy efficient. Factors such as the use of vibratory tools and excessive gripping activity seem to exacerbate the resultant tendon microtears which ultimately result in degenerative changes.

While no universal classification system for tennis elbow currently exists, Baker et al. described an arthroscopic, intraoperative classification predicated on the extent of capsular tear: type 1 lesions had an intact capsule, type 2 lesions had a linear defect, and type 3 had a full capsular tear [9]. However, the authors found no correlation between lesion classification and outcome.

Nirschl and Ashman classified lateral epicondylitis according to the "pain phase" which described exacerbating factors and duration of symptoms (Table 26.1). These authors also identified four pathologic stages of tendinosis which is quantified intraoperatively (Table 26.2) [15]. The authors recommended that stage III and IV pathology requires surgery, which roughly correlates with pain phases VI and VII.

Table 26.1 Phases of pain [15]

Phase I	Mild pain after exercise lasting <24 h
Phase II	Pain after exercise lasting >48 h and resolving with warm-up
Phase III	Pain with exercise, does not alter activity
Phase IV	Pain with exercise that alters activity
Phase V	Pain caused by heavy activities of daily living
Phase VI	Intermittent pain at rest that does not disturb sleep; pain caused by light activities of daily living
Phase VII	Constant rest pain and pain that disturbs sleep

Table 26.2 Pathologic stages [15]

Stage I	Temporary irritation
Stage II	Permanent tendinosis; less than 50 % tendon cross section
Stage III	Permanent tendinosis; greater than 50 % tendon cross section
Stage IV	Partial or total rupture of tendon

26.3 Clinical Presentation and Essential Physical Exam

Patients with tennis elbow typically present in the fifth and sixth decades and often with a history of overuse injury and a high activity level. Men and women appear to be affected equally; manual laborers, especially those working with vibratory tools or requiring excessive gripping forces, are particularly at risk [4, 16]. A history of insidious onset of activity-related, lateral elbow pain is often elicited. The pain may occasionally radiate into the forearm. Symptoms may progress to grip strength weakness or difficulty with activities of daily living such as shaving, shaking hands, lifting grocery bags, or raising a coffee mug. A common complaint is pain while reaching to grab a handbag, groceries, or a briefcase from the backseat of a car.

Inspection of the lateral elbow may reveal mild swelling around a point of maximal tenderness, which is approximately 1 cm distal and anterior to the lateral epicondyle. Resisted wrist and long finger extension while the elbow is extended are generally provocative; reproduction of pain with these maneuvers while the elbow is flexed usually heralds more severe disease (Fig. 26.1a, b). The "chair test" is an additional provocative maneuver, which is considered positive when pain is elicited in the lateral elbow while the patient lifts the back of a chair with a pronated hand (Fig. 26.2) [17]. Elbow range of motion is generally preserved, and significant swelling is unusual.

Other causes of lateral elbow pain should be excluded; radicular pain from the neck, crepitus or clicking with elbow range of motion, and significant weakness of wrist extension are characteristic of other disorders (Table 26.3).

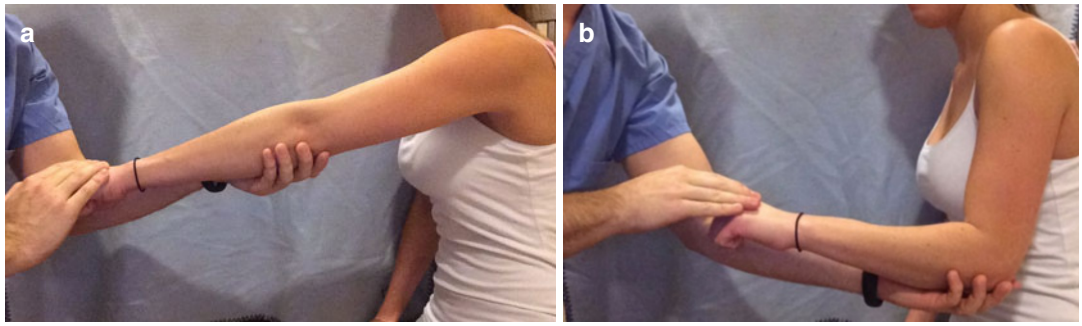

Fig. 26.1 (**a**, **b**) The patient actively extends the wrist against resistance with the elbows extended (**a**) and flexed (**b**). A positive test elicits pain over the lateral elbow. In mild disease, pain may not be present with the elbows flexed; if pain is present, it usually heralds more severe disease

Fig. 26.2 Chair test. The patient holds the back of a chair with the arm extended and hand pronated. Reproduction of pain over the lateral epicondyle with an attempt to raise the chair is considered a positive test

26.4 Essential Radiology

While lateral epicondylitis is principally a clinical diagnosis, standard elbow radiographs are typically obtained to exclude other pathology about the elbow. Typically plain film x-rays are normal, although findings consistent with soft tissue calcification or epicondylar exostosis are not uncommon [15]. Other studies such as ultrasound, MRI, and EMG may be useful for excluding other conditions, particularly in patients with atypical presentation.

Doppler ultrasonography as well as grayscale ultrasound may be utilized to visualize tendon tears, thinning, thickening, calcification, neovascularization, lateral collateral ligament damage, and lateral epicondyle irregularities. Ultrasound is less sensitive but equally as specific as MRI with reported sensitivity and specificity at 72–88 % and 36–100 %, respectively [18, 19]. Interestingly, Clarke et al. demonstrated that patients with ultrasound findings of large intrasubstance tendon tears and/or associated lateral collateral ligament tears were less likely to respond to nonoperative therapy [20].

Magnetic resonance imaging is seldom needed for diagnosing lateral epicondylitis. Images may show increased signal intensity around the extensor origin with varying degrees of tendon tearing, but the intensity of these findings has not been reliably correlated to the severity of the patient's symptoms [21]. In general, this modality is reserved for identifying intra-articular pathology.

26.5 Disease-Specific Clinical and Arthroscopic Pathology

Nirschl and Pettrone described gross specimens of the ECRB in patients with tennis elbow as "grayish, immature scar tissue which appears

Table 26.3 Differential diagnosis of lateral elbow pain

Pathology	History	Physical exam	Imaging
Cervical spondylosis	Radicular pain into the elbow Neck pain	Symptoms with spine compression/extension	XR + MRI of C-spine
Radial tunnel syndrome	Insidious onset of lateral elbow pain	Pain 2–4 cm distal to epicondyle	EMG + NCS[a]
PIN compression	Insidious onset of lateral elbow pain and weakness	Weakness of the wrist and finger extensors	EMG + NCS
Intra-articular loose bodies	Trauma Weight lifting	Clicking or limitation of range of motion	XR of the elbow
Chondral lesions	Trauma Weight lifting	Clicking or limitation of range of motion	MRI of the elbow
Tumors	Prior malignancy, night pain, constitutional symptoms	Palpable mass	XR + MRI of the elbow
Avascular necrosis	Sickle cell anemia, alcohol abuse, HIV, corticosteroids	Joint effusion, mechanical symptoms	XR + MRI of the elbow
Osteochondritis dissecans	Adolescent patients, gymnasts, throwers	Joint effusion, mechanical symptoms	XR of the elbow

PIN posterior interosseous nerve, *XR* x-ray, *MRI* magnetic resonance imaging, *EMG* electromyography, *NCS* nerve conduction studies
[a]EMG + NCS are rarely diagnostic in radial tunnel syndrome

shiny, edematous, and friable." Their histological study of this pathologic tissue revealed an "angiofibroblastic tendinosis" in which micro-tears in the tendon were replaced with disorganized immature collagen, fibroblasts, and vascular tissue [22–24]. As confirmed by subsequent authors, a lack of inflammatory cells was observed; however, this description is also consistent with that of normal tendons that were injected with corticosteroids, and no studies have confirmed the histologic appearance of a diseased tendon without corticosteroid exposure [25]. Arthroscopically, the tendon appears to have varying degrees of fraying, tearing, and avulsion with increasing severity of disease [9].

26.6 Treatment Options

Up to 95 % of patients presenting with tennis elbow will improve without surgery [15]. Although the optimal conservative therapy for lateral epicondylitis is unclear, it is reasonable to approach the patient with a combination of brief rest and nonsteroidal anti-inflammatory drugs followed by physical therapy, bracing, and injections if needed.

26.6.1 Nonoperative Treatment

26.6.1.1 Physical Therapy, Bracing, and Activity Modification

Successful results have been described with physical therapy regimens utilizing a combination of stretches, targeted massage, strengthening, and hot-cold modalities [15, 26–28]. Eccentric muscle training has demonstrated excellent results with subjective improvement in both pain and strength [29]; resistance physiotherapy is the authors' preferred modality. Newer treatments such as extracorporeal shock wave therapy and topical glyceryl trinitrate patches do not have long-term data to support their routine use at this time [28, 30].

Physiotherapy may also include sport-specific technique and equipment modifications. When compared with professionals, amateur players experience tennis elbow symptoms more frequently which suggests that poor technique is contributory [2, 13, 14, 31]. A tennis coach can teach the player to concentrate the core muscles into the forehand swing by striking the ball while it is still in front of the player. Additionally, in a one-handed backstroke, the coach should focus on making sure the player extends the wrist

during ball contact; striking the ball while the wrist falls in flexion creates a greater eccentric load on the wrist extensors. Alternatively, a 2-handed backstroke can also help unload the tension across the dominant wrist. Equipment should also be inspected. Lighter rackets made of low-vibration materials (graphite or epoxies) and rackets that are less tightly strung and/or have more strings per unit area can also reduce the work of the wrist extensors during active play [32]. A proper grip size is also recommended; a good estimate of the racket handle circumference is to measure the distance from the players proximal wrist crease to the tip of the ring finger. Last, playing on "slower" surfaces, such as a clay court, may also reduce the forces experienced across the wrist.

Orthoses can be an adjunctive therapy for tennis elbow but are not usually prescribed as a monotherapy. Counterforce straps are wrapped tightly around the upper forearm in an attempt to move the mechanical origin of the wrist extensors more distal; thus the player, in theory, can still move the wrist while resting the proximal portion of the muscles [28, 33]. Cock-up wrist splints are another option, which restrict wrist extension. Currently, no orthosis has been proven superior [34].

26.6.1.2 NSAIDs and Injections

A recent Cochrane review examined 13 trials that reported on the efficacy topical and oral NSAIDs for symptom relief in lateral epicondylitis and could not recommend for or against their use. Symptom alleviation in some patients was offset by the side effects of stomach pain and diarrhea with oral tablets and skin rash with topical applications [35].

Steroid injections alleviate elbow pain and assist with early return to athletic or work activity, although no long-term benefit has been established [28, 36]. Comparative studies have not found a superior steroid preparation, although universal risks for all steroid injections are skin depigmentation, fat atrophy, tendon rupture, and transient hyperglycemia in diabetics [37, 38].

Platelet-rich plasma (PRP) and whole blood injections are an alternative to steroids and have been used with more frequency in recent years. A prospective study by Mishra et al. with 230 participants demonstrated clinical improvements at 24 months compared with controls [39]. Gosens et al. also showed greater clinical improvement with PRP compared to corticosteroids at 2 years [40]. Thanasas et al. was unable to demonstrate better outcomes with PRP compared to whole blood injections at 6 months [41]. Currently, the optimal timing, concentration, and number of doses of these biologic injectables are not known, and the cost can be prohibitive for some patients.

26.6.2 Operative Treatment

Surgery is reserved for patients who do not improve with 6–12 months of nonoperative therapy. Excellent results have been documented with open, percutaneous, and arthroscopic approaches to the lateral elbow [42]. Overall, long-term pain relief following surgery ranges from 19 to 100 % [43, 50]. The average failure rate is 5.8 % [3], and return to sport following surgery averages 66 days [42]. Arthroscopic release has reported success rates of 93–100 % [50] with an average return to sport at 35 days [42]. No single technique has been proven superior for either relief of symptoms or return to play [46].

The "mini"-open release is the most common approach, where the ECRB origin is released through a 3 cm incision over the lateral epicondyle. The interval between the extensor carpi radialis longus and the extensor digitorum communis is incised and raised subperiosteally. The ECRB origin should then be exposed and the degenerated tissue should be examined and graded. Some surgeons prefer to resect the tendon while others may detach it, debride the undersurface, and reattach it with suture anchors. In both cases, the lateral epicondyle is usually decorticated prior to repair/closure in order to stimulate tissue healing. Strict protection of the lateral ulnar collateral ligament is critical in maintaining elbow stability.

The percutaneous tenotomy is performed through a 1 cm incision in the office or in the operating room. The procedure does not include removal of pathologic tissue and can therefore predispose the patient to recurrence of symptoms. Advantages include convenience and short recovery times [43, 44].

Arthroscopic debridement of the common extensor origin for tennis elbow is becoming a more popular procedure in the sports population. Advantages include minimal dissection, quicker recovery times, and the ability to address concomitant intra-articular pathology [45]. Traditionally, the release is performed through two anterior portals, but an additional posterior portal can be made to remove loose bodies [10, 45, 47].

26.7 Author's Preferred Treatment

We recommend a 1–2 week period of cessation of aggravating activities and prescribe a course NSAIDs as long as no contraindications exist. The patient then begins a 6–8-week course of physiotherapy that focuses on progressive resistance strengthening of the wrist extensors. The therapy is combined with hot-cold modalities, stretching, and massage. If therapy is limited by pain or if the athlete anticipates overexertion during a competition, we offer an injection with dexamethasone. The injection is given at the point of maximal tenderness and redirected multiple times within the tissue to stimulate bleeding. Depending on the progress of the therapy, a home exercise program is begun to continue the strengthening exercises, or we try a second round of therapy with a trial of iontophoresis and dexamethasone. In a select group of athletes or in refractory cases, we may offer a platelet-rich plasma injection.

If the patient has been compliant and has failed the nonoperative protocol, surgery is offered. We prefer the arthroscopic approach for the aforementioned benefits; however, we would not recommend this approach in patients who have distorted anatomy or a history of ulnar nerve transposition. A subluxing ulnar nerve is a relative contraindication; arthroscopy can still be performed as long as the nerve is identified prior to making the medial portals. We generally do not perform a preoperative block because it interferes with the postoperative neurologic examination. We use a 4.0 mm, 30° arthroscope with an end-vented cannula to prevent soft tissue fluid extravasation. The arthroscopic pump is pressure-regulated and usually preset to 30 mmHg. The patient is positioned in the lateral decubitus position with the elbow flexed over a padded post. A tourniquet is applied. The joint is then insufflated with 30–50 ml of saline injected through the anconeus triangle (soft spot between the radial head, olecranon, and lateral epicondyle) to push the neurovascular structures away from the joint. We start by establishing the anterosuperior lateral portal, which is 2 cm superior to the anterior surface of the lateral epicondyle. The trochar is guided over the anterior surface of the humerus toward the center of the joint. The brachioradialis, brachialis, and capsule are pierced. We prefer this portal for the relative safety of surrounding nerves. We introduce the arthroscope into this portal and then establish a proximal medial portal which starts 2 cm proximal to the medial epicondyle and anterior to the intermuscular septum. The trochar also rides the anterior humeral surface in line with the median nerve to reduce the risk of injuring it. The arthroscope is then inserted into the medial portal and a diagnostic arthroscopy is performed. The anterior joint is visualized including the anterior capsule, trochlea, capitellum, radial head, and medial and lateral gutters. The severity of degeneration of the ERCB is noted (Fig. 26.3). The shaver or electrocautery is then placed through the proximal-anterolateral portal, and the undersurface of the ERCB is debrided (Fig. 26.4). Once the degenerative tissue is removed, the lateral epicondyle may be lightly abraded with a burr. The joint is irrigated, the portals are closed with suture, and the elbow is placed into a soft dressing.

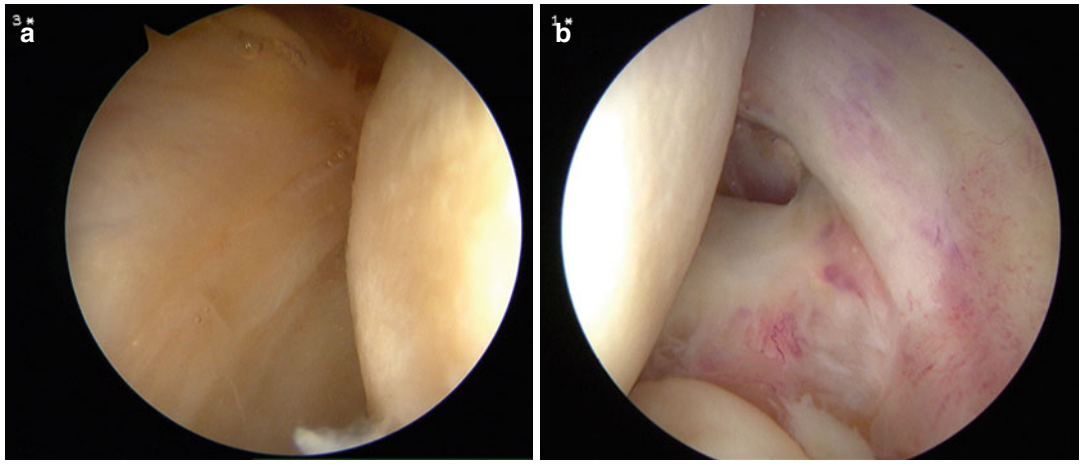

Fig. 26.3 (**a**, **b**) Arthroscopic images of a (**a**) mild and (**b**) severe degeneration of the ERCB tendon

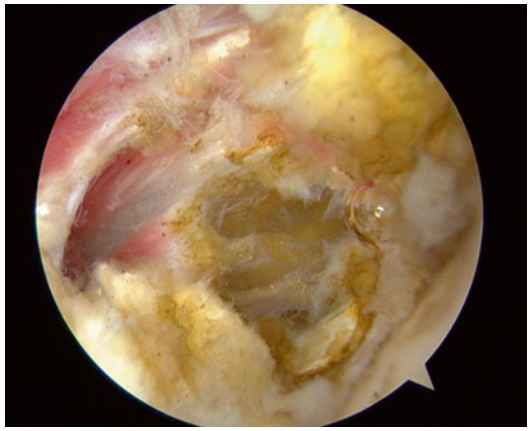

Fig. 26.4 After debridement with shaver and electrocautery

26.8 Rehabilitation

Rehabilitation after arthroscopic ECRB release is usually informal with active range of motion beginning on the first postoperative day. Most surgeons allow return to normal level of activity as tolerated by the patient; transition to heavy or repetitive work is generally restricted for 6 weeks [45, 47–49]. Baker et al. advocated for isometric strengthening beginning as soon as full range of motion is achieved at the elbow, resistance strengthening at 4–6 weeks, and unrestricted activity at 6–8 weeks postoperatively [9].

26.9 Advantages, Pitfalls, and Complications

Advantages of arthroscopic release include excision of diseased tissue under direct visualization, preservation of the common extensor origin, early return to activity, and ability to address intra-articular pathology, which may be present in 11–69 % of cases [9, 46, 47, 50, 51].

Arthroscopic debridement of the ECRB is more technically challenging than the open or percutaneous release techniques with a significant learning curve. The most feared complication is iatrogenic nerve injury, which has been reported in 0–14 % of case series [52, 53]. During placement of the proximal anteromedial portal, the posterior branch of the medial antebrachial cutaneous nerve is most at risk, but injury to the ulnar nerve results in the most severe consequences. It is critical to determine if the patient has a history of a subluxing nerve or a nerve transposition. If the surgeon still wishes to proceed arthroscopically, the nerve should be visualized prior to placing the portal. In native elbows, the ulnar nerve is usually not threatened as long as the portal is placed anterior to the intermuscular septum. The median nerve is less likely to be injured and is usually safe as long as the trochar is directed parallel to the nerve trajectory. With the proximal-anterolateral portal, the

posterior branch of the lateral antebrachial cutaneous nerve is most at risk. The radial nerve may also be at risk, but this portal is farther from the radial nerve than the standard anterolateral portal, which is why we prefer it.

Other complications include elbow instability or recurrence of symptoms. Overaggressive debridement resulting in destabilization of the lateral elbow occurs if the debridement violates the lateral ulnar collateral ligament [9, 49, 54, 55]. However, the most commonly incurred complication is incomplete resection of the pathologic tissue [15, 48].

26.10 Experience in Treatment of Athletes

A 35-year-old personal trainer and competitive weight lifter presented with a complaint of a deep aching pain localized to the lateral aspect of the right elbow. He reported exacerbations of sharp pain that occurred with grasping weights to load a barbell. His pain was affecting his ability to perform a power clean and interfered with his job as a personal trainer. Initially, he was started on an eccentric strengthening therapy protocol with modalities and a counterforce strap during activities, but this strategy only achieved mild relief. At his follow-up visit, he was given a corticosteroid injection and sent for another round of therapy, which also included iontophoresis.

After 3 months of nonsurgical therapy, we recommended surgery, as he noted little improvement with current course of treatment and a significant hindrance to his life. We chose an arthroscopic approach because of his large body habitus and an anticipated faster return to his athletic profession.

Under general anesthesia, a diagnostic arthroscopy of the right elbow was performed in the left lateral decubitus position. We found a Nirschl grade 2 lesion of the lateral capsule and ECRB with no other intra-articular pathology. A 3.0 mm shaver and intra-articular electrocautery were used to resect the lateral capsule and release the origin of the ECRB. Postoperatively, the patient was instructed in self-directed range of motion exercises and was seen in follow-up at 2-, 4-, and 6-week intervals. At his 2-week follow-up, he had full pain-free range of motion and was permitted to begin a progressive return to his forearm and elbow strengthening activities. At 4 weeks, he had returned to his regular weight lifting routine without pain and returned to competitive weight lifting just after his 6th postoperative week.

Conflict of Interest Statement Each author certifies that he or she has no commercial associations (e.g., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted article.

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Osteochondritis Dissecans of the Throwing Elbow

27

Kozo Furushima, Shohei Iwabu, and Yoshiyasu Itoh

27.1 Introduction

Osteochondritis dissecans (OCD) of the capitellum, a sports disorder of the elbow joint that occurs during the growth phase, occurs in approximately 2–8 % of school-age baseball players in Japan. Approximately 800 patients with baseball elbow visit the authors' hospital annually. However, OCD has recently accounted for approximately 12 % of our patients in baseball-related disorders. The common age at onset is 11 years, prior to closure of the epiphyseal line. Although patients with OCD show a decreased range of motion (ROM) before they display clinical signs, they generally do not notice it. Unexpectedly, the initial onset of OCD is not associated with pain. Most patients complain of pain with throwing after the lesion becomes relatively large. Since the advanced clinical condition leads to arthropathic changes, it is important to detect and treat OCD at its early stages. In fact, early detection is the most effective strategy for the treatment of OCD. Thus, in Japan, the importance of screening examination in the field has also been stressed recently.

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Improvement of blood flow to the subchondral bone and bone regeneration are important for lesion repair. Patients typically receive conservative treatment such as topical rest to reduce physical stress and improve pitching form through rehabilitation before returning to sports. For patients whose condition is resistant to conservative treatment, surgical treatment is performed for local blood flow improvement and osteochondral reconstruction.

This chapter describes the clinical condition of, treatment methods for, and the authors' operative procedures for OCD.

27.2 Pathoanatomy, Preferred Reference

27.2.1 Pathophysiology

There are some assumptions for the causes of OCD of the humeral capitellum in juvenile baseball players: inflammatory reaction, hematogenous disorder, endocrine abnormality, traumatic nutritional disorder, and genetic makeup and predisposition. However, they remain poorly understood. Many etiologies have been reported by a large number of researchers. Patients with Panner disease, which consists of osteonecrosis-related lesions in the ossification center of the capitellum, tend to be younger than patients with OCD. Panner disease shows changes in the entire ossification center that recover spontaneously within 1–2 years, which is distinct from OCD.

J.-Y. PARK (ed.), *Sports Injuries to the Shoulder and Elbow*,
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Konig [12] reported that these changes did not result from trauma only and that inflammation of the bone and cartilage should be considered the cause of dissection; therefore, he named it "osteochondritis dissecans." Haraldsson et al. [3] obtained images of the elbow joint of infant cadavers in which the contrast agent was injected and reported that there was no clear blood vessel penetrating the epiphyseal line and that nutrients were supplied via 1–2 blood vessels penetrating the posterior epiphyses during the advanced ossification period of the ossification center of the humeral capitellum. Thus, they suspected that such patients were likely to suffer from ischemia because the humeral capitellum has a poor nutrient supply.

Neilson [7] examined the elbow joint on radiographs of 1,000 healthy men and reported evident OCD findings in 4.1 % of all men and 14.6 % of blood relatives of these patients with OCD; thus, he advocated the association with genetic makeup and predisposition. We cannot deny patients' genetic factors considering the following cases: OCD occurs in some sites other than the capitellum such as the trochlea, radial head, and olecranon; patient's brothers also suffer from OCD; the diseased site was opposite that of the throwing elbow; and the condyle of the knee joint is complicated by OCD. Considering that OCD is frequently associated with a throwing motion, it may also be caused by repeated small injuries on the subchondral marrow, cartilage, and border regions due to compression and the shear force of the radial head into the capitulum of the humerus. It is speculated that necrosis of the subchondral bone marrow advances and then fissure occurs on the articular cartilage secondarily, leading to advanced destruction.

Currently, the way that OCD lesions are formed is a matter of speculation. Cases in which OCD occurs in a site on the non-handed side of athletes are extremely rare. Therefore, repeated direct load rather than genetic and intrinsic factors is thought to be the main cause.

27.2.2 Clinical Condition

Plain radiographs show that OCD lesions originate from the lateral aspect of the capitellum as a small translucency prior to epiphyseal closing. The lesion gradually spreads to the center and then is recovered from the outside (Figs. 27.1, 27.2, and 27.3).

Imaging findings vary depending on exacerbation or repair (Fig. 27.4). If the repair process stops before the entire lesion heals, lesions will be left within the central portion of the capitellum. The epiphyseal line is usually already closed (Fig. 27.4, case 1). When the repair process stops to soon, the lateral walls will not be sufficiently repaired (Fig. 27.4, case 2). When the repair process does not advance, extensive lesions will persist on the entire capitellum from the outside to the center (Fig. 27.4, case 3). In this case, extensive reconstruction of the articular surface will be required.

27.2.3 Preferred Classification

Minami et al. [5] reported a plain radiographic classification for OCD for the first time (1979). Based on plain radiographic images, the classification was defined as follows (Fig. 27.5): translucency type (stage I), translucency of localized bone on the humeral capitellum; separation type (stage II), perifocal bone sclerosis or a clear zone between the lesion and surrounding bone tissues; and free type (stage III), the lesion is completely detached from the humeral capitellum and exists as a free body in the joint.

The conditions for acquiring images were defined as 45° flexion view (tangential view) to assess the disease stages in detail: translucency period, exterior type and central type; separation period, first-term type and latter-term type; and free body period, internal or external to the lesion.

Based on lesion sites, it can be classified as follows: central localized type, external localization type ranging from the outer wall of the lateral epicondyle to less than one-third of the articular surface on the radial head, and extensive type, ranging from the outer wall of the lateral epicondyle to one-third or more of the articular surface on the radial head. It is important to understand the lesion site and its range in surgical treatment.

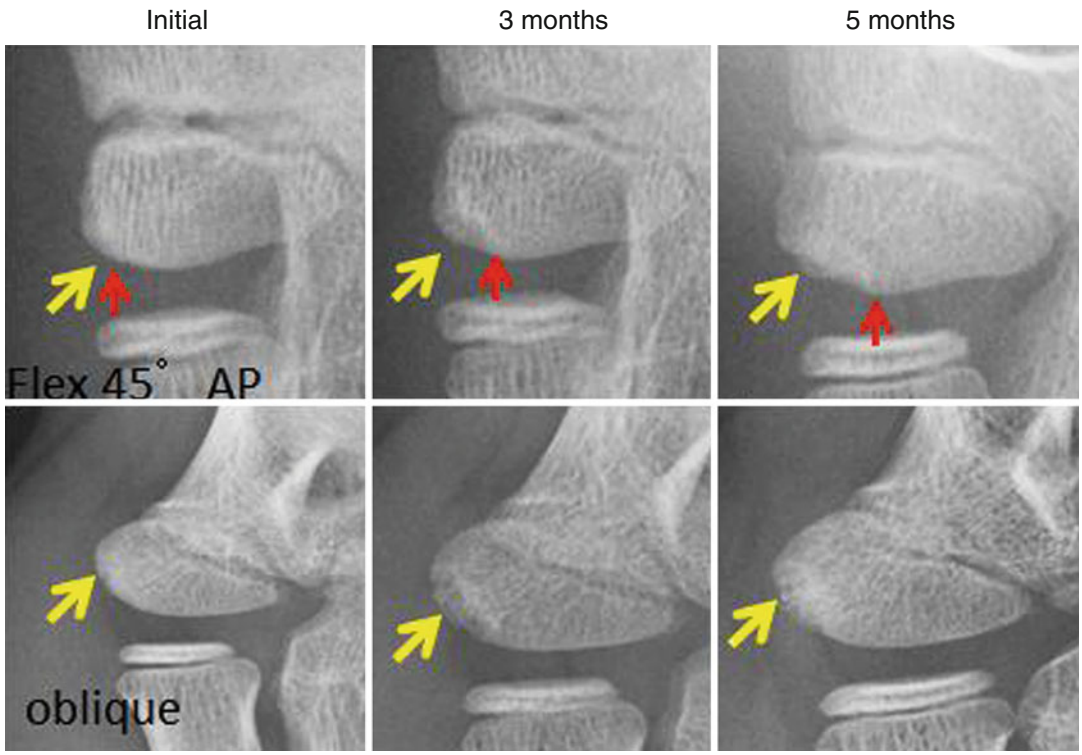

Fig. 27.1 Osteochondritis dissecans during the initial asymptomatic phase. *Upper*: the elbow at AP 45° of flexion (tangential view). *Lower*: the elbow in oblique view at 45°. *Yellow arrow*: lesion. *Red arrow*: lesion boundary

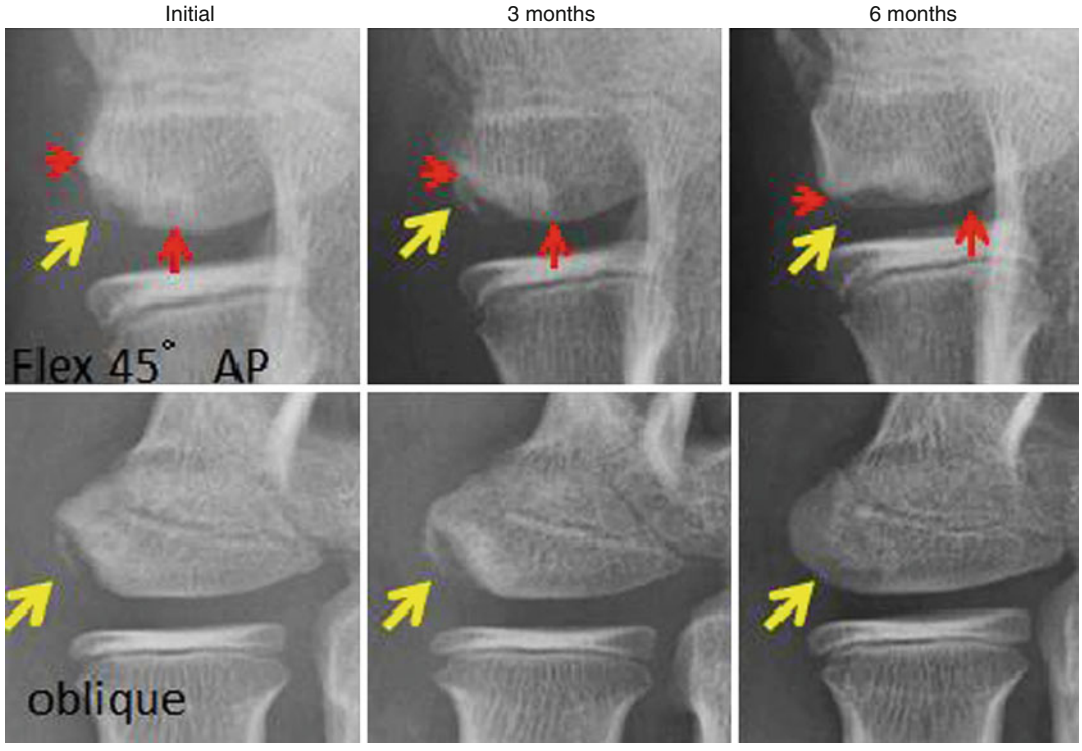

Fig. 27.2 Osteochondritis dissecans discovered after throwing-related pain is reported. *Upper*: tangential view. *Lower*: oblique view. *Yellow arrow*: lesion. *Red arrow*: lesion boundary

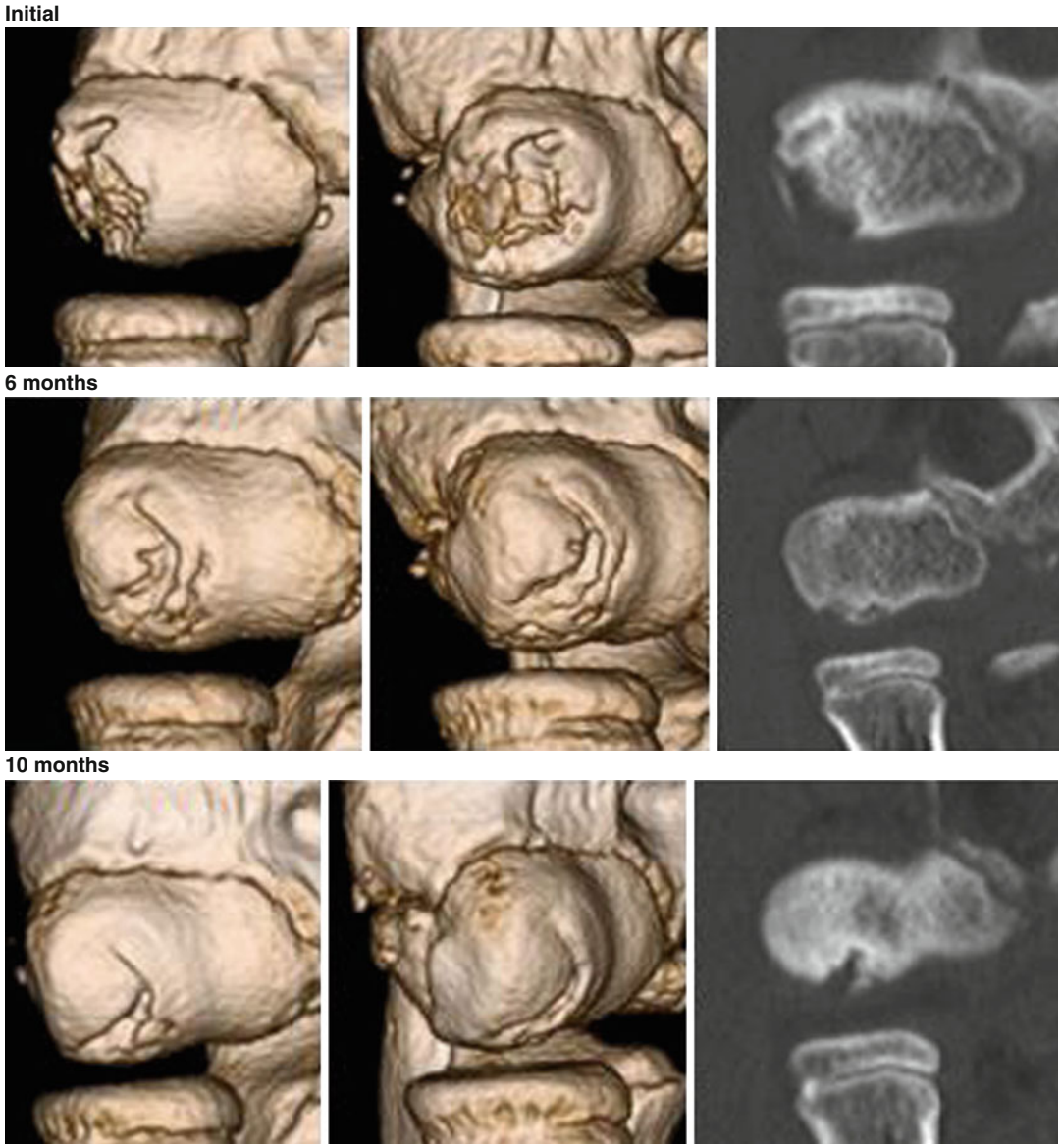

Fig. 27.3 Three-dimensional computed tomography and coronal view. The lesion originates from the lateral aspect of the capitellum and spreads to the center (initial → 6 months). The lesion is repaired from the outside (6 → 10 months)

27.2.4 Statistics of Surgical Patients with OCD

Between 1990 and 2012 (23 years), 741 patients with OCD underwent surgery in our hospital (mean age, 14.5 years). Approximately 90 % of these patients had baseball-related disorders. The others suffered from OCD induced by tennis, handball, basketball, or gymnastics. The age distribution (Fig. 27.6) shows that the majority of patients were 13–15 years of age.

Approximately 600 patients with baseball-related disorders visit our hospital annually. Approximately 80–100 patients with OCD have visited annually over the past 10 years. Of these patients, approximately half required surgery (Fig. 27.7).

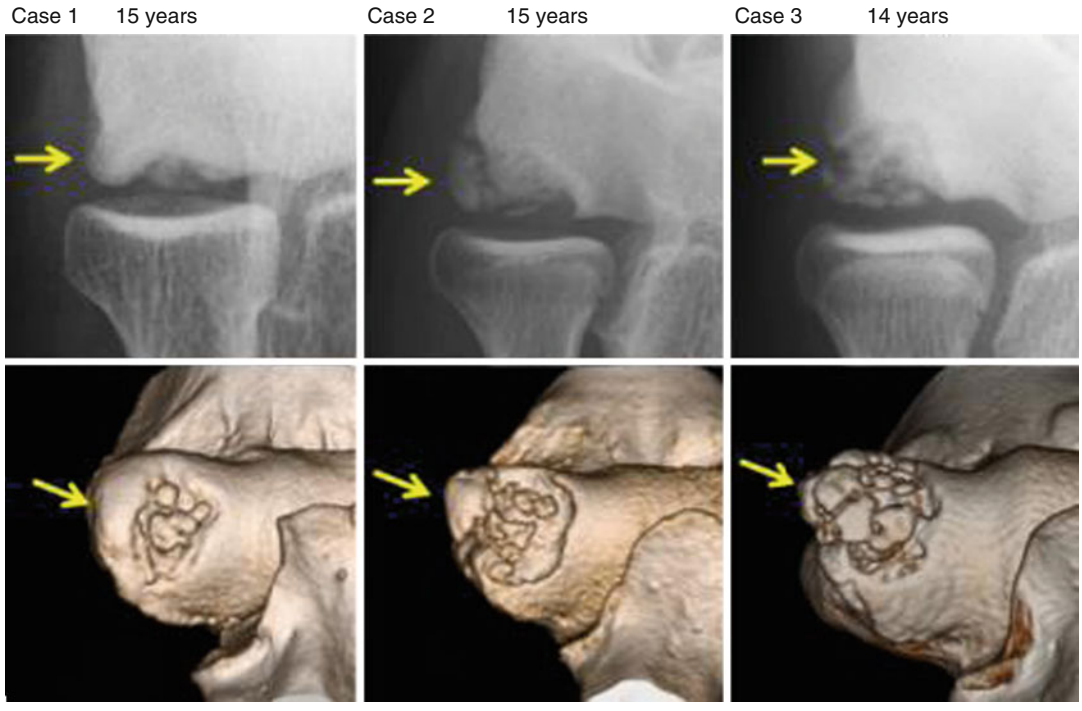

Fig. 27.4 Differences in the lesions depend on when recovery arrests. *Case 1*. Lateral walls are repaired, but a lesion remains in the center. *Case 2*. Lateral wall recovery was arrested and some fragments remain. *Case 3*. Lateral walls are not restored and extensive lesions remain

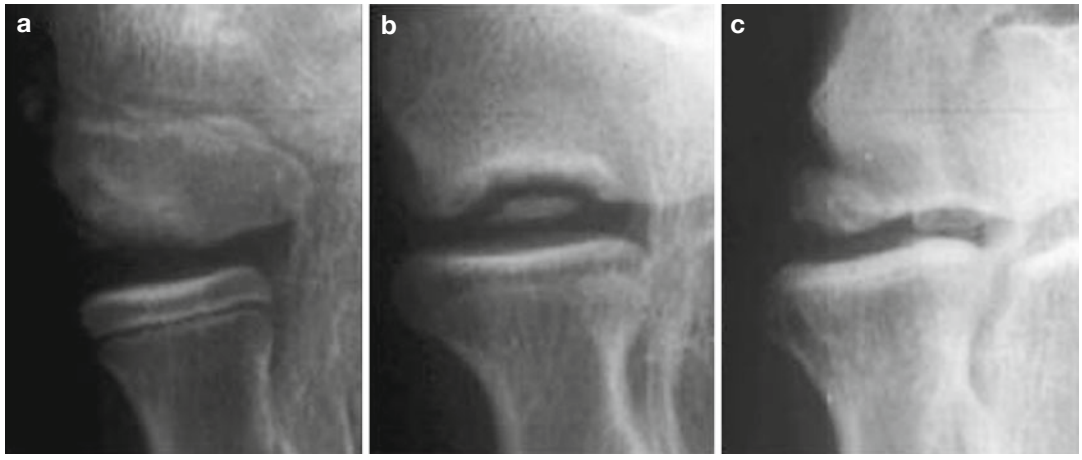

Fig. 27.5 Radiographic classification of osteochondrosis of the humeral capitellum. Stage I was characterized by translucency (**a**). In stage II, nondisplaced fragments were present (**b**). Loose bodies and sclerotic change indicated stage III (**c**) (Matsuura et al. [4])

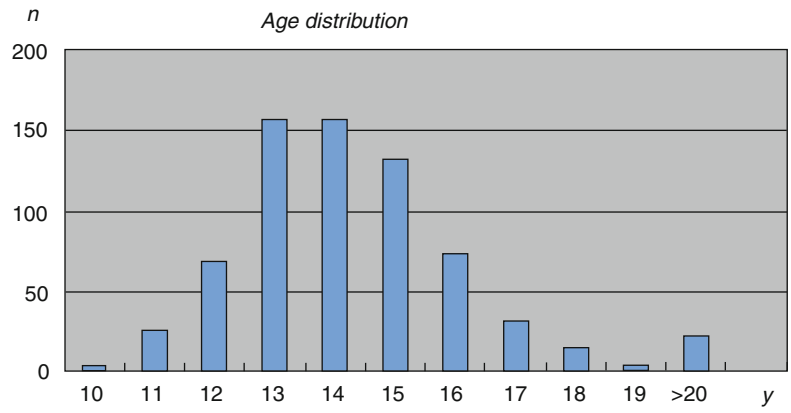

Fig. 27.6 Mean age at surgery of the 741 patients with osteochondritis dissecans

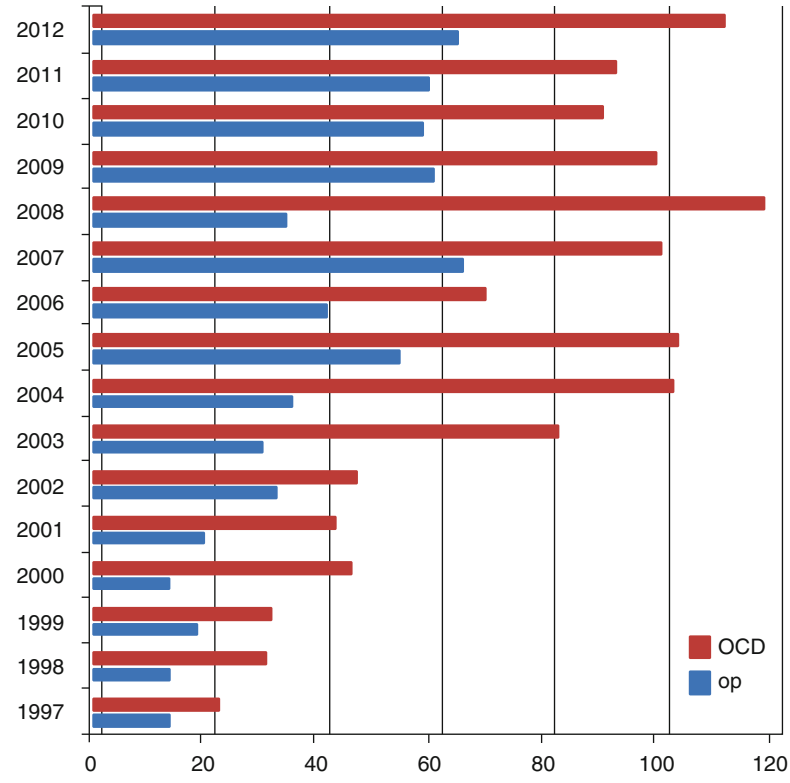

Fig. 27.7 The annual number of outpatients with osteochondritis dissecans (*OCD*) (*in red*) and the annual number of surgeries performed for OCD (*in blue*)

27.3 Clinical Presentation and Essential Physical Examination

Initial symptoms of OCD include discomfort, swelling, and limited extension of the elbow joint after baseball training. However, the pain is commonly mild and can be improved with rest. Consequently, players can continue playing baseball unless such conditions progress, and they are likely to delay visiting medical institutions. In physical examination, patients complain of swelling and reduced range of motion as well as localized tenderness when the capitellum is compressed on the anteroinferior side with the elbow joint in flexion. Bulging may be found in the soft spot

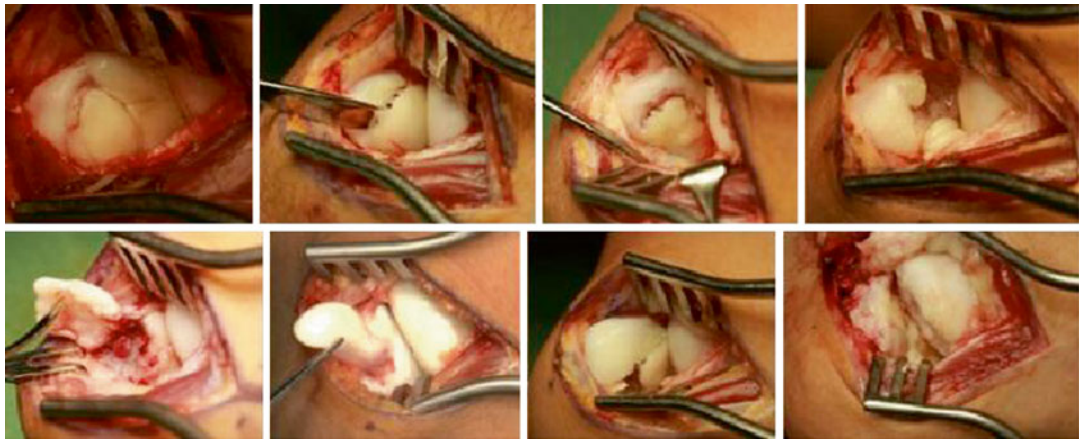

Fig. 27.8 Various findings on operation. Plain radiographs showed that the lesion is in the separation type, but the articular cartilage shows various statuses

outside of the elbow because of synovial fluid or swelling of the synovial plica. Compared with the unaffected side, flexion or extension of the elbow joint shows apparent limited range of motion, but forearm pronation and supination are usually not limited. For patients in whom flexion or extension and pronation and supination of the forearm cause catching, clicking, locking symptoms, or pain, detachment of the cartilage surface or the presence of a free body is suspected. Conservative treatment before epiphyseal closing leads to a better therapeutic effect. Recovery tends to stop when the epiphyseal line closes.

Patients with bone sclerosis around the lesion and tenderness of the capitellum often tend to have instability of the articular cartilage. In that case, compared with imaging findings, surgery findings include extensive destruction of the articular surface beyond the surgeon's expectation and show differences in cartilage degeneration (Fig. 27.8). Thus, it is difficult to preoperatively determine a specific surgical method.

27.4 Essential Radiology

27.4.1 Plain Radiographs

For patients with a throwing elbow disorder, plain radiographs must be obtained from four directions. Tangential and oblique views at 45° in particular are required to detect OCD since it is likely to be overlooked when only plain anterior-posterior (AP view) and lateral radiographic images are used (Fig. 27.9).

The above images were obtained from the same patient. The upper images were obtained from the AP view and lateral directions, in which the OCD is unclear. The lower tangential and oblique views visualize the OCD outside the capitellum. In the initial diagnosis of OCD, taking a sufficient sports history into consideration is important and diagnostic examinations are essential for identifying OCD. Plain tangential and oblique radiographs are available for follow-up of bone regeneration of the lesion treated by conservative treatment or surgical therapy.

27.4.2 Ultrasonography (US) Aids in the Diagnosis of OCD

Its advantages include early detection is possible; the subchondral bone and the articular cartilage can be observed simultaneously; more detailed tomographic images than plain radiographs can be obtained; not only osteogenic but also cartilaginous free body can be detected; and observation is possible during flexion and extension of the elbow joint (Fig. 27.10). It can also be useful as a screening examination outside of medical institutions (fieldwork) when portable US devices are used.

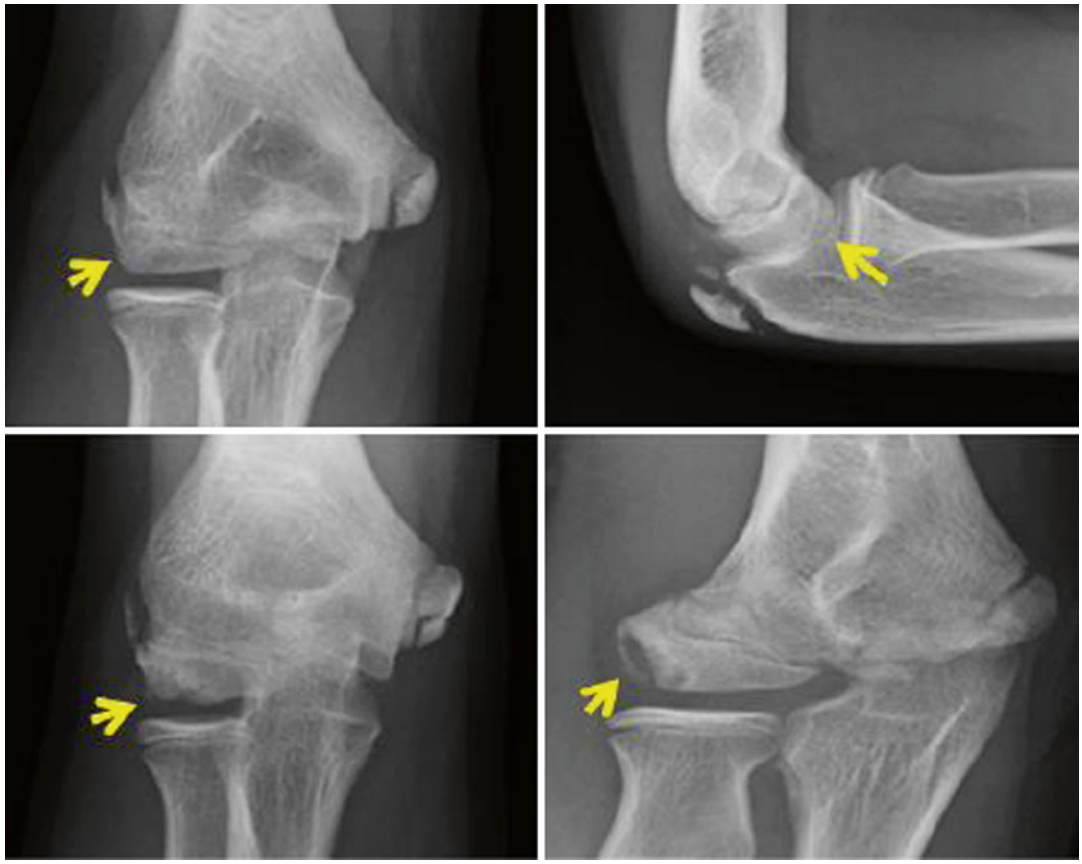

Fig. 27.9 *Upper*: the elbow in flexion from the AP view and lateral directions. *Lower*: the elbow in 45° of flexion (tangential view), the elbow in oblique view. *Arrow*: lesion

27.4.3 Computed Tomography (CT)

CT can visualize even an unclear subtle lesion on plain radiographs. It is useful for confirming bone fragment properties, free body location, exact affected width and depth, subchondral bone status, and bone sclerosis absence or presence. CT can also be used to determine the healing effects of conservative and surgical treatment. In addition, three-dimensional (3D) CT is available to observe lesion dimensions, which is helpful for planning surgery. However, it should be noted that it is difficult to understand the state of the cartilage and detect cartilage-rich free bodies.

27.4.4 Magnetic Resonance Imaging (MRI)

MRI can be used to detect initial OCD that is difficult to detect on plain radiographs. MRI is also available to evaluate cartilage degeneration and fissure as well as lesion instability. T1-weighted imaging reveals a low-intensity area of the capitellum from the early stage of OCD. T2-weighted fat suppression imaging in the sagittal section shows changes in articular cartilage (Fig. 27.11). Surgical treatment is considered when lesion instability is evaluated by synovial fluid penetration into the subchondral bone or articular cartilage discontinuity. However, screening

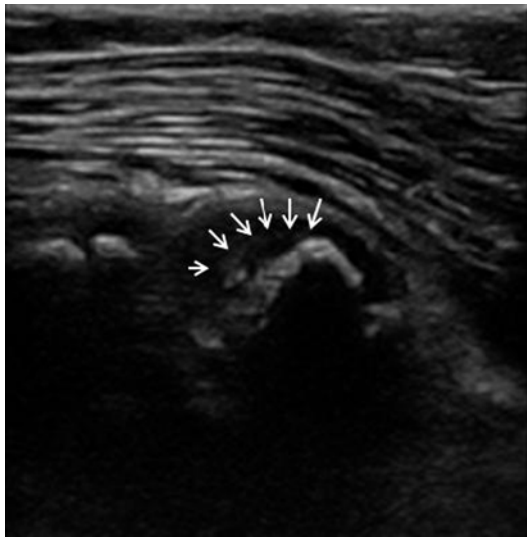

Fig. 27.10 Ultrasonography of osteochondritis dissecans of the humeral capitellum (sagittal view). *Arrow*: defect of the subchondral bone

is not usually performed because of the time and cost involved. Nelson et al. [6] classification (Table 27.1) can be used to classify OCD using MRI.

Diagnostic imaging techniques have undergone tremendous technological advances in this decade. Diagnostic techniques such as 3D CT and US are progressing remarkably, while MRI precision improves yearly. MRI will be available for further detailed evaluation. However, to decide whether surgical treatment should be provided or conservative treatment should be continued, it is necessary to evaluate local findings and various imaging studies.

27.5 Arthroscopic Pathology

27.5.1 Advantages of Arthroscopy

The International Cartilage Repair Society (ICRS) [2] classifies OCD lesions into four classes based on arthroscopy findings of knee joint lesions (Table 27.2). Baumgarten et al. [1] proposed a classification for OCD of the humeral capitellum using arthroscopy (Table 27.3).

These classifications show the degree of lesion instability by probing under arthroscopy. Arthroscopy can be used to understand the status of the affected cartilage and diagnose lesions to choose the appropriate surgical method. It is available for evaluating lesions and treating complicated lesions such as free body removal (Fig. 27.12) as well as resection of osteophytes on the olecranon or the synovial plica.

27.5.2 Postures for Arthroscopy

The authors perform arthroscopy of the elbow with the elbow in anterior flexion and the patient in a supine position (Fig. 27.13). The supine position is easy to set up and enables the surgeon to switch to open surgery as needed (such as with osteochondral mosaicplasty). We place a sandbag under the patient's shoulder to stabilize the arms and position the forearm on an armrest to hold the upper extremities.

We then inject 10–15 mL of 1 % lidocaine with epinephrine into the joint. After confirming the optimal portal site using a 23 G needle, we perform arthroscopic surgery from the posterior, posterolateral, and soft-spot portals. Implantation from the anterolateral and anteromedial portals is performed with special attention paid to the cutaneous nerves of the forearm. The scalpel is inserted to run parallel to the skin's surface. Minor forceps are then bluntly inserted subcutaneously from the muscular fasciae into the joint capsule to extend the portal. We then observe the lesion using 30° or 70° arthroscopy with a diameter of 2.7–4.0 mm.

To perform bone peg grafting or osteochondral mosaicplasty, we immediately switch to open surgery. Open surgery is superior for extensive lesions requiring articular surface reconstruction and can be used to reconstruct the articular surface from ideal directions. Since long-time arthroscopy may result in periarticular edema and the working space is small, manipula-

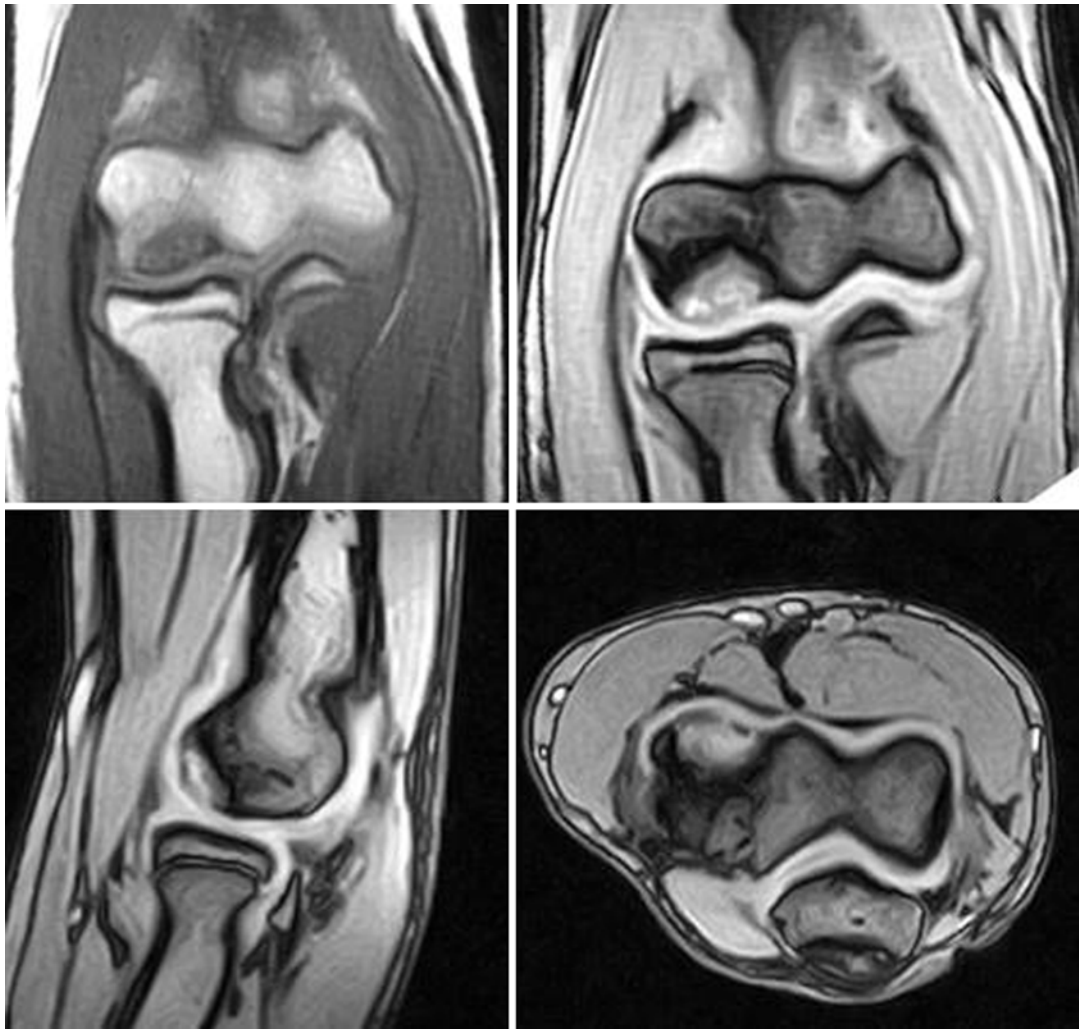

Fig. 27.11 Magnetic resonance imaging. T1-weighted image in the upper left shows low-intensity lesions. T2-weighted fat suppression images in the *upper right* and *lower right* and *left* show high-signal articular cartilage and low-signal subchondral bone

Table 27.1 The MRI grading system for osteochondritis dissecans lesions

Grade	Description
0	Normal
1	Intact cartilage with signal changes
2	A high-signal breach of the cartilage
3	A thin, high-signal rim extending behind the osteochondral fragment, indicating synovial fluid around the fragment
4	Mixed- or low-signal loose body, either in the center of the lesion or free within the joint

Nelson et al. [6]

Table 27.2 ICRS classification system for OCD lesions

Stage I: Stable lesion with a continuous but softened area covered by intact cartilage
Stage II: Lesion with partial discontinuity that is stable when probed
Stage III: Lesion with a complete discontinuity that has not yet dislocated (“dead in situ”)
Stage IV: Empty defect, or defect with a dislocated fragment or a loose fragment within the lesion bed

From Brittberg and Winalski [2]

Table 27.3 Classification system for osteochondritis dissecans

Grade	Description
1	Intact but soft ballottable cartilage
2	Fissuring of the overlying cartilage
3	Exposed bone or attached osteoarticular fragment
4	Loose, but nondisplaced, osteoarticular fragment
5	Displaced fragment with resultant loose body

Baumgarten et al. [1]
Lesions from the American Sports Medicine Institute

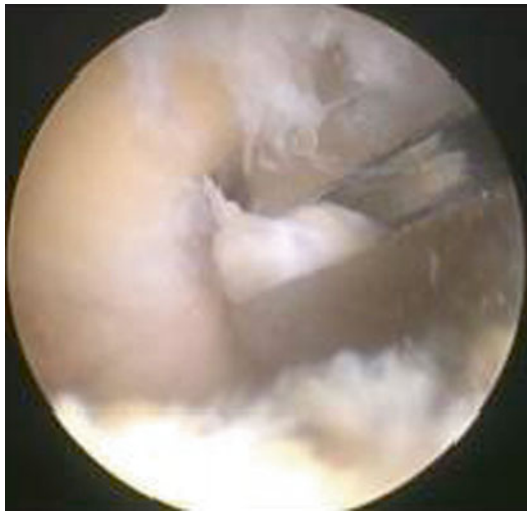

Fig. 27.12 Free body resected under arthroscopy

tion under arthroscopy is not required. Except in simple surgeries such as drilling, arthroscopy is not suitable when reconstructive surgery of the articular surface is required.

27.5.3 Pitfalls of Arthroscopy

The articular cartilage observed through arthroscopy, even if it is an apparently extensive lesion on CT or MRI, may be graded as grade 1 according to the ICRS scale. Caution should be taken because arthropathic changes may progress if such a lesion is treated with drilling only. Attention should be paid to adapt arthroscopic drilling, as it should not be performed in extensive lesions since it can promote destruction of the articular surface.

Although the lesion in Fig. 27.14 has no subchondral marrow, it was treated by arthroscopic drilling because the cartilage surface was clean. In this case, the patient visited our hospital several months after surgery. Plain radiographs showed that the joint space of the humeroradial joint had disappeared and the radial head was enlarged. On 3D CT, the humeroradial joint, transformation of the trochlear, and a superiorly extended radial head were found. Remarkably limited range of motion was also seen at elbow extension of 45° and flexion of 90°. Arthrography showed that the articular cartilage of the capitellum and the radial head were destroyed (Fig. 27.15).

The arthroscopy in Fig. 27.16 shows that alignment of the joint surface was maintained and the cartilage surface was very clean without softening (ICRS I). However, it was revealed unstable when the bone fragment was pushed up from under the lateral walls with probe. In such a case, it is often difficult to confirm instability using arthroscopy. It is hazardous to determine a surgical method by evaluating only the articular cartilage condition because the OCD lesion is caused by necrosis of the bone marrow beneath the articular cartilage.

27.6 Treatment Options

27.6.1 Nonoperative Treatment

For OCD in school-age children, conservative treatment is given priority. Especially for baseball players, throwing and batting are forbidden and fielding practice is prohibited. Healing tends to occur late when motions other than throwing are permitted. For patients with extensive lesions ranging from outside the capitellum to its center, bicycle riding is also prohibited. If patients show clear swelling and pain on motion, it is desirable that they wear a splinting with the elbow in 45° of flexion whenever leaving home (for 4–8 weeks) (Fig. 27.17).

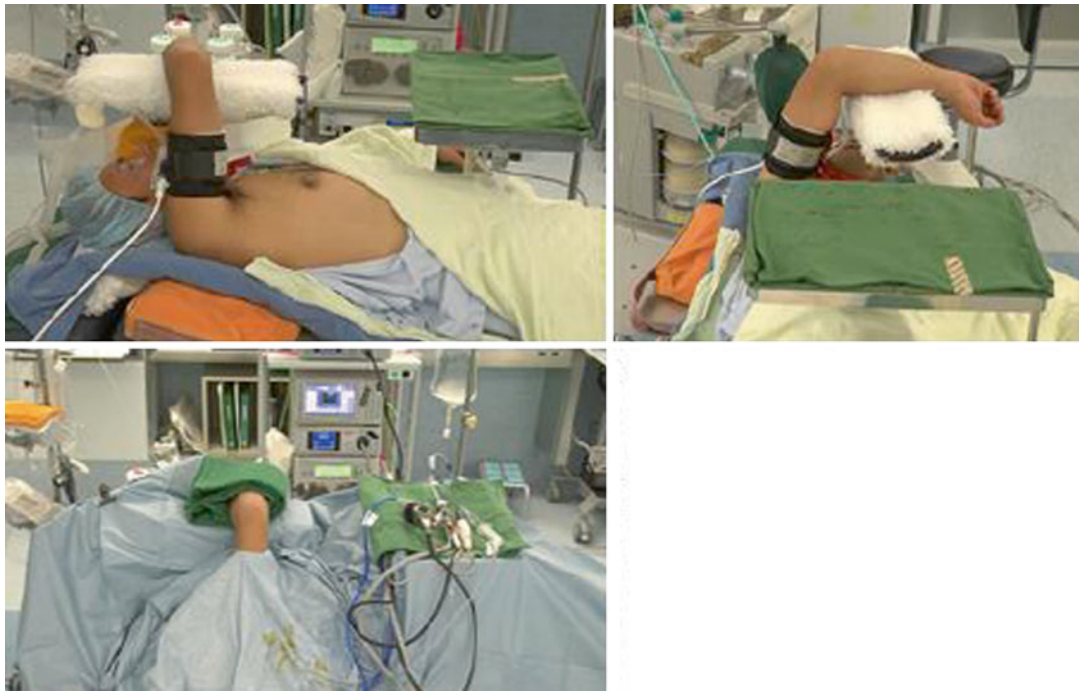

Fig. 27.13 Arthroscopy posture. A sandbag is placed under the patient's shoulder joint in the supine position and the elbow is positioned in flexion on the armrest

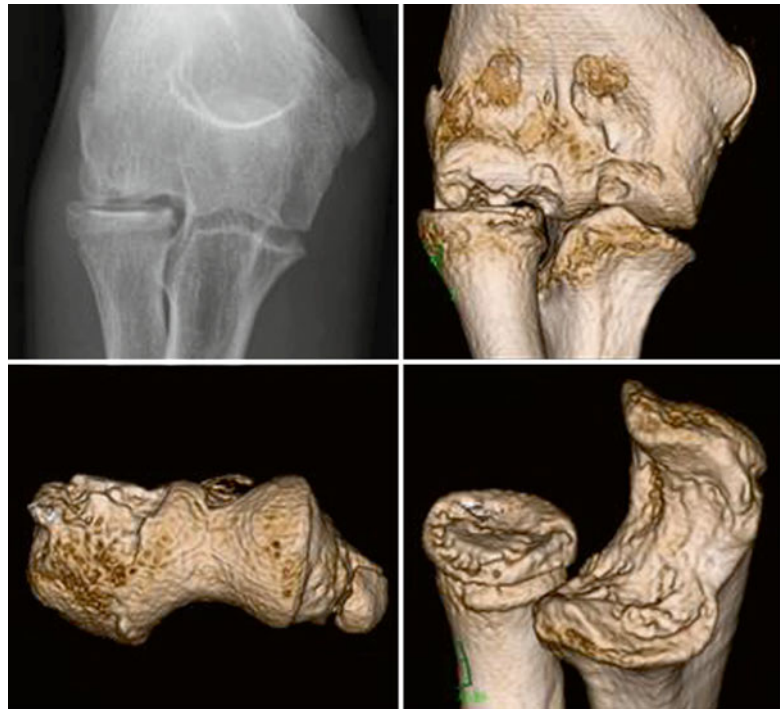

Fig. 27.14 Deformation progressed in the extensive osteochondritis dissecans lesion treated with drilling

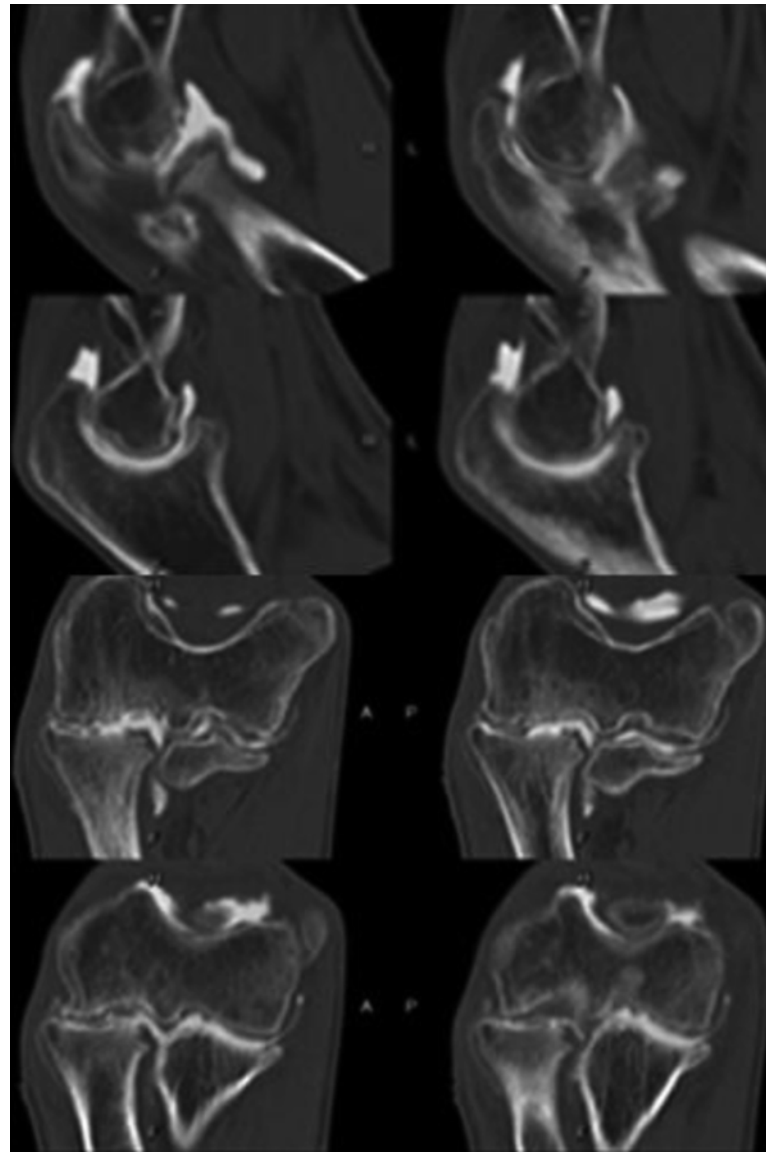

Fig. 27.15 Arthrography+ computed tomography image. The articular cartilage of the humeral capitellum and the radial head have almost disappeared

Conservative treatment is initially provided for at least 4–6 months. If healing process is seen upon various imaging examinations, the same conservative treatment is continued. Lesions in which continuity of the subchondral bone is confirmed on MRI and/or CT are likely to respond to conservative treatment (Fig. 27.18).

After the healing process declines after considerable improvement, and their lesions are classified into the central type, and the lesion area is stable and <30 % of the area of the capitellum, the authors allow the patients to return to sports with awareness that if they have no pain. A free body may subsequently form, in which case its removal should be performed earlier.

27.6.2 Low-Intensity Pulsed Ultrasound (LIPUS) Treatment

LIPUS treatment is currently used as an adjuvant fracture treatment. However, there is no evidence whether LIPUS treatment for OCD promotes recovery and whether a case of delayed union is

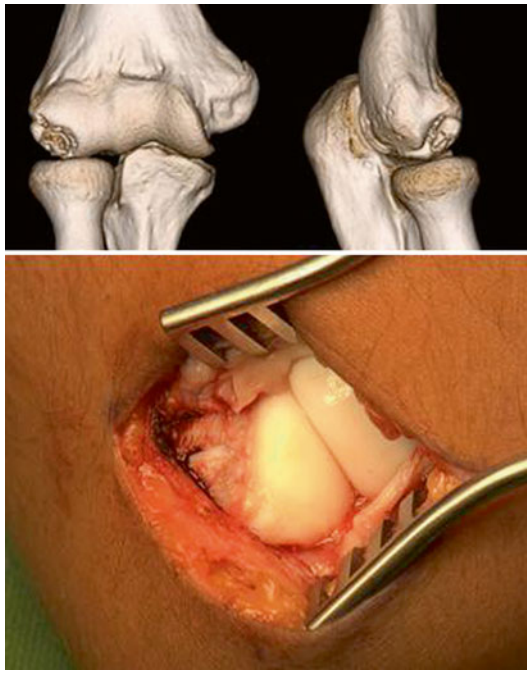

Fig. 27.16 The surface of the articular cartilage of the extensive type of osteochondritis dissecans. Note the small fragments in the lateral walls and the deficiency of the subchondral bone in the center in MRI. In the intraoperative photograph, the articular cartilage is clean and its instability cannot be confirmed from the surface view. However, the surface was unstable when the lateral wall was pushed up with the probe

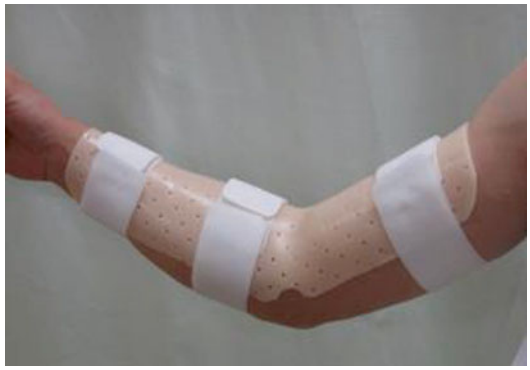

Fig. 27.17 Wearing of the splint at 45° of flexion of the elbow. This brace is taken off while bathing, eating, and sleeping

also repairable. Accordingly, if the number of the cases to be switched to surgery decreases or if it promotes recovery, the effectiveness of LIPUS for OCD will be expected.

The authors compared the patients treated with LIPUS (LIPUS) or without LIPUS (non-LIPUS) in similar stages of OCD. As shown in Fig. 27.19, clean repair in the LIPUS group took 2 months and 3 days, while that in the non-LIPUS group took 5 months and 21 days. This finding suggests that the repair period can be shortened.

In another study, the authors performed LIPUS in 51 patients with OCD regardless of disease stage and found that LIPUS was effective and ineffective in 22 and 29 patients, respectively. The repair period was approximately 6.4 months in the effective group, including 14 patients in the advanced disease stages, which showed earlier recovery than the usual repair period (10–12 months). Recovery seems to be rapid when the surface of the articular cartilage has not fissured. The recovery state on CT was also examined from the viewpoint of the presence or absence of perifocal osteosclerosis. As a result, in the group without osteosclerosis, 19 of 35 patients (54.3 %) showed an almost fully repaired state. In the group with osteosclerosis, 3 of 16 patients (18.8 %) showed a repaired state (Table 27.4). The difference between groups was significant.

The effect of LIPUS will be expected if patients without perifocal osteosclerosis are chosen. On the contrary, it will be less likely to be treated with conservative treatment if osteosclerosis is found around translucency. The images of osteosclerosis indicate that the repair process stopped, which means that further recovery cannot be expected. In addition, in the patients treated with LIPUS showing poor results, intraoperative macroscopic findings showed a larger cartilaginous fissure around the lesion and abnormal mobility. Therefore, for patients with instability of the lesion, its effect cannot be expected.

Regarding the adverse effect of LIPUS on epiphyseal growth cartilage, the report seemed to show no influence on bone length. In the patients treated with LIPUS here, none showed premature epiphyseal closure.

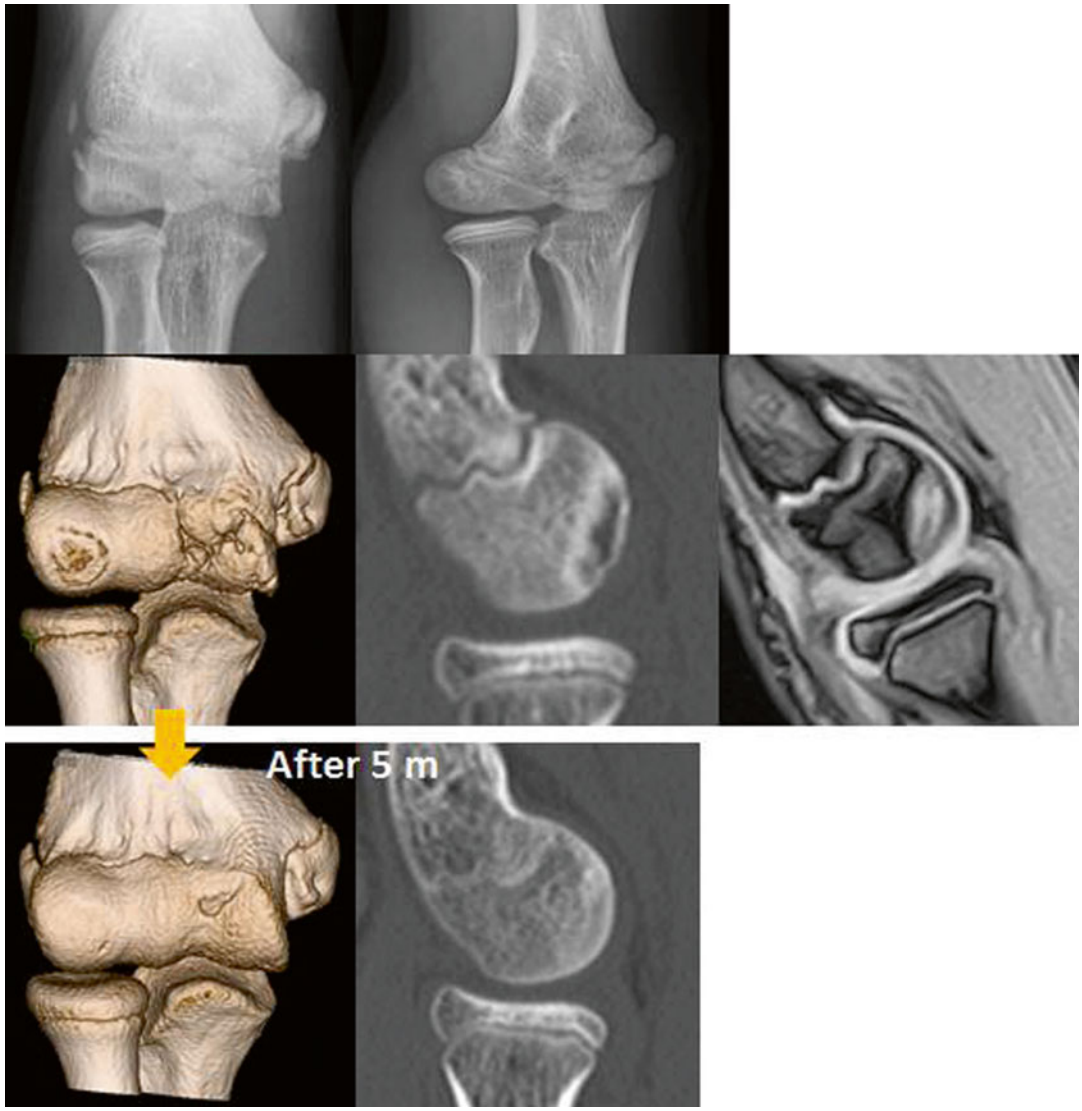

Fig. 27.18 Plain radiographs in the translucency period. Computed tomography and magnetic resonance imaging show the continuity of the subchondral bone and cartilage. The lesion was completely repaired 5 months after treatment initiation

27.6.3 Operative Treatment

For surgical treatment of advanced OCD, various techniques such as free body removal, isolated site curettage, drilling without resection, osteochondral mosaicplasty technique, and free body refixation remain controversial. The purpose of treatment for juvenile baseball players with OCD is to repair the osteochondritis anatomically, prevent osteoarthritis, and allow patients to return to sports activities at the same level as prior to injury. Therefore, it is important to sufficiently repair the articular surface.

It is impossible to strictly judge whether surgery is indicated by using plain radiographs only. To choose the appropriate therapy, age, disease

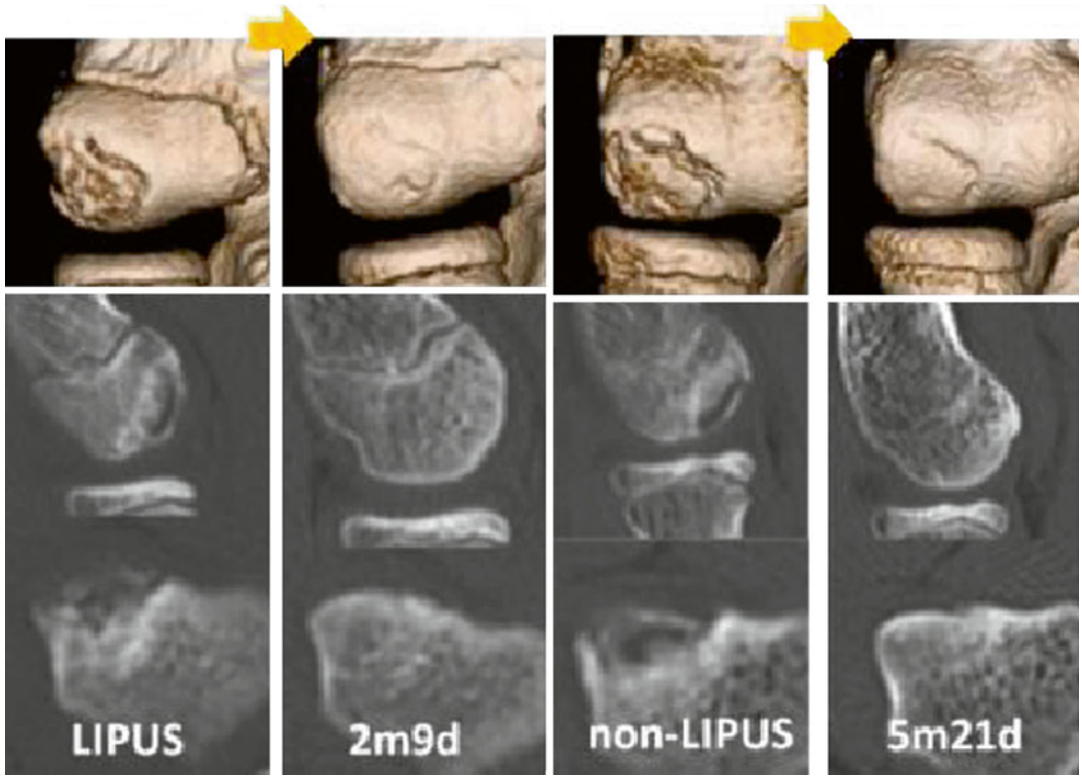

Fig. 27.19 Comparison of the images of osteochondritis dissecans in the translucency period. *Left*: low-intensity pulsed ultrasound was performed. The entire lesion was repaired at 2 months and 9 days after treatment initiation. *Right*: the lesion was treated with rest only as conservative treatment

Table 27.4 Conservative treatment (LIPUS) of the osteochondritis dissecans

Sclerotic change	+	–
Failed	13 (81.2 %)	16 (45.7 %)
Effective	3 (18.8 %)	19 (54.3 %)
Total	16	35

$p < 0.05$, risk ratio: 3.3

stage, and lesion size must be considered. However, the precision of diagnostic imaging including CT and MRI has recently been improved. Accordingly, it is possible to plan a reconstruction method to some extent. Age, especially bone age, has a major influence on prognosis, and the healing tendency differs before and after epiphyseal closure. For patients ≤12 years old, conservative treatment is expected. However, for patients >12 years old, if conservative treatment is continued for a year or more without an aim, recovery will not be complete and they will have to undergo surgery, which should be avoided. For patients with a long-term treatment course, it is necessary to determine the limit from the viewpoint of the degree of recovery with conservative treatment considering the psychological stress of not playing baseball.

Extensive lesions are likely to lead to cartilage fissures around the lesion, isolates of free bodies. Therefore, surgical treatment is chosen unless an improvement trend is shown after conservative treatment for approximately 4–6 months. The most desirable timing of surgery is the period at which the epiphyseal line has been closed.

The incidence of spontaneous cure decreases after epiphyseal closure. For patients with a clearly limited range of motion or strong tenderness of the articular surface of the capitellum, surgical treatment should be chosen immediately due to unstable lesion isolation.

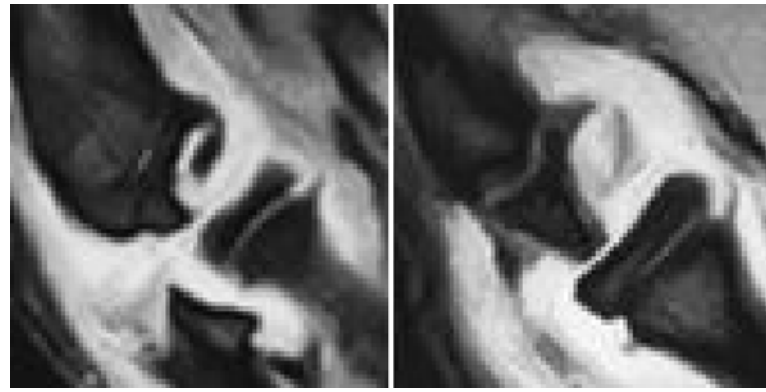

Fig. 27.20 Magnetic resonance imaging (MRI), T2 sagittal images. A high-intensity area is found in the lesions on MRI, T2-weighted images. Alignment of the subchondral bone is poor

If a high-intensity area is found in lesions on MRI, synovial fluid has likely penetrated into the lesions from the fissure of the arthrodial cartilage; in such a case, recovery is rarely achieved by conservative treatment only. When the cartilage of the involved site and the outline of the subchondral bone do not match the outline of the healthy capitellum, instability is likely (Fig. 27.20).

For postoperative evaluation, there are useful evaluation methods such as the scoring system developed by Timmerman and Andrews et al. The Japanese Orthopaedic Association (JOA) sports score (100-point scale) is commonly used in Japan.

27.6.4 Choice of Surgical Method

Various surgical methods include drilling (microfracture), lesion curettage, bone peg fixation, mosaicplasty, costal osteochondral grafts, closed wedge osteotomy of the lateral humeral condyle, and autologous chondrocyte transplantation. It is difficult to determine the ideal surgical procedure before surgery. As mentioned above, imaging and intraoperative findings do not necessarily match, and the same surgical procedure may not always be chosen in patients in the same stage (Fig. 27.8). The appropriate surgical procedure is determined during surgery after consideration of the subchondral bone status, lesion instability, and cartilage degeneration. Thus, preparations of surgical instrument or devices are necessary to support multiple surgeries. Each surgical method is described below.

The details of bone peg grafts, osteochondral mosaicplasty, and costal osteochondral grafts are described in the Sect. 27.7.

27.6.4.1 Drilling (or Microfracture)

Drilling is sometimes indicated for patients who are classified into the translucency period, central localized type, and grade 1 according to the ICRS scale for articular cartilage classification. The range of indication for drilling is small. This leads to a problem if drilling is used because a patient wants an early return; in such a stage, many patients can be treated with conservative treatment. Some patients in such a stage underwent surgery too early that led to aggravation. Therefore, early surgery requires special attention. The authors rarely performed drilling.

27.6.4.2 Curettage of Lesion

Lesion excision is indicated for microlesions of the center type. However, it is contraindicated in patients with extensive and deep outside-type lesions. This procedure should be avoided if only for the purpose of returning to sports earlier; rather, it should be performed after the epiphyseal growth line of the lateral humeral condyle is closed unless there is a compelling reason.

27.6.4.3 Wedge Osteotomy of the Lateral Humeral Condyle

In 1983, Yoshizu reported the use of closed wedge osteotomy for the purpose to decrease the pressure applied to the humeral capitellum and to improve revascularization of the damaged region.

Closed wedge osteotomy in the sagittal plane may improve blood circulation of the capitellum. The degree of surgical invasion is relatively higher, but its results are stable in patients in the early stages of disease. For instability of the lesion or the articular cartilage, bone peg and/or osteochondral mosaicplasty can be combined in the involved site. The authors have no experience with this surgery.

27.6.4.4 Autologous Chondrocyte Transplantation

The cultured autologous cartilage cells on a collagen gel can be transplanted to the cartilaginous defect. The graft is covered with autologous periosteum. However, there are problems with this method as it is not suitable if the defect of the subchondral bone is deep since the transplanted tissue does not have sufficient thickness and strength. However, it is expected to bring composite graft tissues of the cancellous bone and the articular cartilage.

27.6.4.5 Others

Resection is required for patients who are having free bodies. If the original lesion of the capitellum is considerably repaired, they can be treated with resection of the free body alone. Other methods include bone peg grafting with the anconeus muscle pedicle, free autologous periosteal implantation, and lifting and fixing of the isolated fragment with soft wire.

27.7 Authors' Preferred Treatment

27.7.1 Operative Treatment

We have mainly performed extirpation of loose bodies and/or bone peg grafts since 1990. Instead of using a uniform operative method, we have mixed various methods according to pathology, introducing mosaicplasty in 2000 and costal osteochondral graft in 2005 (Fig. 27.21).

27.7.2 Bone Peg Graft

Indications

This method is used when plain radiography indicates a translucent-type or split-type lesion with firmly remaining subchondral bone, and the

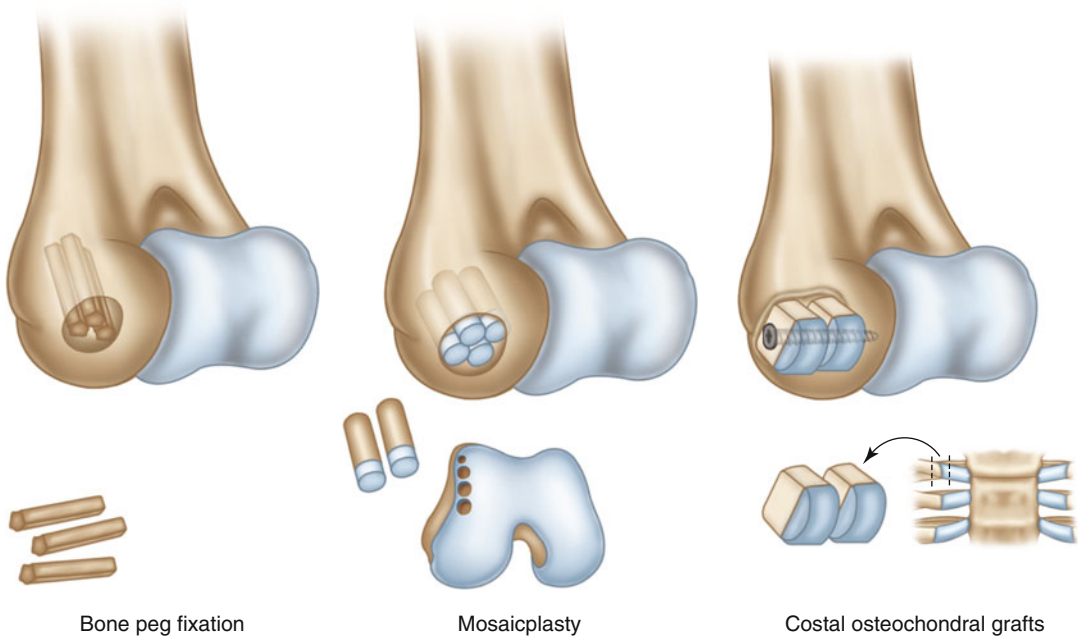

Fig. 27.21 Schema of each method. *Left*: bone peg graft. *Center*: mosaicplasty. *Right*: costal osteochondral grafts

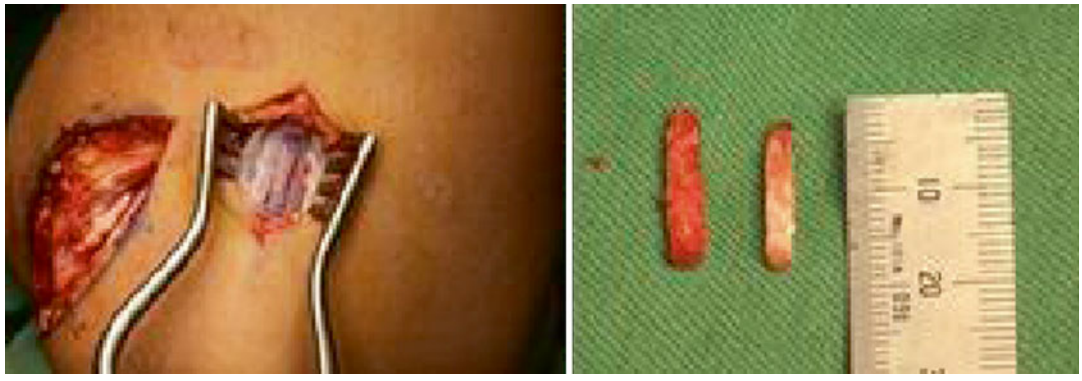

Fig. 27.22 Collection of bone pegs. Bone pegs (approximately 20 mm long with a diameter of 2.5–4 mm) are collected from the olecranon. Collection with a bone saw should be avoided to reduce necrosis of the osseous tissue. Small holes arranged like a *dotted line* for cutoff are prepared using a Kirschner wire, followed by collection of the bone pegs with a bone chisel

lesion is localized with no or mild degeneration of the cartilaginous surface. This method is generally not used when the articular cartilage is strongly degenerated.

Method

Bone pegs (approximately 20 mm long with a diameter of approximately 2.5–4 mm) collected from the olecranon are driven from the articular surface (Fig. 27.22).

In the case of a wide or unstable lesion, care needs to be taken to avoid any increase in instability of the lesion or fissure on the cartilaginous surface when preparing the drill holes. For this reason, a Kirschner wire with a diameter of 1.2 mm is temporarily fixed from a site that does not introduce any interference. This is the preferred method used to widen the fixation hole from a small diameter to the desired diameter (3.2–3.5 mm). A bone peg is driven into the bone, followed by removal of the wire and preparation of the same bone hole. This procedure is repeated. The bone pegs should be approximately 1–2 mm below the cartilaginous surface (Fig. 27.23). In case of a wide lesion, care needs to be taken to avoid mutual interference of the bone pegs by taking the driving angles into consideration. After fixation, compatibility with the radial head should always be confirmed. The site of bone peg collection will be fully regenerated in 3–6 months.

The postoperative limb position in external fixation should be in an angle with which the facing radial head can sufficiently enfold the graft (30–60° of elbow flexion).

According to the results of our 149 cases with a postoperative follow-up of at least 2 years (Fig. 27.24), the return rate was 98 %, and residual pain was seen in 4.7 %. The range of motion (flexion/extension) showed approximately 10° improvement, whereas the JOA score (out of 100 points) improved from 62.5 points to 93.8 points on average, showing good performance (Table 27.5).

Residual pain was frequently seen in patients before the introduction of the mosaicplasty. We believe this was because we also performed the bone peg graft in patients with degenerated articular cartilage. Currently, we use mosaicplasty for those who cannot undergo a bone peg graft.

27.7.3 Osteochondral Mosaicplasty

Indications

This method should be used for cases showing remarkable degeneration on the surface of articular cartilage, cases with a detached or loose lesion, cases with an extensive lesion with a remaining lateral wall of the capitellum, and others [10]. In cases accompanied by cartilaginous degeneration and disappearance of subchondral bone, a bone

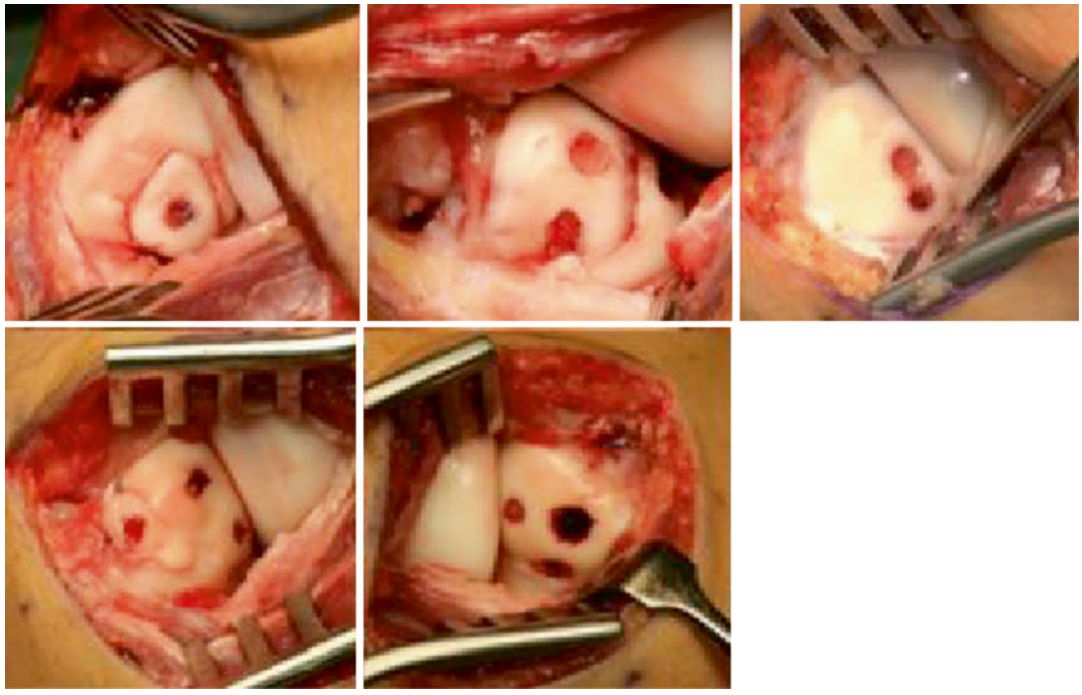

Fig. 27.23 Bone peg graft. Bone pegs are driven to 1–2 mm below the cartilaginous surface

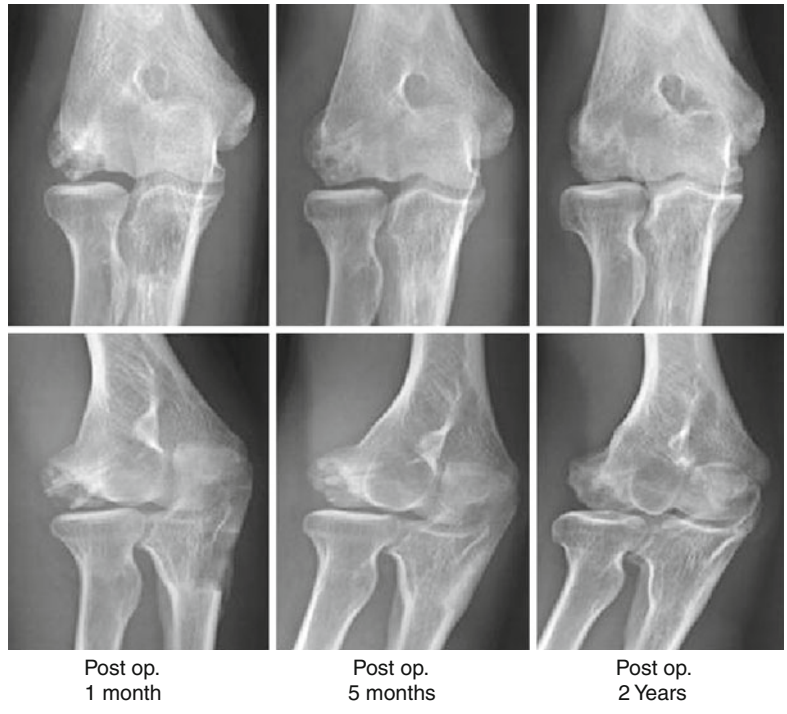

Fig. 27.24 Postoperative course of bone peg graft in plain radiographic images (tangential view and oblique view). Postoperative 1 month, 5 months, and 2 years

peg graft is insufficient. The advantage of this operative method is that the degenerated surface of the articular cartilage can be anatomically reconstructed by using hyaline cartilage connected with a subchondral bone. The disadvantage is that the knee joint is invaded because the graft is collected from a non-weight-bearing site of the lateral femoral condyle of the healthy knee joint [8]. It is accepted that there is no problem in the knee joint at postoperative 1 year. We have performed arthrocentesis for postoperative knee joint hematoma 2 days after surgery. We observe no remarkable subsequent swelling. Indeed, in our experience, there have been no patients with a complaint on the long-term basis either (Fig. 27.25).

Grafts with a diameter of 3–6 mm are frequently used. We frequently use osteochondral pillars with a diameter of 4.5 mm and a length of approximately 15–20 mm. We transplant 2–5 combined grafts conforming to the size of the lesion (Fig. 27.26).

When the cartilage in the lesion is strongly degenerated, detached, and unstable, the grafts should be transplanted after curetting the degenerated cartilage. When the instability is low because of mild cartilaginous degeneration, grafts with a diameter of 4.5 mm are driven into the drill holes with the same diameter, leaving the cartilaginous surface intact. It is important to reconstruct the spherical surface of the capitellum by taking the inclination of the grafted cartilaginous surface into consideration. For cases in which the graft surface is uneven, we then make a spherical shape conforming to the shape of the capitellum using a surgical knife. Regarding external fixation, we perform fixation that is retained for around 2 weeks in a limb position with which the radial head can compress the lesion of the capitellum and grafts, similar to a bone peg graft.

According to the results of our 276 cases with a postoperative follow-up of at least 2 years, the return rate was 97.8 %, and residual pain was seen in 6.5 % of cases. The range of motion (flexion/extension) also showed improvement of approximately 10° on average, whereas the JOA score improved from 57.1 points to 94.3 points on average, showing good performance (Table 27.6). The capitellum surface was firmly formed even

Table 27.5 Performance over 2 years after bone peg graft ($n = 149$)

Bone peg fixation ($n = 149$)				
Return	Failed	No pain	Pain	Unclear
146	3	135	7	4
98.0 %	2.1 %	90.6 %	4.7 %	2.7 %

		Pre op.	Post op.
JOA sports score		62.5	93.8
ROM	Flex.	129.2	133.1
	Ex.	–12.8	–6.3

JOA Japan Orthopaedic Association, *ROM* range of motion

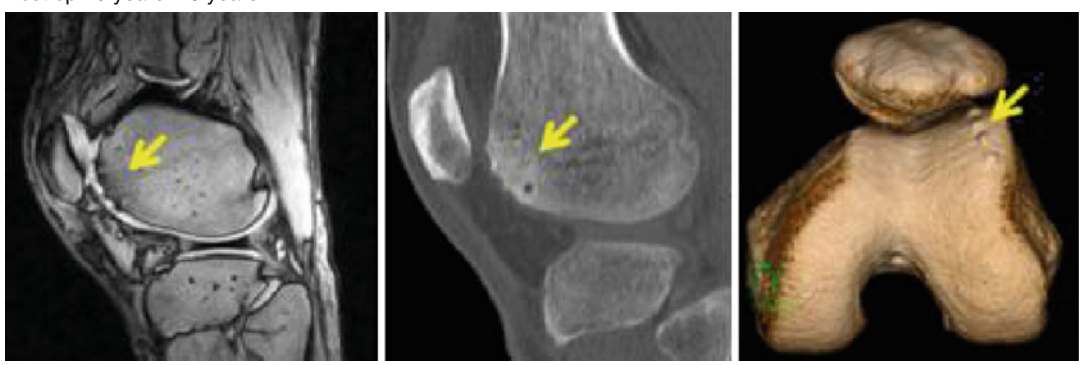

Fig. 27.25 Donor site: 10 years after harvest of osteochondral graft for lateral femoral condyle

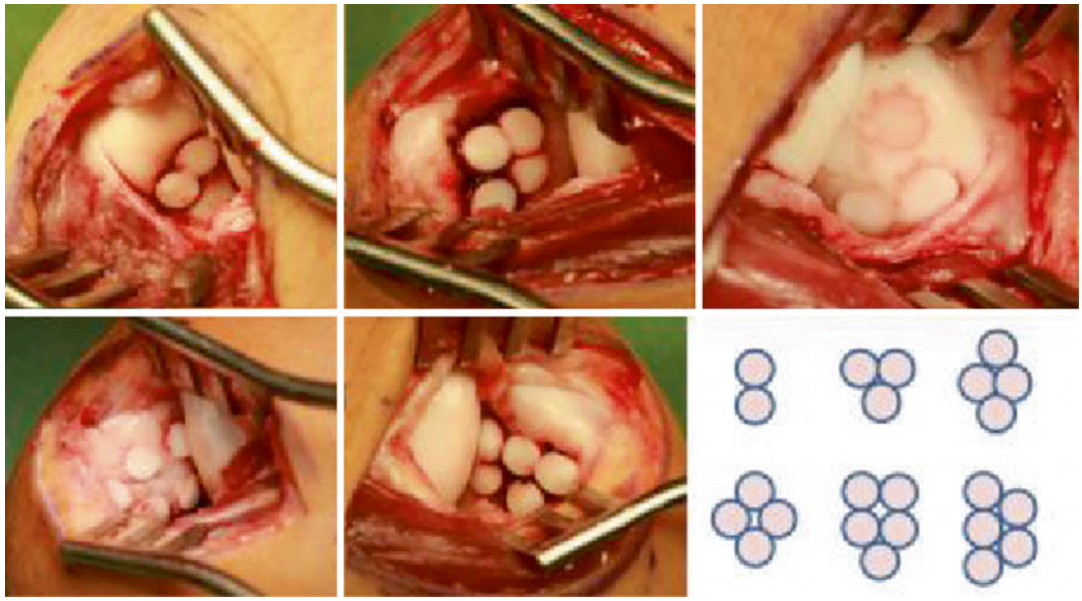

Fig. 27.26 Osteochondral mosaicplasty. Two to five grafts with a diameter of 4.5 mm and a length of 15–20 mm, conforming to the size of the lesion, are transplanted

Table 27.6 Performance over 2 years after mosaicplasty ($n=276$)

Mosaic plasty ($n=276$)				
Return	Failed	No pain	Pain	Unclear
270	6	250	18	8
97.8 %	2.2 %	90.6 %	6.5 %	2.9 %

		Pre op.	Post op.
JOA sports score		57.1	94.3
ROM	Flex.	129.8	134.1
	Ex.	–10.1	–4.7

10 years after operation (Fig. 27.27), demonstrating a good prognosis.

Investigation of osteoarthrosis (OA) changes in OCD cases that underwent mosaicplasty resulted in finding an increase in OA changes from a preoperative incidence of 13.6 % to a postoperative incidence of 65.9 %. Progression of OA was observed even in cases given the operation who then returned to playing sports. In cases with an extended lesion, a trend was seen that OA changes were likely to develop, and changes that developed once were likely to progress even after operation. We are currently implementing the hybrid technique for lateral extended lesions.

27.7.4 Hybrid Arthroplasty (Bone Peg Fixation + Mosaicplasty)

Indications

This method is used for cases with a lateral extended lesion of OCD of the humeral capitellum, ranging from the lateral to the central part, in which the capitellum in the lateral wall side is also destructed (Fig. 27.28). This is because the strength of the humeral capitellum in the lateral side is insufficient against compressive force added on the radial head at the time of throwing [11]. We use this method for cases that are difficult to treat using only a bone peg graft or mosaicplasty.

In cases with an unstable osteochondral fragment in the lateral wall, it is possible to reconstruct the lateral wall by using the osteochondral fragment. The fragment is fixed by using bone pegs or pegs and a Kirschner wire.

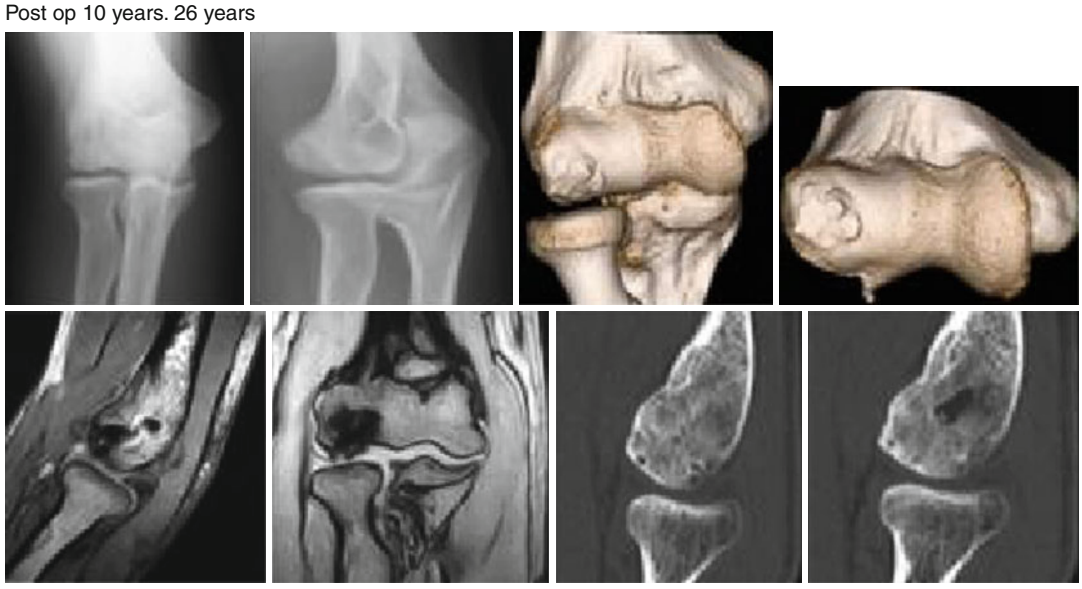

Fig. 27.27 Results 10 years after mosaicplasty. Four osteochondral pillars were grafted. Currently, the patient is a baseball pitcher but has no complaint regarding his elbow

This operative method combines bone pegs and an osteochondral graft (hybrid arthroplasty). The advantages are articular surface alignment is easy to arrange through utilization of the intact lateral osteochondral fragment without extirpation, a large osteochondral fragment can be fixed without modification, the use of bone pegs allows us to easily obtain bone union, and so on.

27.7.4.1 Operative Method

At first, the lateral osteochondral fragment is fixed with bone pegs. In this situation, the fragment should be temporarily fixed using a Kirschner wire to avoid rotation or fissure of the fragment caused by the drill. In the case of a small osteochondral fragment, the fragment is fixed by using a Kirschner wire in some cases. One or two pegs with a diameter of approximately 3 mm and a length of approximately 15–20 mm are driven into the lateral osteochondral graft. Next, the osteochondral graft is transplanted in the center. The important note in this operation is to avoid interference among the grafts (Fig. 27.29).

The pictures below are the CT images showing postoperative courses. The bone union of the lateral bone fragment is good at postoperative 1 year. At postoperative 2 years, bone union of the graft in the center also shows further progression (Fig. 27.30).

According to the results of our 50 cases with at least 2 years of postoperative follow-up, the return rate was 98.0 %, and residual pain was seen in 10 %. Range of motion (flexion/extension) also showed an average improvement of 13°, whereas the JOA score improved from 43.0 points to 92.2 points on average, showing good performance (Table 27.7). This is a very useful operative method for a lateral extended lesion of OCD.

27.7.5 Costal Osteochondral Graft

Indications

We perform this operation for cases of lateral extended lesions accompanied by lateral wall defect and joint destruction that we cannot manage with any of the abovementioned operations (Fig. 27.31).

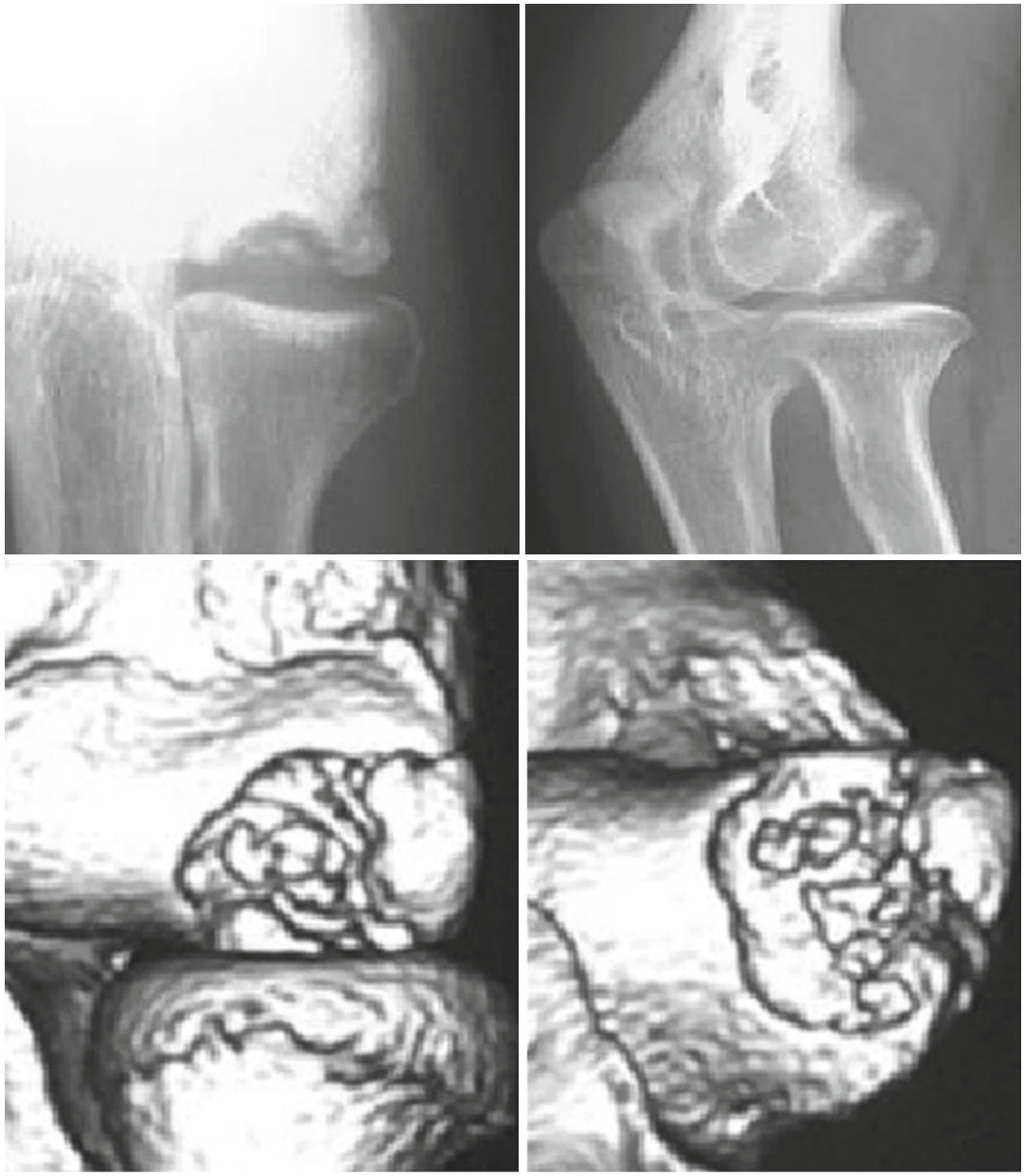

Fig. 27.28 An extended lesion ranging from the lateral to the central part with instability of the lateral wall

Due to the repeated valgus force to the elbow in throwing, the radial head strongly compresses the capitellum. For this reason, a defect of the lateral wall enhances the valgus elbow and further increases the load on the internal supporting mechanism. For that reason, rigid reconstruction with a hard osteochondral substance is required. Because grafts collected from the knee have little strength in the marrow pillar, leading to a risk of collapse or fracture due to repeated throwing, use of a rib with costal cartilage and high bone substance strength is preferred [9]. The rib with costal cartilage is collected from the right fourth or fifth rib (2 pieces are collected in some cases). The fifth rib is the easiest to use.

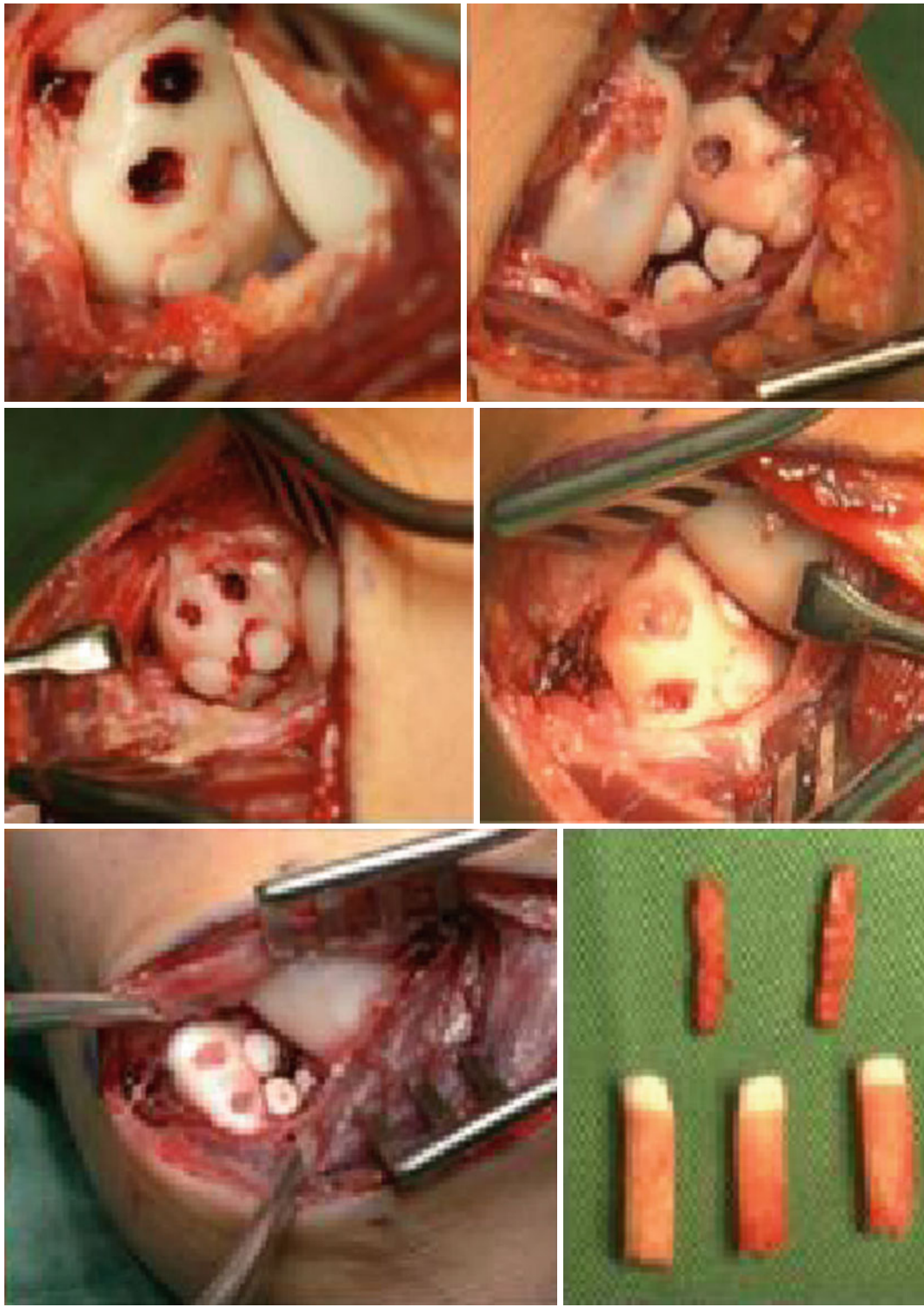

Fig. 27.29 Hybrid arthroplasty. One or two bone pegs with a diameter of approximately 3 mm and a length of approximately 15–20 mm are driven into the lateral bone fragment. The graft is transplanted in the central part

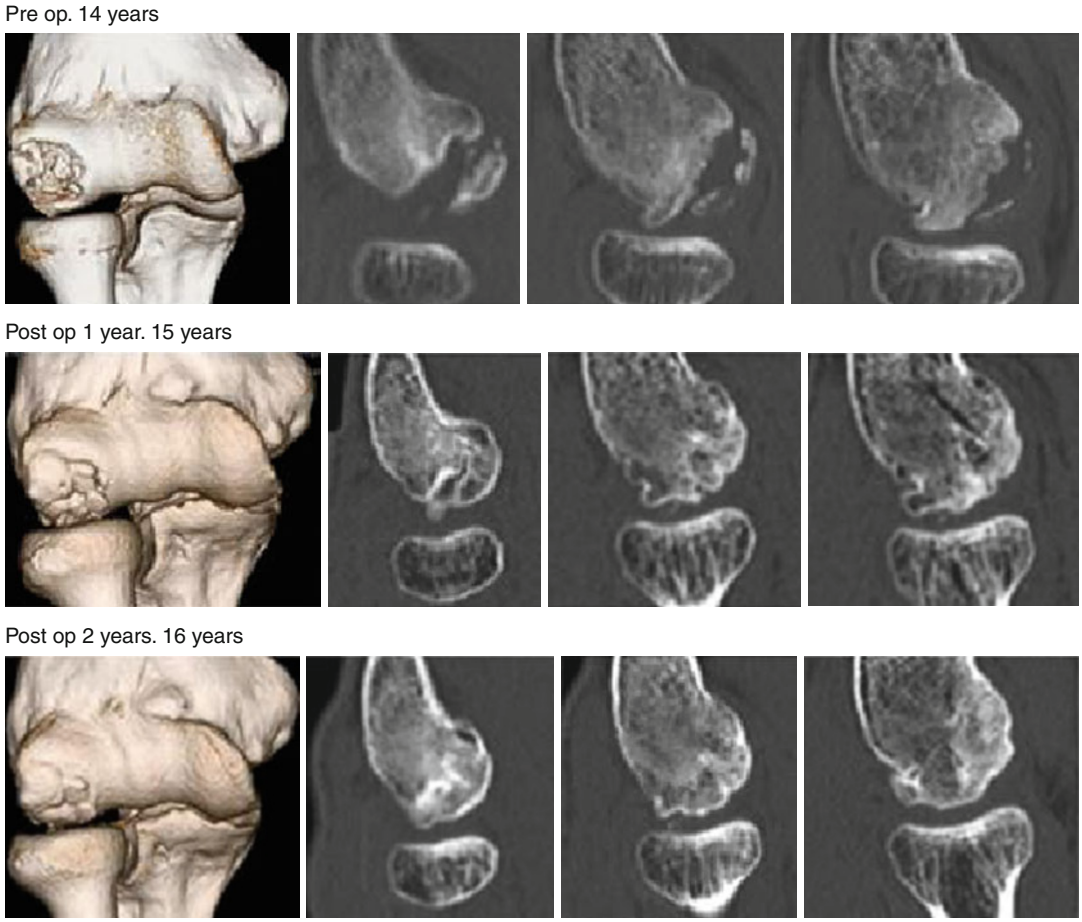

Fig 27.30 Courses of hybrid arthroplasty. *Top*: preoperative CT images showing a bone fragment in the anterolateral side and an extended lesion ranging from the lateral to the central part. *Middle*: postoperative 1 year. Bone union is obtained in the lateral bone fragment and central part. *Bottom*: postoperative 2 years. Further bone formation is seen compared to postoperative 1 year

Table 27.7 Performance over 2 years after hybrid arthroplasty ($n=50$)

Hybrid (mosaic plasty + bone peg fixation) ($n=50$)				
Return	Failed	No pain	Pain	Unclear
49	1	43	5	2
98.0 %	2.0 %	86.0 %	10.0 %	4.0 %

		Pre op.	Post op.
JOA sports score		43.0	92.2
ROM	Flex.	127.4	131.3
	Ex.	–18.0	–9.1

Operative Method

In graft collection, collecting a graft from the fourth rib is difficult, but the graft has a good shape. The sixth rib is flat, and the frontal plane is frequently curved. We collect the graft from the fifth rib. Usually, the rib and costal cartilage part are reached via a horizontal skin incision of the right precordium (Fig. 27.32) followed by subperiosteal detachment, while being careful to avoid pleural damage. Approximately 10 mm of the costal cartilage part and approximately 15–17 mm of the rib part are collected while maintaining continuity of both (Fig. 27.33).

Because a lesion of the capitellum has a defect of the lateral wall and the hypertrophic radial head in many cases, it is easier to excavate the graft bed from the lateral surface (Fig. 27.34). Limbs of the rib with costal cartilage are shaped

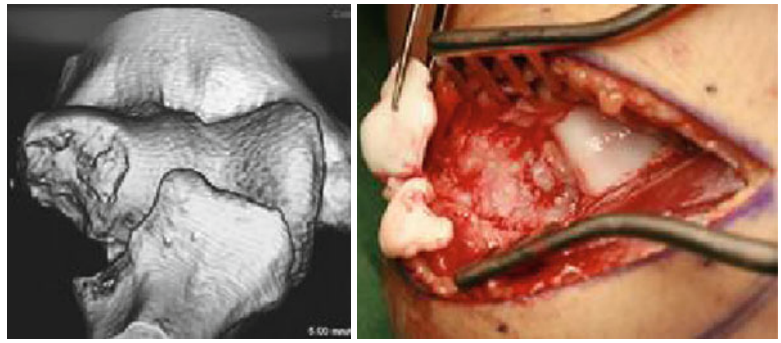

Fig. 27.31 A case of lateral-type destruction. A widespread lesion accompanied by lateral wall defect and joint destruction

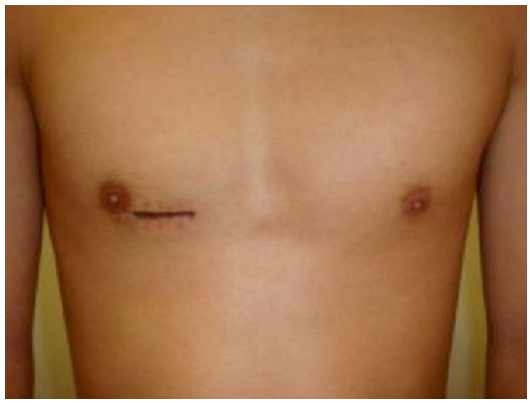

Fig. 27.32 Skin incision. A fragment of the fifth rib with costal cartilage is usually collected from the right precordium through a horizontal skin incision

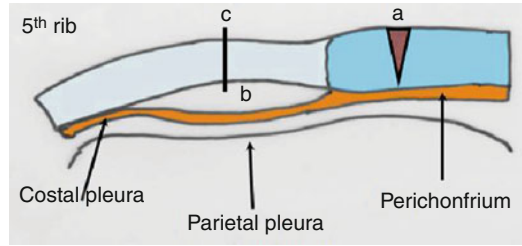

Fig. 27.33 Schema of graft collection. For graft collection, separation of the rib and costal cartilage at their junction at the time of collection can be avoided if sufficient mobility is secured. This can be done by excising in advance the costal cartilage in a V shape using a small round blade knife at the site, approximately 15 mm apart from the costal cartilage junction (*a*). Next, the upper and lower borders of the rib part are detached using a raspatory so that the costal pleura detaches off the back surface of the rib (*b*). The rib is cut off with a bone saw at 2–2.5 cm from the junction, while protecting the pleura with a curved levator to avoid damage (*c*)

with a surgical knife so that it conforms to the bed to allow sufficient contact between the rib with costal cartilage and the bed.

For graft trimming, making the costal cartilage part less than 5 mm in thickness has an advantage of reducing breakage at the costochondral junction. Increasing the area of bone union at the rib part allows for easy acquisition of synostosis. Rib with a length of 10–15 mm is sufficient (Fig. 27.35).

The cartilaginous surface is trimmed conforming to the humeroradial joint surface and fixed with a DTJ screw, etc. (Fig. 27.36). External fixation should be retained for 2–3 weeks. A rehabilitation schedule similar to that of bone peg graft cases should be followed.

According to the results of our 29 cases with at least 2 years of postoperative follow-up, the

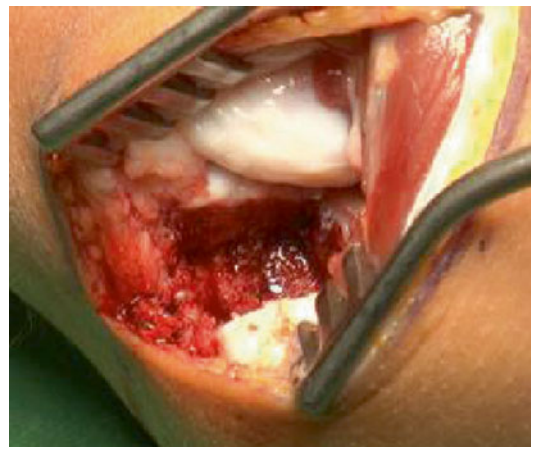

Fig. 27.34 Formation of the bed into a boxy shape

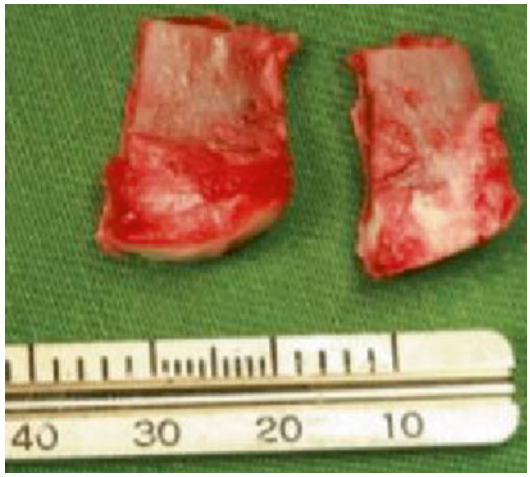

Fig. 27.35 Limbs of the rib with costal cartilage should be shaped with a surgical knife so that it conforms to the bed to allow sufficient contact between the rib with costal cartilage and the bed

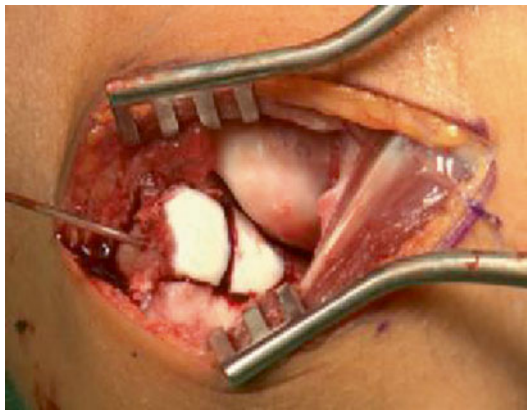

Fig. 27.36 The cartilaginous surface should be trimmed conforming to the humeroradial joint surface and fixed with a DTJ screw, etc.

return rate was 86.2 %, and residual pain was seen in 10.3 % of cases. The range of motion (flexion/extension) also showed approximately 17° of improvement, whereas the JOA score improved from 41.5 points to 85.4 points on average, showing good performance (Table 27.8).

Because this operative method is performed in highly destructed cases, a lower return rate is inevitable. Patients with poor improvement in scores were mainly those with increased OA changes from the preoperative period.

Table. 27.8 Performance over 2 years after costal osteochondral graft ($n=29$)

Costal osteochondral grafts ($n=29$)				
Return	Failed	No pain	Pain	Unclear
25	4	26	3	0
86.2 %	13.8 %	89.7 %	10.3 %	0 %

		Pre op.	Post op.
JOA sports score		41.5	85.4
ROM	Flex.	121.4	129.8
	Ex.	–20.5	–11.6

27.8 Rehabilitation

Rehabilitation programs are basically similar among the operative methods. The programs may be extended in some cases according to the degree of regeneration of individual cases, but they should not be shortened.

Postoperative Schedule

Postoperative 2 weeks: Removal of the plaster slab and stitches. Active range of motion (flexion/extension) exercises and forearm rotation exercises (Fig. 27.37). Stretching of the shoulder, trunk, hip joint, and lower limbs.

Postoperative 4 weeks: Wrist/arm curling (loaded above 0.5 kg).

Postoperative 6 weeks: Wrist/arm curling (loaded above 1 kg) and start of triceps extension.

Postoperative 3 months: Start of coordinated movement, mild practice swinging, and throwing a foam ball straight downward (Fig. 27.38).

Postoperative 4 months: Net throwing and toss/tee batting.

Postoperative 5 months: Light throwing and free batting.

Postoperative 6 months: Throwing no farther than 40 m.

Postoperative 7 months: In the case of a pitcher, throwing to a standing catcher (50–80 %).

Postoperative 8 months: Full pitching from a mound.

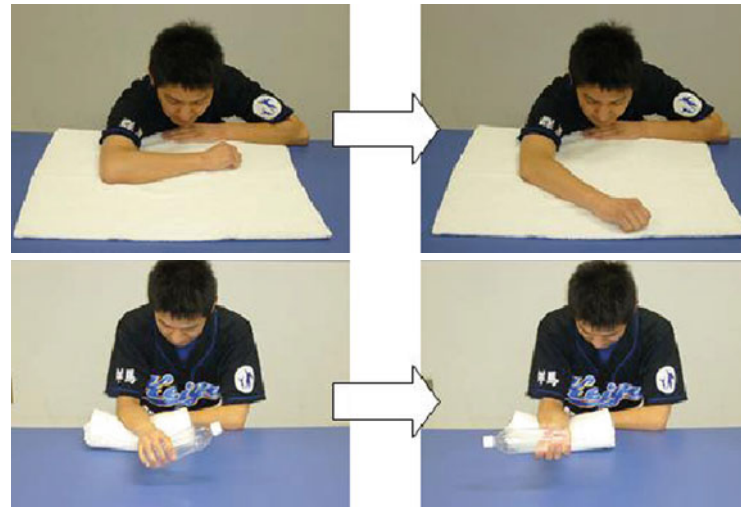

Fig. 27.37 Postoperative training of the range of motion. This should always be an active range of motion (flexion/extension) exercise. Passive exercise is prohibited. *Top*: flexion and extension of elbow. *Bottom*: forearm rotation

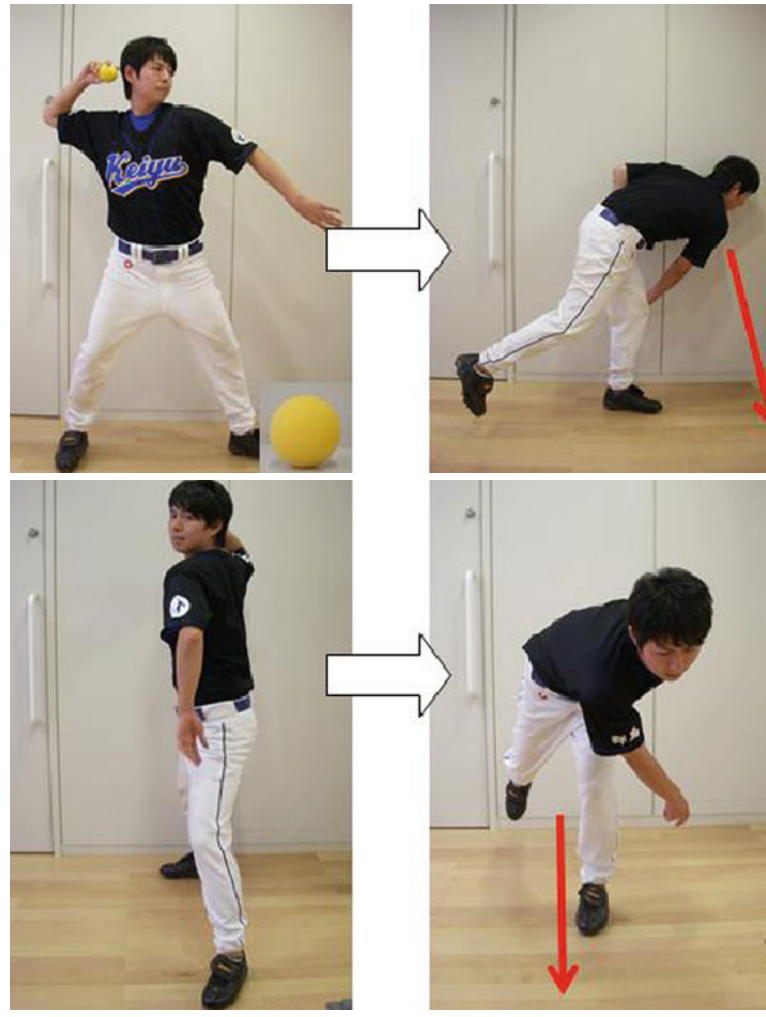

Fig. 27.38 Throwing a sponge ball straight downward. The patient throws a sponge ball straight downward from a limb position of late cocking using rotation of the trunk. This is an exercise in which a ball is thrown so that it bounces straight upward

27.9 Advantages, Pitfalls, and Complications

For both conservative and surgical treatments of this illness, an appropriate treatment leads to a good course. However, a uniform treatment does not necessarily result in good performance for all cases. It is important to choose an operative method depending on the lesion size and state of the articular cartilage.

Important Notes for Conservative Treatment

In case of the initial lateral type at ages of 10–11 years, the lesion progresses slightly even if the patient is resting. We consider this is because vascular insufficiency causes necrosis, and it takes some time to become able to confirm regeneration in imaging findings. Therefore, it is important to continue the conservative treatment first, even if the lesion expands because it is in the initial state.

Natural Healing Course

Despite taking the necessary time for regeneration, the entire lesion is not necessarily regenerated securely. In cases for which the regeneration stops midway because of long-term conservative treatment, we cannot help but move to surgical treatment in some cases. Practically, it is quite difficult to discern the limit of conservative treatment. Based on our clinical experience, we judge that it is time to operate in cases for which the lesion is extended, osteosclerosis around the lesion is obvious, range of motion of the joint is poor, and clear tenderness continues in the capitellum.

Rapidly Progressive Case

We have experienced cases in which continuance of pitching caused deterioration of the lesion, resulting in remarkable OA changes over a short period. Prohibition of pitching is essential for conservative treatment. In addition, it is better to prohibit both batting and strength training of the upper limbs because these also place a load on the lesion.

Postoperative Course

OA changes progress gradually. In addition, loose bodies and synovial plica syndromes may develop in some cases, even if plain radiography shows clear regeneration. For long-term outcomes after surgical treatment, we have observed no progression of remarkable OA changes at present, except for a case of return by self-determination before full regeneration. In our hospital in the period between 2000 and 2004, we conducted a survey of 46 cases that allowed postoperative follow-up over a year to investigate progression of OA changes (13.6 years of age on average, range 11–17 years; 23.5 months postoperative follow-up period, range 12–65 months). Plain radiographic evaluation detected OA changes in 13.6 % of preoperative cases and 65.9 % of postoperative cases. In cases of extended lesions that included the lateral type, a trend was seen in which OA changes were likely to develop even if a surgical treatment was given and OA changes that developed once progressed slightly even after the operation.

Untreated OCD

There are cases in which patients do not necessarily have pain even if there are OA changes, and patients continue to play baseball even though their range of motion is poor. However, OA changes gradually progress, leading to restriction in the range of motion, ulnar nerve symptoms, pain in movement, etc., and this pain can exert influences on daily life. There are not a few patients requiring a surgical treatment because of difficulty in their daily life and job after growing up (Fig. 27.39).

27.9.1 Associated Symptoms

Complications accompanying OCD were seen in 42.8 % of cases. These consisted of medial collateral ligament injury in 29.9 %, articular loose bodies in 26.2 %, synovial plica syndrome in 16.6 %, OA changes in 13.3 %, medial epicondyle avulsion fracture in 13.0 % of cases, and others.

27.9.2 Postoperative Complications

Restricted range of motion, loose bodies, synovial plica syndrome, progression of OA changes,

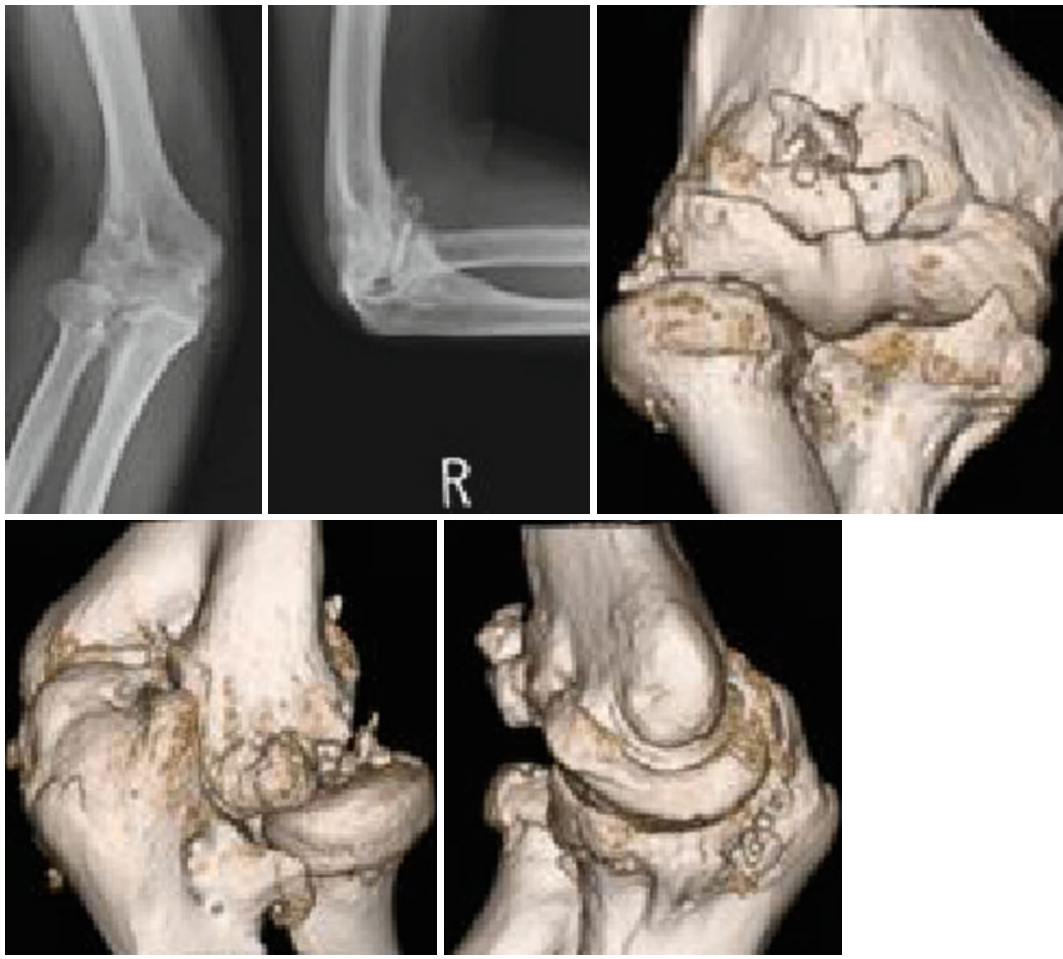

Fig. 27.39 A 38-year-old man who started playing baseball when he was 9 years old became subjectively conscious of reduction in the range of motion when he was 15 years old. He continued to play baseball because he had no pain. However, he experienced inconvenience in his job (nursing care profession) all the time. Because numbness and pain in movement worsened, he visited us. Flexion contracture was 45°, and further flexion was 90°. Intra-articular loose bodies, proliferation of osteophytes, and swelling of the radial head were seen

and other complications are seen in patients who make a full recovery. In severe cases, swelling of the radial head and subluxation are seen.

27.10 Experience in Treatment of Athletes

Case 1

A 13-year-old infielder that started playing baseball at the age of 8 years had shown reduced range of motion of the elbow since 12 years of age. He was receiving a painful training of the passive range of motion at a bonesetter's clinic for 6 months. Because the range of motion of the elbow continued to decrease, the patient was referred to us by a local doctor. His elbow joint was remarkably swollen. The range of motion was −40° of extension, 95° of flexion (+15° and 135° on the healthy side), 45° of pronation, and 80° of supination (Fig. 27.40).

We performed an operation after 3 months, during which we waited for reduction of the swelling. There were irregular loose bodies behind the capitellum, and articular cartilage and subchondral bone of the capitellum were

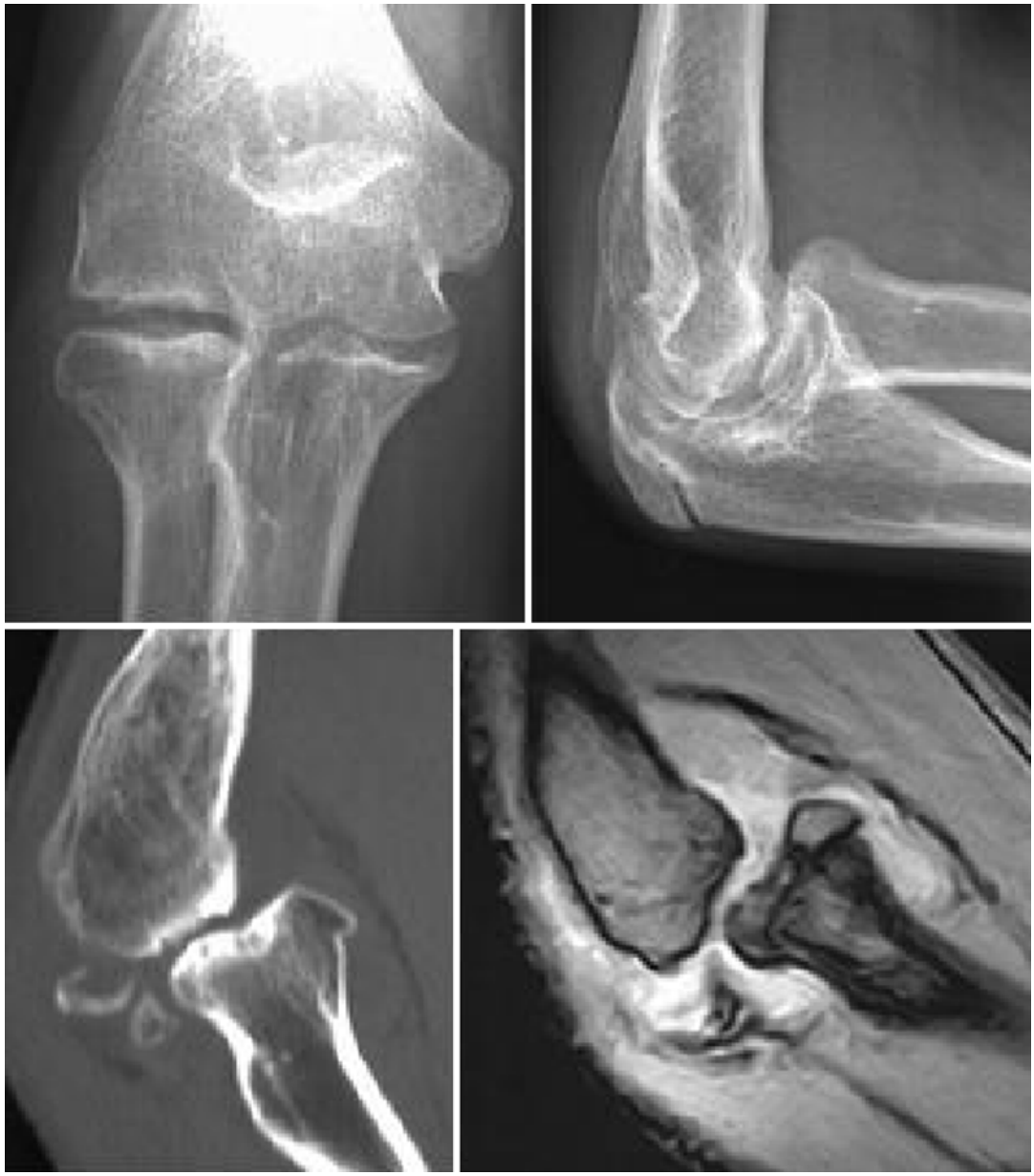

Fig. 27.40 At the initial diagnosis: Plain radiographic images showed a flattened capitellum, swollen radial head, and anterior subluxation. CT detected collapse of the capitellum, loose bodies, and a swollen radial head. The deformation was high, and anterior subluxation was obvious. MRI detected synovitis, flattened capitellum, and subluxation

destroyed in a wide range. At one time, we performed ulnar extension to reposition the radial head after first securing a space of the humeroradial joint. Because cartilage and bone components remained on the large loose body, the loose body was regrafted after refreshing the bed of the capitellum, followed by fixation and collection of the bone pegs from the olecranon. The central part was grafted with 4 osteochondral pillars with a diameter of 4.5 mm from the elbow joint. Postoperative plain radiography showed repositioned subluxation of the radial head (Fig. 27.41).

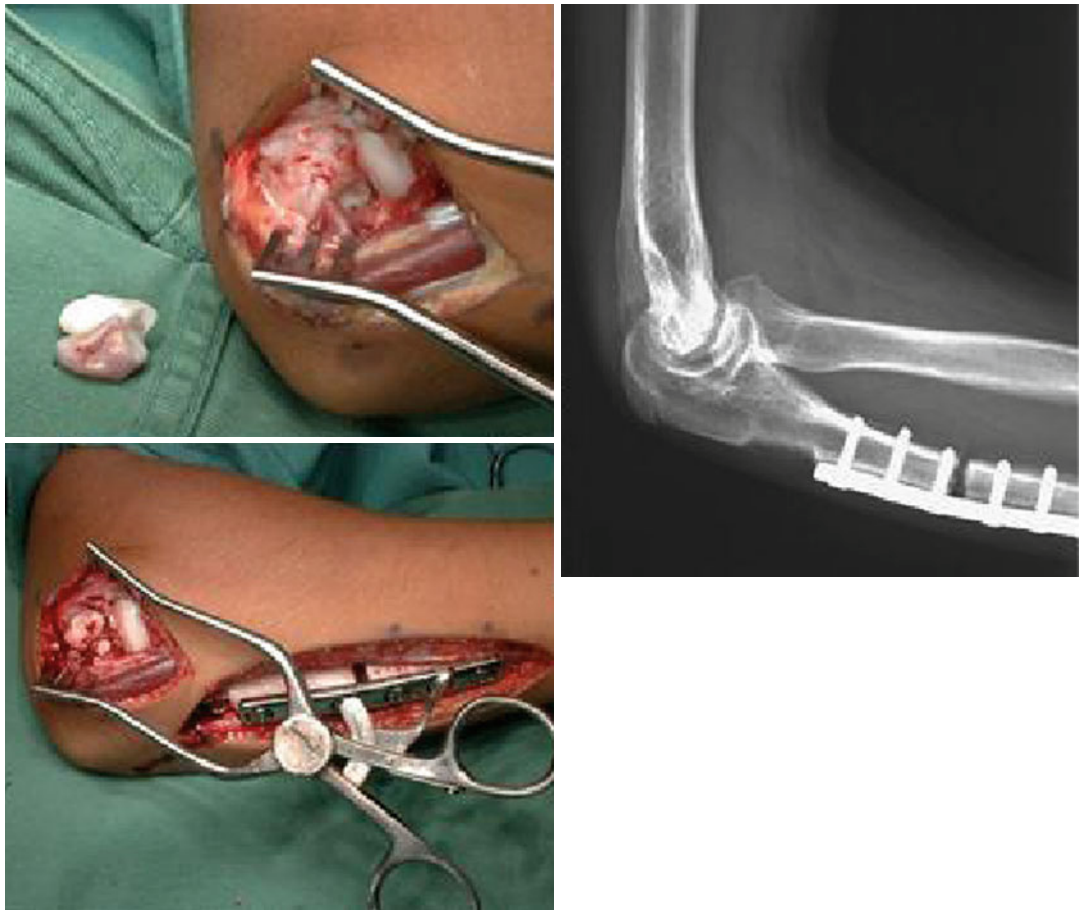

Fig. 27.41 Operative findings: Although the cartilage and subchondral bone were destroyed, a lump of loose bodies was present. We extended the radial head 4 mm by making the ulnar bone slightly curved convexly backward to reposition the radial head luxation. The fixation was made by regrafting the loose body after refreshing the bed of the capitellum. The central part was grafted with osteochondral pillars. Postoperative plain radiography showed repositioned subluxation of the radial head

The postoperative range of motion of the elbow showed no remarkable change because of the long preoperative contracture. Because the patient visited us from afar, making regular visits difficult, we prohibited passive exercise at home and allowed only active exercise. However, the patient received a painful massage at a bonesetter's clinic because an acquaintance of the patient's parent strongly recommended it. The articular range of motion further deteriorated, showing −50° of extension and 80° of flexion. We removed the pegs from the plate and performed mobilization of the joint at postoperative 8 months. Intraoperative improvement of the range of motion was no more than 30–85°. At postoperative 2 years and 8 months, subluxation of the radial head improved, but the capitellum collapsed again. A bone remaining behind the capitellum was observed to extrude, contacting the radial head. The patient was then playing in games because he experienced no pain (Fig. 27.42).

At postoperative 3 years and 8 months, the range of motion was −20° of extension, 85° of flexion, 45° of pronation, and 90° of supination. There was no pain, and only the restricted range of motion remained (Fig. 27.43).

In the initial operation, we considered that even regraft of the loose body would sufficiently

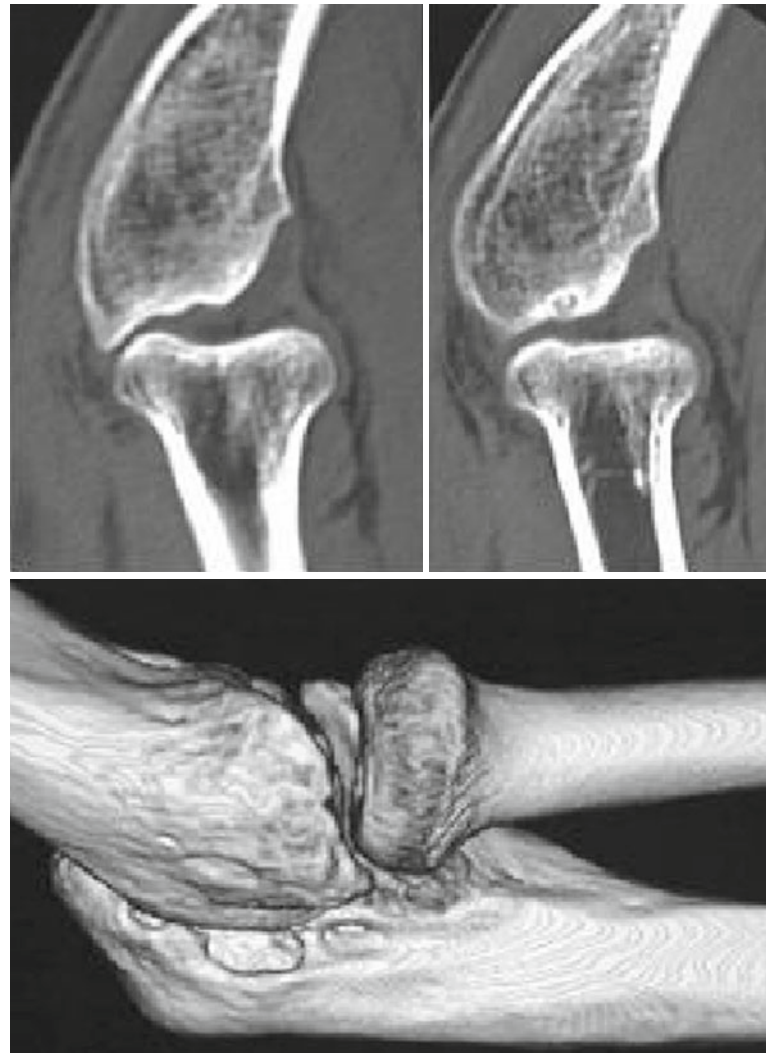

Fig. 27.42 Postoperative 2 years and 8 months. Extrusion and contact behind the capitellum are seen

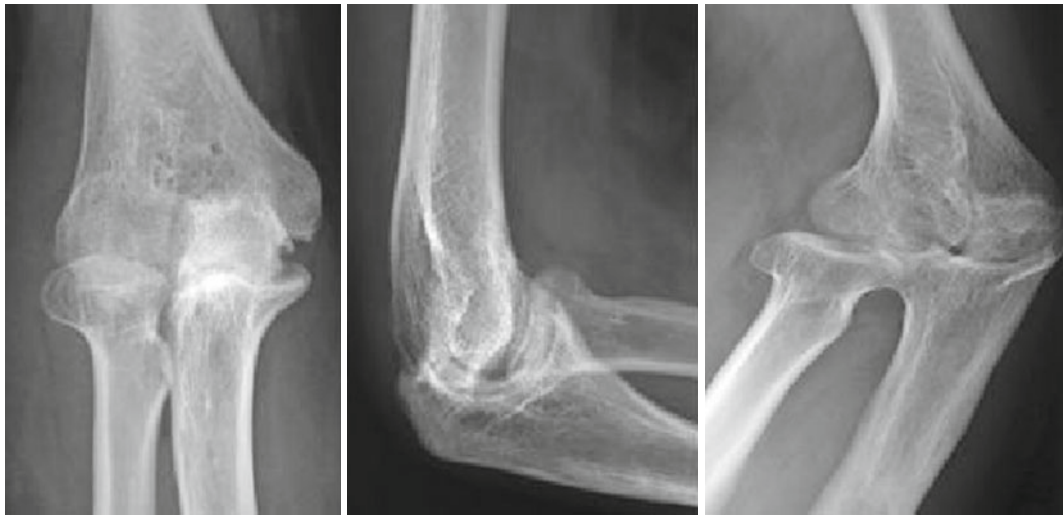

Fig. 27.43 Postoperative 3 years and 8 months (17 years of age). We observed deformation of the capitellum and swelling of the radial head

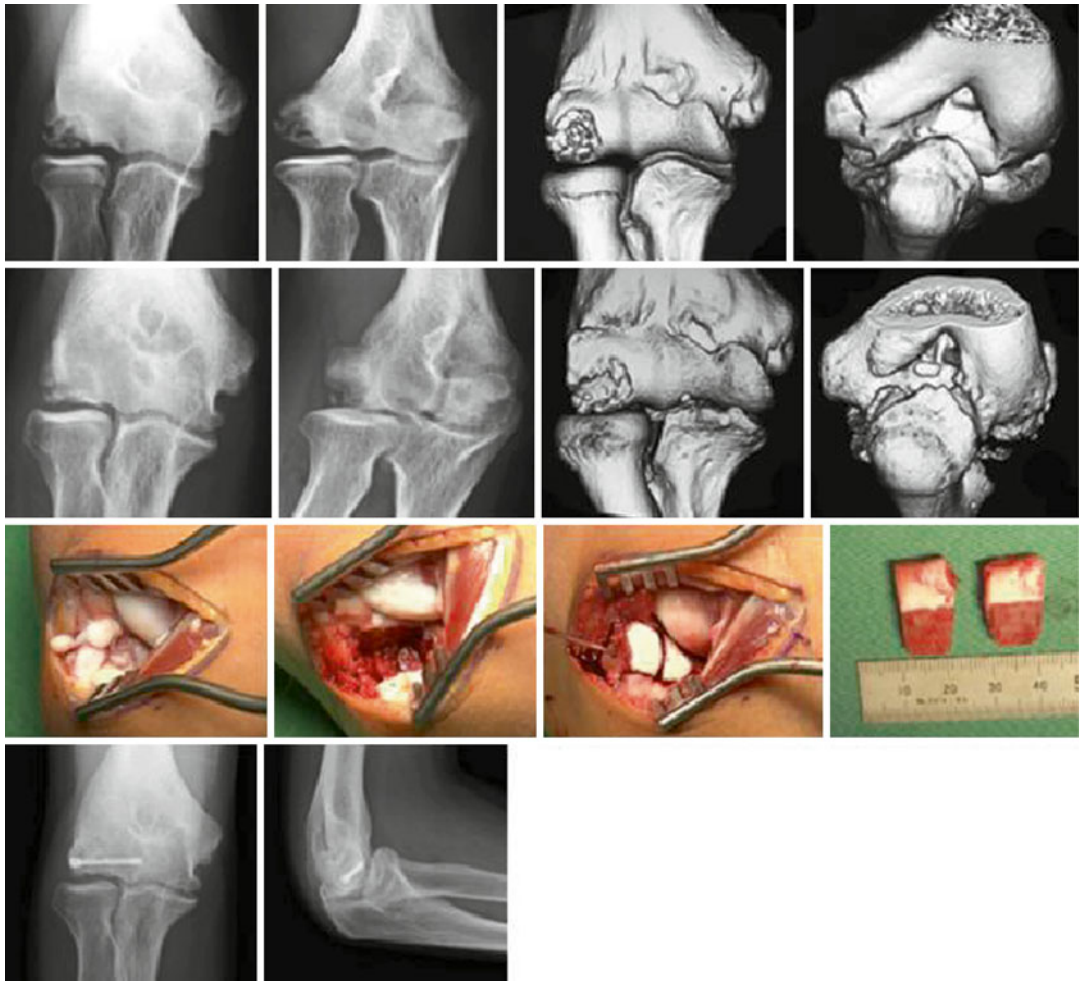

Fig. 27.44 *Top*: plain radiographic and CT images at the initial diagnosis showing an extended lesion ranging from the lateral to the central part. *Second column*: plain radiographic and CT images 8 months after the initial diagnosis showing bone defect of the lesion and osteophytes ranging all over the joint. *Third column*: operative findings showing the loosened articular cartilage of the capitellum and the exposed subchondral marrow. The bed of the capitellum is formed in a boxy shape. The shaped ribs with costal cartilages are grafted and fixed. *Fourth column*: postoperative plain radiographic image showing the fixation with a DTJ screw

allow for reconstruction. However, we subsequently reflected that we should have performed a costal osteochondral graft in this case. Painful manual redress must absolutely not be done because it will further reduce the range of motion of the elbow.

Case 2

A case of costal osteochondral graft (Fig. 27.44).

A 14-year-old infielder belonging to a baseball club experienced pain at the time of throwing that continued for half a year. Pain gradually occurred in his daily life as well. Plain radiography at the time of initial diagnosis showed a lateral extended lesion in the progressive stage, ranging from the lateral capitellum to the center. CT detected necrosis reaching the subchondral marrow that required an operation. However, the patient chose not to visit us and continued playing baseball. The patient revisited us in 8 months because the pain had worsened. For that reason, the lesion had expanded compared to its size at the initial diagnosis. Osteophytes developed all over the joint, causing remarkable OA changes with loose bodies.

The gross pathology at the time of operation showed a wide range of loosened articular cartilage

and lost original form of the capitellum. We reconstructed the joint by using the right fourth and fifth ribs with costal cartilages. The postoperative course was good, and the patient returned to playing baseball. Plain radiography at postoperative 2 years showed slight OA changes. Care needs to be taken because a delayed start of treatment may cause such OA changes, even in younger patients.

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Medial Collateral Ligament Injuries in the Overhead Athletes

28

Elliot S. Mendelsohn, Christopher Dodson, and Joshua S. Dines

28.1 Introduction

Medial ulnar collateral ligament (MUCL) injuries can cause pain and elbow instability in the overhead throwing athlete. One of the earliest reports of an injury to the MUCL was in javelin throwers [1]. Periarticular loose bodies identified on radiographs were initially recognized in professional baseball pitchers [2]. Pitchers were noted to have valgus deformity of the throwing arm, and these loose bodies were discovered to be a result of compression of the radiocapitellar joint surfaces secondary to "medial elbow strain" [3]. Case reports of MUCL injuries were later described in baseball pitchers [4]. Some of the initial reports were of surgical repair of the torn ulnar collateral ligament in an acute setting [5]. Reconstruction of the MUCL became popularized with one of its first success stories, professional baseball pitcher Tommy John by Dr. Frank Jobe. Tommy John surgery, or MUCL reconstruction, was first described using the Jobe technique in 1986 [6]. A modification of this original procedure is still widely used today. As our understanding of the anatomy and biomechanics of the MUCL has evolved, additional techniques have been described to minimize morbidity associated with the procedure and improve outcomes.

28.2 Pathoanatomy, Biomechanics, and Preferred Classification

28.2.1 Pathoanatomy

The medial ulnar collateral ligament complex (MUCL) is composed of three structures: anterior bundle, posterior bundle, and transverse segment [7, 8]. The posterior bundle is a thickening of the elbow joint capsule [7]. The transverse ligament does not cross the ulnohumeral joint, is difficult to identify in all cadaver specimens, and plays no role in the stability of the elbow joint [8]. The origin of the MUCL is located on the posterior and inferior aspect of the medial epicondyle [7, 8]. Although originally thought to have a common origin, separate origins have been described for the different bundles on the medial epicondyle. The anterior bundle is divided into anterior and posterior fibers [7, 8]. The anterior fibers insert on to the sublime tubercle of the ulna. There is variability in the anatomy of the origin, insertion, and width of the anterior bundle

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of the ulnar collateral ligament [9]. The mean length of the anterior portion of MUCL is about 27 mm and the mean width is 4–5 mm [8]. Part of this variability can be explained by an increase in width of the ligament toward its insertion. It has a broad insertion on the sublime tubercle from within several millimeters of the joint line and it tapers distally [9]. The flexor carpi ulnaris is the predominant muscle of the flexor-pronator muscle group that originates overlying the MUCL and is anatomically situated as an important dynamic restraint to valgus elbow instability [10]. The flexor digitorum superficialis muscle is the only other significant contributor. The role of these muscles as dynamic stabilizers may have implications in the rehabilitation of the overhead throwing athlete.

28.2.2 Biomechanics

The pathology generated in the thrower's elbow is a result of the forces generated during throwing. Tensile forces are generated medially (which leads to injuries to the MUCL, ulnar nerve, flexor-pronator musculature), and compressive forces are generated laterally (which leads to radiocapitellar arthrosis and loose bodies). Shear forces are generated posteriorly during late acceleration and follow through phases of pitching (which leads to posteromedial impingement and osteophytes). Kinematic studies of baseball pitchers have shown that the elbow experiences valgus forces that are greatest during the late cocking and early acceleration phase of throwing [11]. When the shoulder reaches maximum external rotation, 64 N-m of elbow valgus torque is generated [11]. Biomechanical tests of the strength of the anterior bundle of the MUCL show average failure load of 260 N [12]. Every pitch approaches the maximal load to failure of the MUCL complex [11]. This finding reinforces the importance of the dynamic stabilizers of the elbow. The palmaris longus tendon, the most common source of autograft for ligament reconstruction, has been shown to have a similar failure load of 357 N [12].

The MUCL is the most important ligamentous static restraint to valgus elbow instability [13]. Deficiency of the anterior bundle of the MUCL alone will create valgus elbow instability [14, 15]. In contrast to the isometric position of the lateral collateral ligament, the MUCL origin is posterior to the axis of flexion-extension of the ulnohumeral joint. [8] Flexion and extension of the elbow joint creates reciprocal tension in the anterior and posterior fibers of the anterior bundle of the MUCL due to a cam effect as the elbow is brought into flexion [14]. The distance between the origin and insertion of the anterior bundle of the MUCL increases slightly from extension to 60° of flexion and then remains relatively constant. An isometric group of fibers within the MUCL does not exist, but there are fibers within the central portion that approximate true isometry and serve as the basis for single-strand reconstruction techniques [16]. The posterior bundle of the MUCL is a secondary restraint to valgus instability. Isolated sectioning of the posterior bundle does not lead to valgus elbow instability unless the anterior bundle of the MUCL is also deficient [14]. As the anterior bundle of the MUCL is the primary ligamentous restraint to elbow valgus instability, reconstructive efforts have focused on restoring the function of this ligament.

The flexor-pronator muscle groups act as an important dynamic restraint to valgus elbow instability. There is increased EMG activity of the flexor-pronator muscle group during the late cocking and early acceleration phase of throwing [17, 18]. The FCU is considered to be a primary dynamic stabilizer and the FDS a secondary stabilizer because contraction of the FCU alone allowed correction of the valgus instability in cadaveric specimens with MUCL tears [19]. Muscles that cross joints increase the joint reaction force during contraction. This effect can increase the constraint from the bony geometry of the ulnohumeral articulation which has been described as a "sloppy hinge joint."

28.2.3 Preferred Classification

Injuries to the MUCL are commonly classified as acute or chronic. Chronic injuries are repetitive, overuse injuries without a history of a traumatic

event. Acute injuries are injuries where the athlete recalls a single throw or traumatic event where a valgus load was applied to the arm. Oftentimes, injuries to the MUCL are the result of an acute episode or traumatic event in the setting of underlying microtrauma as a result of repetitive overhead throwing.

28.3 Clinical Presentation and Essential Physical Examination Maneuvers

A detailed history is important in the evaluation of the throwing athlete. They may recall a particular throw where they experienced a "pop" in the elbow. Oftentimes, there was not a specific injury, but the patient will complain of a decrease in throwing velocity or accuracy. It is important to elicit which phase of the throwing cycle the athlete experiences symptoms. The athlete with an injury to the MUCL commonly describes medial elbow discomfort prior to ball release. Pain after ball release is more often due to valgus extension overload syndrome. Numbness and tingling should alert the examiner to concomitant ulnar neuritis. Arm dominance, player position, level of play, and duration of nonoperative treatment are important elements of the history that may guide operative decision making. For example, symptoms in the nondominant arm of an outfielder who plays in a recreational league may be successfully managed with nonoperative treatment, whereas symptoms in the dominant arm of a major league baseball pitcher may be a career-ending injury without surgical reconstruction.

A deficiency in the kinetic chain of throwing can occur anywhere from the core to the upper extremity, so physical examination in the overhead athlete begins with an evaluation of core strengthening. Evaluate for "cork screwing" or inability to maintain balance while squatting on one leg. It is important to examine the ipsilateral shoulder for rotation deficits. There is some evidence to suggest that glenohumeral internal rotation deficit is associated with valgus elbow injuries in baseball players [20]. On examination of the elbow, inspect the medial skin for signs of acute injury such as overlying ecchymosis and edema. Range of motion deficits, particularly flexion contractures, are common in baseball pitchers and usually do not create functional impairment. A variety of tests have been described to assess for valgus elbow instability. We prefer the moving valgus stress test, described by O'Driscoll [21]. With the patient sitting upright and the shoulder at maximal external rotation, a valgus stress is applied to the elbow as it is extended to 30° of flexion (Fig. 28.1). The test is considered positive if the pain is reproduced or the point of maximum pain is from 120° to 70° of flexion. In a group of 21 patients, the test had a sensitivity (100 %) and a specificity (75 %) when compared to arthroscopic diagnosis [21]. The examiner should evaluate for ulnar neuritis in all patients with suspected MUCL sufficiency because of the high degree of association. Assess for a Tinel's sign over the cubital tunnel or reproduction of numbness and tingling in the ulnar one and one half digits with prolonged flexion of the elbow. Spontaneous subluxation of the ulnar nerve with elbow flexion can be an asymptomatic

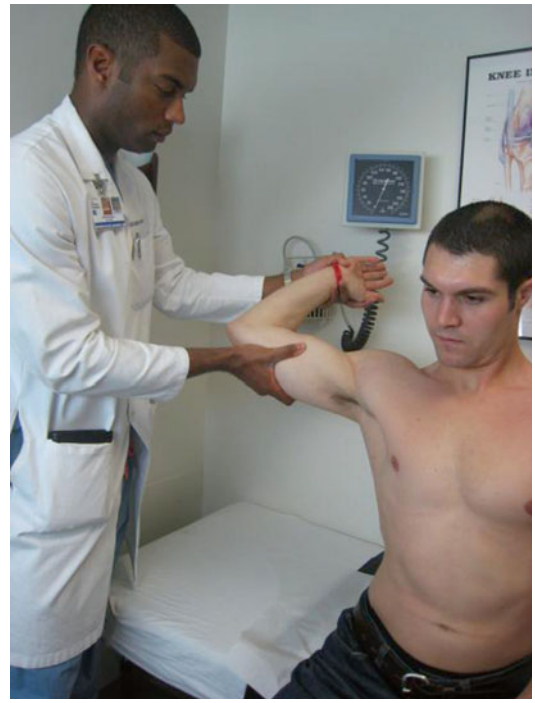

Fig. 28.1 Moving valgus stress test

finding. Nerve conduction studies can be obtained but may be falsely negative because compression of the nerve is often a dynamic phenomenon experienced only during the throwing motion. We have found the history and physical examination findings to be more accurate in guiding treatment. In athletes with acute traumatic valgus injuries, palpate for muscle ruptures along the origin of the flexor-pronator mass muscle belly. Remember that medial epicondylitis is a common source of medial elbow pain in the overhead throwing athlete.

28.4 Essential Radiology

The workup of medial elbow pain in the overhead athlete begins with an anteroposterior and lateral radiograph of the elbow. Radiographs should be examined for osteophytes associated with posteromedial impingement, radiocapitellar arthritis, and intra-articular loose bodies. An oblique view has been described for optimal visualization of posteromedial olecranon spurs, but in our experience, a good lateral radiograph is sufficient. Stress radiographs have fallen out of favor and are not routinely obtained. Magnetic resonance imaging (MRI) of the elbow is usually performed with contrast to evaluate for an injury to the MUCL and intra-articular pathology. A T-sign has been described to diagnose tears of the MUCL as fluid extravasates between the MUCL and its origin on the humerus where it has peeled off [22] (Fig. 28.2). Whereas nonenhanced MRI has a high specificity (100 %) but a low sensitivity (57 %) [22], saline-enhanced MRI arthrography increases the sensitivity to 92 % [23]. Stress ultrasound is emerging as an alternative technique to MRI in the evaluation of MUCL injuries [24].

28.5 Disease-Specific Clinical and Arthroscopic Pathology

MUCL injuries are often partial-thickness tears associated with chronic overuse injuries and microtrauma. Full-thickness tears may be seen in patients who sustain contact-associated valgus loading of the elbow. Ulnar neuritis may develop in association with medial elbow instability as traction is applied to the ulnar nerve from valgus instability.

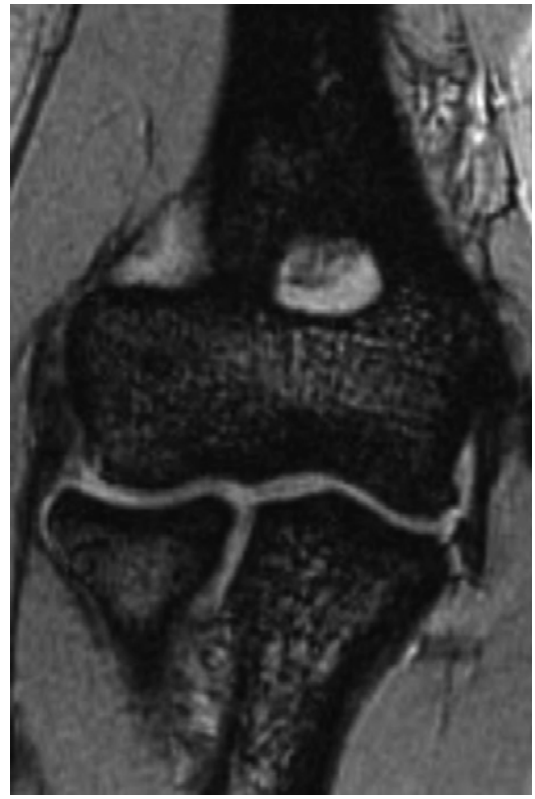

Fig. 28.2 T-sign

Several authors have described arthroscopic techniques to assist with diagnosis of an MUCL injury [25, 26]. Only the most anterior 25 % of the anterior bundle can be visualized arthroscopically [26]. Because the ligament cannot be visualized in its entirety arthroscopically, a cadaveric study looked at the degree to which the medial compartment gaps open with stress arthroscopically. Field and Altchek found that at 70° of elbow flexion, sectioning of the anterior bundle of the MUCL leads to 1–2 mm of opening, whereas sectioning of the entire MUCL leads to 4–10 mm of opening [26]. The advantage of arthroscopy is that it can be used to address intra-articular pathology that otherwise might not be

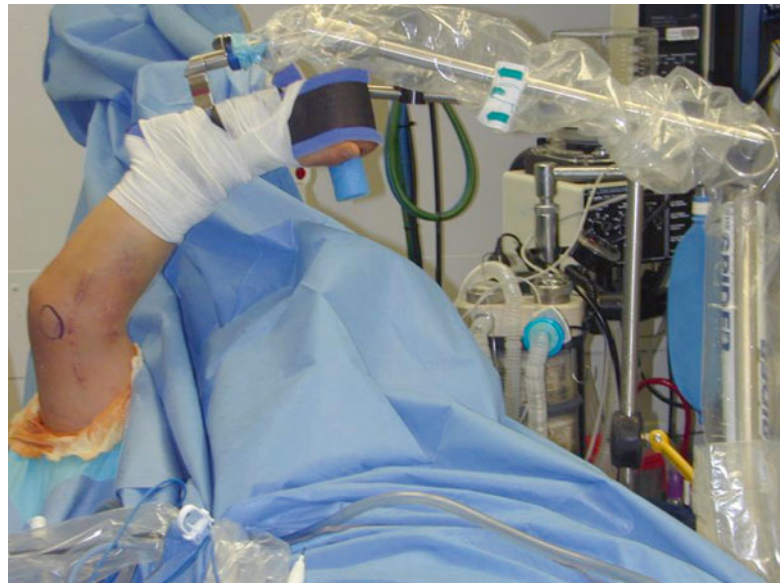

Fig. 28.3 Setup for elbow arthroscopy

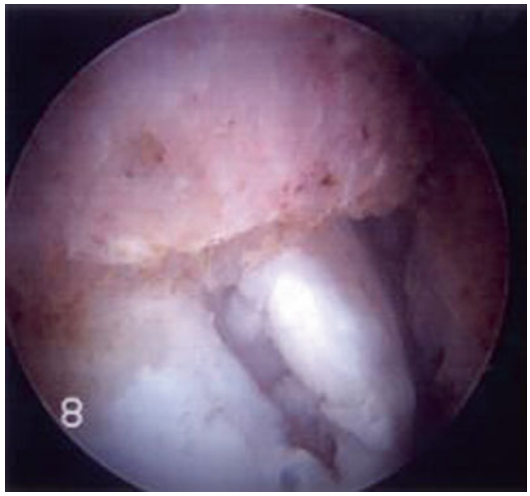

Fig. 28.4 Chondral full-thickness lesion

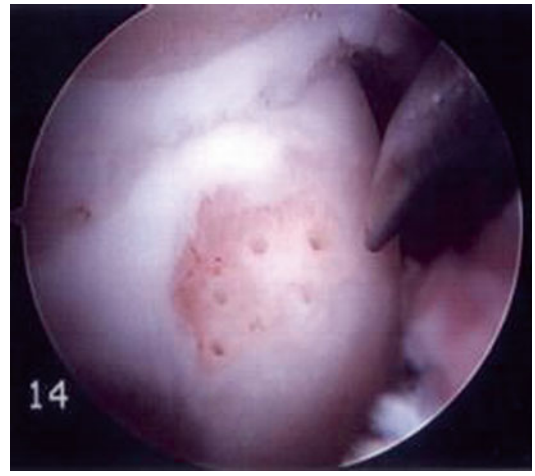

Fig. 28.5 Microfracture

accessible from an open medial exposure of the ulnohumeral joint. Chondral lesions typically present on the radiocapitellar joint surface due to lateral compressive loading. Microfracture and chondroplasty are arthroscopic techniques that can be used to address chondral lesions. Posteromedial olecranon spurs can be removed arthroscopically or from an open medial approach (Figs. 28.3, 28.4, 28.5, and 28.6).

28.6 Treatment Options

Surgical treatment options for MUCL insufficiency generally involve reconstruction of the ligament. Repair of the MUCL is mainly of historical interest only. There is some evidence to suggest that repair of the MUCL may be beneficial in the younger, nonprofessional athlete. Savoie et al. performed a repair using mostly

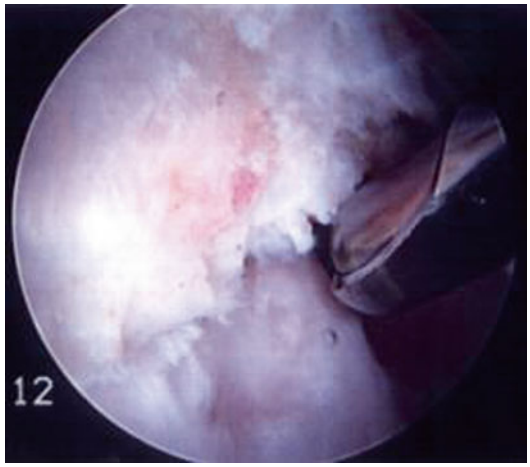

Fig. 28.6 Removal of posteromedial osteophyte with burr

suture anchors in 60 patients (average age 17) and reported good to excellent outcomes in 93 % with return to play at 6 months. There were 4 failures and the average follow-up was 5 years. Other studies have shown higher failure rates with MUCL repair compared to reconstruction [27].

The Jobe technique is the first procedure to describe reconstruction of the MUCL. The procedure involves elevation of the flexor-pronator mass off the medial epicondyle, creation of tunnels in the sublime tubercle and medial epicondyle, and passage of a free graft (usually palmaris longus autograft) in a figure-of-eight fashion. Ten of 16 (68 %) patients returned to previous level of play in his original series [6].

A muscle-splitting approach was described by Smith and Altchek to minimize the morbidity associated with surgical dissection of the flexor-pronator musculature [28]. Twenty-two patients underwent repair or reconstruction using a muscle-splitting approach without neuropathy. It involves tunnel placement and graft passage through the raphe of the flexor carpi ulnaris in the safe interval between the median and ulnar nerves.

Andrews described a modified Jobe technique that involved routine transposition of the ulnar nerve under a fascial sling. Cain et al. reported Andrews' experience using this technique in 1,281 athletes with 79 % follow-up at 2 years. Eighty-three percent of reconstructions returned to the same level. Sixty-three percent of repairs returned to the same level. Athletes returned to play on average at 11.6 months and initiated a throwing program at 4.4 months.

Altchek coined the docking technique, which involved "docking" of the graft into a single tunnel on the ulna and the humerus with the sutures tied over smaller tunnels to create a bone bridge. This reduces the number of large drill holes in the medial epicondyle from three to one. Dodson et al. described Altchek's experience using the docking technique in 100 consecutive patients. A subcutaneous ulnar nerve transposition was performed selectively in a few cases. Ninety out of 100 competed at the same level or higher for more than 12 months. There were 2 poor results. There were 3 postoperative complications (2 late ulnar nerve transpositions and 1 arthroscopic lysis of adhesions).

Another technique is called the DANE TJ procedure named to give credit for those who envisioned it (David Altchek, Neal ElAttrache, Tommy John). It involves a hybrid form of fixation with a docking technique proximally and interference screw fixation distally. Dines et al. described the results of the DANE TJ technique in 22 athletes. Nineteen of 22 achieved excellent outcomes, 4 of 22 (17 %) had complications, and 3 required second surgery (2 with arthroscopic lysis of adhesions, 1 with posteromedial osteophyte debridement, all achieved excellent outcomes). Advantages of this technique include its application for revision procedures and sublime tubercle insufficiency where tunnel fracture and/or placement may be potential issues. Proponents of this technique argue that optimal graft tensioning is easier. Ahmad et al. described a technique that involved interference screw fixation proximally and distally in an attempt to more closely recreate ligament isometry [29].

In the initial reports of reconstruction of the MUCL, ulnar nerve transposition was routinely concomitantly performed. However, high incidences of postoperative ulnar neuropraxia led to more selective use of ulnar nerve transposition. When Conway et al. reported Jobe's 13-year experience, there was transfer of the ulnar nerve in 56 patients that led to 68 % return to previous

level of performance, 24 % ulnar nerve-related symptoms(both transient and non-transient), and a 13 % reoperation rate for ulnar nerve-related symptoms [30]. In a later report of 83 patients without nerve transfer using the Jobe technique and the muscle-splitting approach, 82 % returned to their previous level of performance, 5 % had transient nerve-related symptoms, and there were no reoperations [31]. Today most surgeons recommend nerve transposition on a select basis.

Biomechanical studies using a cyclic loading protocol compared the docking technique, figure-of-eight technique, interference screw fixation, and suspensory fixation (Endobutton) [32]. All failed at lower loads than the native MUCL. The docking technique and suspensory fixation showed the highest peak loads to failure. Clinically, excellent outcomes and low failure rates have been obtained with many of these techniques, and none has demonstrated superiority.

28.7 Authors' Preferred Treatment

We prefer the docking technique as described by Altchek. The first stage of the procedure involves arthroscopy of the elbow, if indicated, to address intra-articular pathology such as chondral injury or posteromedial osteophytes. The graft is then harvested (Fig. 28.7). We use a palmaris longus autograft if available from the ipsilateral or contralateral extremity. If not available, we use a gracilis autograft from the contralateral (plant leg when throwing) lower extremity. The palmaris longus tendon is harvested from a 1 cm incision placed over the volar wrist crease. The visible portion of the tendon is tagged with a no. 1 Ethibond suture in a Krackow fashion. The proximal portion of the tendon is harvested with a tendon stripper. The incision is closed and the tendon is placed in moistened lap sponge.

The arm is exsanguinated and a tourniquet is elevated. We make an 8–10 cm incision over the medial elbow from the distal third of the intermuscular septum to 2 cm beyond the sublime tubercle. Branches of the medial antebrachial cutaneous nerve are identified with vessel loops

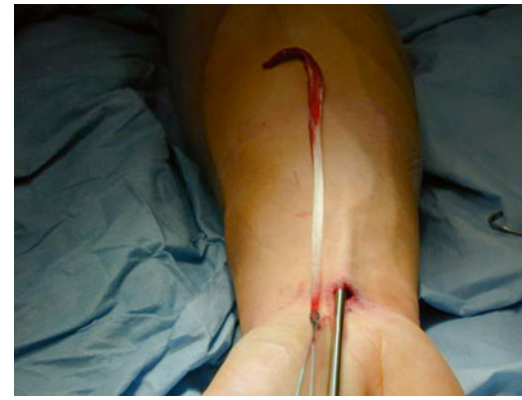

Fig. 28.7 Harvest of palmaris longus tendon

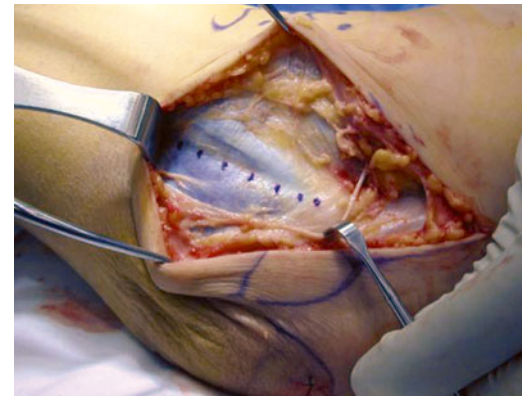

Fig. 28.8 Muscle-splitting approach

and carefully retracted. A muscle-splitting approach is developed through the posterior third of the common flexor-pronator mass musculature and within the anterior fibers of the flexor carpi ulnaris (Fig. 28.8). An incision is made longitudinally along the anterior bundle of the MUCL.

The location of the ulnar tunnel is identified after exposing 4–5 mm posterior to the sublime tubercle in a subperiosteal fashion. We use a 3 mm burr for creation of anterior and posterior tunnels on the sublime tubercle with a 2 cm bone bridge between tunnels. The tunnels are connected with a small, curved curette. A suture passer is passed through the tunnels to shuttle looped sutures through them and aid with graft passage. Sutures are passed and tied over the bony bridge after the graft is docked in the ulnar tunnel. The humeral epicondyle is carefully

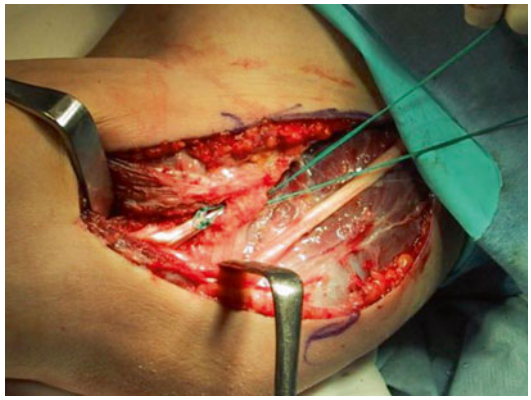

Fig. 28.9 Passage of sutures through bone tunnels on the medial epicondyle after docking of graft

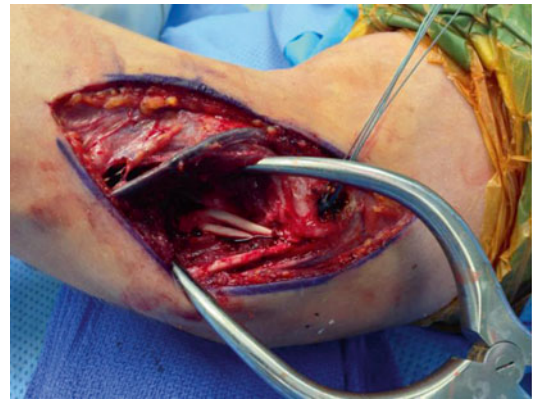

Fig. 28.10 Appearance of graft after final tensioning

exposed without dissection of the ulnar nerve unless transposition is planned. The origin of the humeral tunnel is identified and a longitudinal tunnel is created using a 4 mm burr. Two smaller anterior tunnels are created with the use of a 1.5 mm burr anterior to the intramuscular septum approximately 5–10 mm apart. The incision in the native MUCL is repaired with 2-0 absorbable suture. Sutures are shuttled through the tunnels using a suture passer and shuttling technique as previously described.

The forearm is supinated and a slight varus stress is applied to the elbow. The limb of the graft with sutures is passed through the ulnar tunnel from anterior to posterior and "docked" into the humeral tunnel with sutures exiting one of the smaller 1.5 mm tunnels. The graft is tensioned in flexion and extension to determine what length is optimal before securing the second limb of the graft in the humeral tunnel. The other limb is marked and a no. 1 Ethibond suture is placed in a Krackow fashion. The excess graft is removed and the graft is docked into the humeral tunnel with the sutures exiting the other 1.5 mm tunnel (Fig. 28.9). The elbow is taking through full range of motion prior to final graft tensioning, and once satisfied, the sutures are tied over the bone bridge on the medial epicondyle (Fig. 28.10). The tourniquet is deflated and hemostasis is obtained. The flexor-pronator fascia is reapproximated and the wound is closed in layers. We perform an ulnar nerve transposition only if indicated based upon preoperative examination. The elbow is placed in a well-padded, plaster splint at 45° of flexion.

28.8 Rehabilitation

At the first postoperative visit, the sutures are removed and the patient is placed in a hinged elbow brace. For the first 3 weeks, we allow motion from 30° to 90°. From the third to the fifth week, motion is advanced to 15° of extension and 115° of flexion. We remove the hinged elbow brace after 6 weeks. Patients are then started in physical therapy. Physical therapy initially focuses on passive elbow, shoulder, forearm, wrist, and hand range of motion. At 12 weeks, we allow a more aggressive program that includes shoulder and scapula strengthening. Usually a formal tossing program is begun at 4 months. If patients can throw pain free to 180 ft at 9 months, we allow them to begin pitching from a mound. Patients are generally now allowed to return to competitive pitching about 1 year after surgery.

Nonoperative treatment can be successful in returning some athletes to competition. A supervised rehabilitation program consisting of rest for 2–3 months followed by progressive strengthening and throwing with gradual return to play allowed 41 % of athletes to return to play at their previous level of performance at an average of 24.5 weeks [33].

28.9 Advantages/Pitfalls/ Complications

The most common complication described with reconstructive MUCL surgery is injury to the medial antebrachial cutaneous injury. Other more serious complications include retear, ulnar neuropathy, fracture, arthrofibrosis, graft site morbidity, valgus extension overload, infection, saphenous neuropathy (gracilis autograft), and RSD [34]. Revision surgery for ligament reconstruction is not as successful and the overall return to play after a failure or complication is 84 % [34].

We present some pearls that may decrease the risk of complication. Perform meticulous superficial dissection with bipolar electrocautery and vessel loops to minimize iatrogenic injury to branches of the medial antebrachial cutaneous nerve. Maintain at least 10 mm between bone tunnels to minimize iatrogenic fracture. Consider interference screw fixation as a bailout for tunnel fracture. Protect the ulnar nerve carefully at all times, especially during tunnel placement to minimize risk of ulnar nerve injury. Avoid violation of the posterior cortex of the medial epicondyle during creation of the humeral tunnel. Carefully protect the ulnar nerve during subperiosteal exposure of the ulnar tunnel on the sublime tubercle. Plan tunnel placement based upon anatomic landmarks to minimize the risk of tunnel anisometry. Avoid aggressive posteromedial resection and limit resection to only pathologic structures to minimize risk of valgus instability and stress on graft.

28.10 Experience in Treatment of Athletes

Professional and college-level athletes generally have good to excellent outcomes after MUCL reconstruction. Several studies have demonstrated that elite-level athletes can return to sport at a rate of 82–92 % after MUCL reconstruction [31, 35]. Athletes without prior surgery undergoing a primary procedure have been shown to have a higher rate of return to play [31]. A high rate of failure (26 %) has been documented in high school baseball players after MUCL reconstruction [36]. Year-round baseball was the number one risk factor for MUCL tears in this group. Professional quarterbacks with MUCL injuries can be successfully managed with nonoperative treatment [37]. Throwing a football may place different stresses on the elbow compared to throwing a baseball. Carefully consider the player's sport and position when managing athletes with these injuries.

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Treatment of Unstable Simple Elbow Dislocations

29

Tracy Webber and Jennifer Moriatis Wolf

29.1 Introduction

The elbow is the second most common joint dislocation in adults, with the shoulder being the most common. Elbow dislocations occur at a median age of 30 years [1]. The annual incidence of elbow dislocation is 6.1/100,000 [2]. Elbow dislocations make up 10–25 % of all injuries to the elbow [1]. A simple elbow dislocation involves injury to soft tissue only without fractures of the radial head, proximal ulna, or distal humerus, while a complex elbow dislocation involves associated fractures of the radial head, coronoid, or distal humerus [2]. Most elbow dislocations are stable after closed reduction, and treatment should consist of early range of motion [1]. However, there is a small subset of simple elbow dislocations that are not stable after closed reduction and require operative treatment for stability and function. In an unstable simple elbow dislocation, the elbow joint is not congruent or subluxes after reduction, or it requires more than a 45° extension block to maintain reduction. This is often due to interposed soft tissues or alternately to ligamentous instability. Isolated elbow dislocation without any osseous lesions causes chronic instability in only 2 % of cases and is usually treated nonoperatively with early range of motion, while most fracture-dislocations require surgical treatment [3].

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29.2 Pathoanatomy/ Biomechanics/Preferred Classification

The most common mechanism of injury for elbow dislocation is a fall onto an outstretched hand. Common activities that lead to elbow dislocations include sports and motor vehicle accidents [4]. With a fall onto the outstretched arm, the elbow experiences axial compression, and the body internally rotates while the forearm externally rotates resulting in forearm supination. This causes a valgus moment on the elbow [5]. The combined motion of valgus, supination, and axial load on the elbow leads to posterior dislocation as the coronoid process passes posterior to the trochlea.

Biomechanically, the elbow fails in three sequential stages, described by O'Driscoll as a ring of instability. Stage 1 is failure of the LCL, creating posterolateral rotatory subluxation, which self-reduces. Stage 2 occurs with continued force leading to anterior and posterior capsule disruption. An elbow with only LCL and capsular failure but an intact MCL is a perched dislocation as demonstrated biomechanically by O'Driscoll et al.

In stage 3A the posterior bundle of the MCL is disrupted as well, allowing posterolateral rotation

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and posterior dislocation. However, the anterior bundle of the MCL is intact, explaining post-reduction valgus stability. Stage 3B is disruption of the entire MCL, with varus, valgus, and rotatory instability. Articular congruity and muscle tone provide post-reduction stability [5, 6]. Although three stages to dislocation are described, clinically a dislocated elbow will have a disrupted MCL (stage 3A or 3B). Both the LCL and MCL usually tear from their humeral origin. Additionally, there can be injury to the brachialis muscle.

Elbow dislocations are first classified as simple or complex, based on the presence or absence of associated fractures. Simple elbow dislocations make up 80–90 % of elbow dislocations [4]. While there are often bony flecks noted at the disrupted ligament attachments, including coronoid tip fractures, the bony anatomy is grossly intact in a simple dislocation.

Next, the dislocation is classified by the direction of olecranon displacement. It can be posterior, posterolateral, lateral, medial, anterior, or divergent. Posterior and posterolateral dislocations make up 90 % of all elbow dislocations [1]. Anterior and divergent dislocations are rare and are associated with high-energy mechanisms.

The degree of instability is correlated with the amount of damage to the extensor and flexor muscle origins at the epicondyles [7]. These dynamic stabilizers along with the inherent bony stability of the joint keep the elbow reduced while the avulsed collateral ligaments are healing [7].

Dislocation can also be classified based on the time since injury. Acute dislocations present within 2 weeks of injury, subacute within 2–6 weeks, and chronic greater than 6 weeks after injury. The more time that has passed since dislocation, the more difficult it is to achieve closed reduction [7].

29.3 Clinical Presentation and Essential Physical Examination

The evaluation begins with the history, including the mechanism of injury and any other associated injuries. An associated head injury significantly affects the management of an elbow dislocation [4]. Once it is ascertained that there are no other life or limb threatening injuries, the physical exam begins with inspection of the elbow. One should look for swelling, gross deformity, ecchymosis, or open injuries. Acutely, patients with an elbow dislocation will have deformity and soft tissue swelling at the elbow. It is important to ask about symptoms of pain, paresthesias, and weakness.

A good neurovascular examination is key and should be documented both before and after reduction. Injuries to the brachial artery and nerves are rare findings but must be identified if present [7]. Additionally, it is important to evaluate muscular compartments and pain with passive muscle stretch, to avoid missing a possible compartment syndrome.

Once the elbow has been evaluated, attention is turned to the wrist and shoulder. Concomitant upper extremity injuries occur in 20–25 % of patients [1]. When examining the wrist and forearm, tenderness of the interosseous membrane or distal radius and ulna should increase the suspicion of concomitant injury, specifically an Essex-Lopresti injury [1]. Appropriate forearm, wrist, and shoulder radiographs should be obtained based on symptoms and exam findings.

A torn LCL is responsible for the majority of persistent or recurrent elbow dislocation or subluxation after an elbow dislocation.

29.4 Essential Radiology

Standard AP and lateral radiographs of the injury are needed to determine the direction of dislocation and any associated fractures [4, 7]. After elbow reduction, AP, lateral, and oblique radiographs should be taken to evaluate reduction and associated fractures [2].

The most common fractures associated with elbow dislocations involve the radial head (5 %), coronoid process (10 %), and avulsion fractures of the medial or lateral epicondyles (12 %). Pre-reduction and post-reduction radiographs reveal periarticular fractures in 12–60 % of elbow dislocations; however, osteochondral injuries are

recognized in nearly 100 % of elbows explored surgically [1].

Repeat radiographs should be taken within the first week and again during the second week to confirm reduction during conservative management, as unstable elbows can dislocate even when in a well-placed splint. CT scans can be helpful to evaluate complex fracture patterns, but typically are not necessary for simple dislocations [2].

MRI is not necessary in the early evaluation, as acute ligamentous injury can be diagnosed clinically. However, it may be useful to evaluate the integrity of the interosseous membrane for patients with concurrent forearm and wrist pain. It also can be helpful to look for pathology in chronically unstable elbows [4].

Radiographs of the elbow under a valgus load can be helpful in determining the extent of MCL injury. Patients with an MCL injury are more likely to have persistent valgus instability and subsequently worse outcome in the future. Therefore, identifying these patients early and treating them with a brace in slight varus may help prevent valgus instability [8].

29.5 Disease-Specific Clinical and Arthroscopic Pathology

Simple dislocations requiring operative treatment typically present in one of two ways: either with persistent ligamentous instability after attempted closed reduction or inability to obtain a concentric reduction. Persistent ligamentous instability is often seen in obese individuals, where the weight of the forearm overcomes bony congruency and causes redislocation. Operative exploration in this situation typically reveals rupture of both the MCL and LCL, as well as the extensor and flexor-pronator origins, all of which require repair. Temporary external fixation is often required for 3–4 weeks as well.

If interposed tissue causes inability to obtain a concentric reduction, operative exploration to relieve the tissue block is required (Fig. 29.1). Interpositions of the brachialis, biceps, ulnar nerve, median nerve, and extensor or flexor origin have all been described [2, 7, 9, 10].

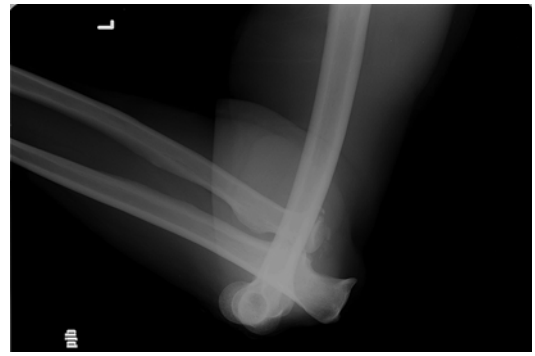


Fig. 29.1 Lateral radiograph demonstrating an irreducible posterior simple elbow dislocation

Arthroscopy is not typically performed for simple elbow dislocations, as the pathology needs to be addressed in an open fashion.

29.6 Treatment Options

After physical exam, including documentation of the neurovascular status, and injury radiographs have been obtained, closed reduction should be immediately attempted. Although ideally performed in the operating room, most closed reductions occur in the emergency room. In either setting, the patient should be given IV sedation with a short-acting benzodiazepine and a short-acting narcotic. Sedation allows muscle relaxation, preventing a traumatic reduction [2]. A palpable or audible "clunk" on reduction is a good indicator that the joint will be stable [1].

29.6.1 Reduction Maneuvers

29.6.1.1 Posterior or Posterolateral Dislocations

The patient should be supine with the forearm supinated and elbow in 20–30° of flexion. Supination protects the coronoid from fracture during reduction. Medial or lateral displacement should be corrected first. With an assistant holding countertraction on the upper arm, gentle traction and further flexion should be applied to the forearm to reduce the olecranon distally around

the trochlea [2, 7]. The olecranon can be pushed distally to aid the reduction [4]. Hyperextension of the elbow is to be avoided, as this can cause neurovascular entrapment [9].

Reduction can also be attempted in a prone position by extending the elbow with countertraction on the arm, with another hand guiding the coronoid over the trochlea [5].

29.6.1.2 Anterior Dislocation

This is a more difficult reduction, which is better managed in the operating room. After medial or lateral displacement has been corrected, the elbow should be flexed and supinated. With an assistant providing counterpressure on the upper arm, the surgeon should push the forearm posteriorly to bring the olecranon proximally around the trochlea [2].

29.6.1.3 Divergent Dislocations

These injuries are associated with high-energy mechanism and are very unstable. Reduction should be performed in the operating room. The reduction can be performed by reducing the radius and then the ulna to the distal humerus, or the radius and ulna can be reduced and then the forearm reduced to the humerus [4].

After successful reduction, the elbow should be taken through a full range of motion while the patient is still sedated. It is important to note the degree of flexion when the elbow begins to subluxate, as this will be important in post-reduction management. According to Hildebrand, an elbow is stable if it stays reduced from at least 60° of flexion to full flexion [4]. The elbow should also be tested for varus and valgus instability with the elbow in full extension and 30° of flexion, which unlocks the olecranon from the olecranon fossa. Valgus stress is measured with the humerus in full external rotation and a valgus force applied to the forearm, while varus stress is measured with the humerus in internal rotation and a varus force applied to the forearm [7]. Usually the elbow is unstable to a valgus stress [5].

If the elbow is stable to flexion and extension, varus and valgus, the next step is to evaluate the forearm for stability in supination and pronation. With a torn LCL, the elbow is more stable in supination, and an elbow with a torn MCL is more stable in pronation. However, most elbow dislocations involve tears of both the LCL and MCL and therefore should be immobilized in a plaster splint with the forearm in a neutral position and the elbow in 90° of flexion [10].

Theoretically, after reducing a simple elbow dislocation, the joint should retain inherent stability from the contours of the joint surfaces. The elbow should be immobilized for 5–10 days as splinting for more than 3 weeks has been associated with loss of motion [1]. A well-padded posterior plaster splint should be applied with the elbow in 90° of flexion, and forearm rotation in whichever position provides the most stability, usually neutral [4].

Post-reduction radiographs should be performed and evaluated for a concentric joint on both AP and lateral views. If there is a widened joint space, there may be an osteochondral fragment trapped in the joint requiring surgical treatment or there may be persistent instability requiring bracing [1, 7].

Surgical vs. nonoperative treatment depends on the stability of the elbow after reduction. In rare situations open reduction is required in a simple elbow dislocation for irreducible dislocations from entrapped soft tissue or osteochondral fragments [7]. Most elbow dislocations are stable after closed reduction. An elbow that subluxes or dislocates at 45–60° of flexion is unstable and requires surgical intervention as rehabilitation with greater than 60° of extension block is difficult and has a high incidence of flexion contracture. Primary ligament repair in an acute unstable elbow is generally successful because it provides stability to allow early rehabilitation and range of motion.

Recurrent instability in simple elbow dislocations occurs in less than 1–2 % of cases [1]. If an elbow redislocates after reduction and splint immobilization, nonsurgical treatment will most likely fail [10]. An unstable simple elbow dislocation is most likely to have an injured MCL, LCL, and anterior capsule as well as injury to secondary elbow stabilizers with no associated fractures. The amount of soft tissue injury to the flexor-pronator and extensor origins is correlated

with the instability of the elbow and likelihood of the elbow to redislocate [1]. McKee et al. evaluated the lateral ligamentous disruption in 61 unstable elbow dislocations and found that 100 % of cases had a LCL and 66 % had concurrent rupture of the common extensor origin [11]. This supports the theory that damage to the secondary stabilizers on the lateral side of the elbow during an acute dislocation results in recurrent instability. Josefsson et al. prospectively compared treatment of simple elbow dislocations by randomizing 30 patients to nonsurgical or surgical treatment with identical postoperative rehab. They found no difference in outcome at 3–7 years in regard to range of motion, grip, and elbow strength. There were no recurrent dislocations or episodes of instability in either group [12].

29.6.2 Nonoperative Treatment

Nonoperative treatment of simple and stable elbow dislocations includes early splinting or sling for 3–5 days, followed by early range of motion. If the elbow has instability past 60°, a hinged brace with an extension block can be used. Strengthening can begin at 6–8 weeks, but weight through the elbow joint such as pushups and overhead press should be avoided for 3 months [2]. Prolonged immobilization is not recommended, because it can lead to long-term loss of motion [13]. Splinting for more than 3 weeks results in worse patient outcome [14]. Even with early range of motion, patients lose 10–20° of terminal extension [15, 16].

29.6.3 Operative Treatment

For simple unstable elbow dislocations treated in the acute setting, treatment consists of ligament repair. The posterolateral elbow dislocation involves sequential tears of the LCL, anterior capsule, and MCL. For stabilization of these injuries, the LCL should be reconstructed first. After LCL reconstruction, the elbow should be tested for stability. If the elbow is stable through full range of motion, it is not necessary to repair the MCL [17, 18]. Repair of the MCL is not routinely needed for stabilization of an elbow after dislocation. Leaving the MCL unrepaired is advantageous because there is less operative dissection and no need to transpose the ulnar nerve [8].

The patient should be positioned supine with a radiolucent arm table. Standard fluoroscopy via mini-fluoroscopy should be used. If the surgeon would like to use the large C-arm, the image intensifier (larger side) should be used as the operating table.

The incision is made through either a midline posterior approach or a Kocher posterolateral approach. The advantage of a midline posterior approach is that it allows access to the medial elbow if an MCL repair is needed [2].

Kocher approach: Full-thickness skin and subcutaneous flaps are made and elevated laterally. The intermuscular interval is between the anconeus and extensor carpi ulnaris (ECU.) The common extensor origin is often avulsed off the lateral epicondyle (50 % of the time), which helps with the exposure of the LCL. At this point, the torn LCL should be identified. The ligament is usually avulsed from the humeral origin, and there is little tissue left behind from its insertion point on the humerus [11].

The LCL and the extensor origin should be repaired primarily. This can be done with suture anchors or bone tunnels with #0 or #1 braided nonabsorbable suture. Bone tunnels are created at the humeral and ulnar attachments of the lateral UCL. Isometry should be evaluated to ensure constant tension through full range of motion [7]. In the acute setting, primary repair of the avulsed tendons can usually be performed (Fig. 29.2). However, in the rare case when ligament reconstruction is needed, the palmaris longus can be used as a graft through bone tunnels in the humerus and proximal ulna [2]. If the disruption of the LCL is intrasubstance, then direct repair can be performed with large, braided nonabsorbable sutures [7].

After the LCL has been repaired, the elbow should be tested for stability through a full range of motion. If the elbow is stable, no further surgical repairs are needed. However, if the elbow is

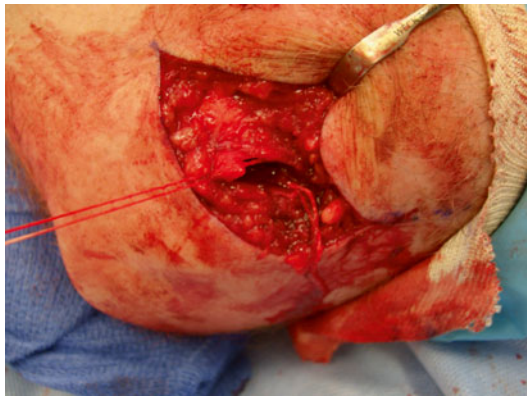


Fig. 29.2 Intraoperative photograph of LCL repair with a suture anchor for fixation of an unstable simple elbow dislocation

still unstable, then the MCL will need to be repaired. This is performed through a medial approach. The flexor carpi ulnaris (FCU) is identified and either elevated from the distal humerus or split longitudinally, while the ulnar nerve is found and protected. The avulsed MCL is identified and repaired using the same techniques as the LCL repair. This is done with either suture anchors or bone tunnels and nonabsorbable sutures from the medial epicondyle to supracondylar ridge. The FCU should then be repaired back to its origin. If the flexor-pronator muss is avulsed this should also be repaired [2]. Care should be taken to avoid the ulnar nerve on the medial aspect of the elbow.

After MCL and LCL reconstruction, the elbow should again be tested for stability through a full range of motion. If the elbow is still unstable, it will need additional stabilization with either a hinged or static external fixator or transarticular pins. The hinged external fixator allows for range of motion and does not violate the articular cartilage. Hinged fixation allows early range of motion while assuring reduction of the elbow joint [7]. Alternately, static fixation is used. Transarticular pinning is only indicated in patients with multiple medical comorbidities who cannot tolerate prolonged anesthesia.

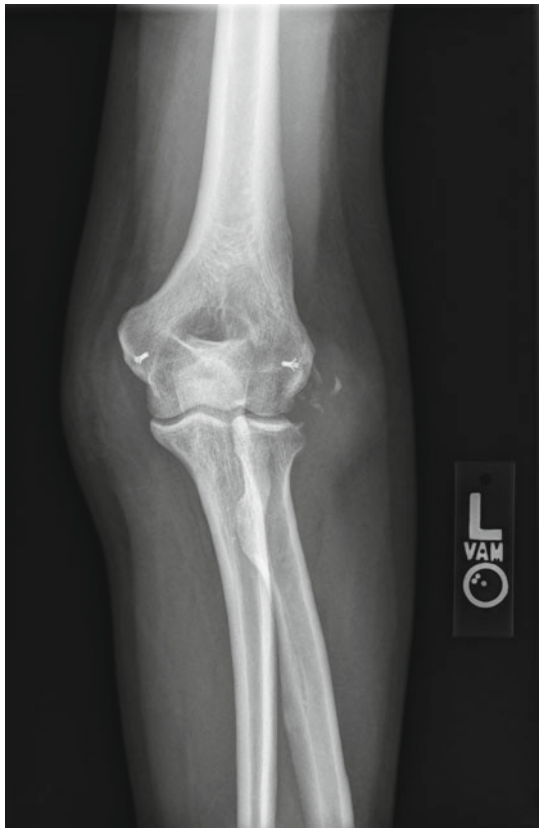
Proper application of an external fixator is contingent on a well-placed axis pin. The axis pin is placed in the center of the trochlea parallel to the joint [2]. The fixator is assembled around the axis pin. Once the fixator has been assembled, the axis pin is removed. When placing humeral pins, 1–1.5 cm incisions should be made with blunt dissection down to the bone to avoid the radial nerve. While assembling the external fixator, live fluoroscopy can be used to access the reduction [2].

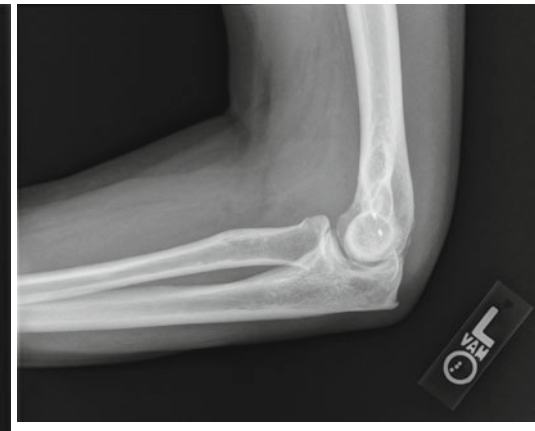
Micic et al. evaluated 20 acute unstable simple elbow dislocations that underwent surgical treatment. All ligament injuries were proximal avulsions except for two partial injuries to the MCL. Extensor tendons avulsed with collateral ligaments, while some flexor tendons tore at the musculotendinous junction [14].

29.7 Authors' Preferred Treatment

A posterolateral incision is made and the interval between anconeus and ECU is split. The lateral epicondyle and lateral aspect of the elbow joint are exposed. Any interposed soft tissue is then removed from the elbow joint. Once this soft tissue has been removed, the joint should be concentrically reduced. At this point in time, attention is turned to repair of the LCL. The avulsed LCL is repaired to its isometric point on the lateral humeral epicondyle with a suture anchor. Location of suture anchor should recreate isometric tension on the LCL throughout full elbow range of motion. If the extensor origin is also avulsed, as is typical, this is also repaired. After the LCL is repaired, the stability of the elbow is tested. If the elbow is stable through a full range of motion without posterior or posterolateral instability, MCL repair is not necessary.

If the elbow remains unstable, a medial incision is made for repair of the MCL. The FCU is elevated from the proximal humerus if needed, but usually one will note it also is avulsed from the humeral origin, while the ulnar nerve is protected. The proximally avulsed MCL is repaired to the medial humeral epicondyle with a suture anchor placing the MCL at its isometric point, and the FCU is repaired back to its origin. The elbow is again tested for stability. If the elbow is stable, the surgical procedure is complete (Figs. 29.3 and 29.4).





Figs. 29.3 and 29.4 Postoperative radiographs demonstrating concentric reduction after suture anchor repair of LCL and MCL for treatment of an unstable simple elbow dislocation

If the elbow is still unstable through full range of motion after LCL and MCL repair, then a static external fixator is placed and fluoroscopic concentric reduction is confirmed. The static external fixator remains on for 3 weeks and is then removed to begin range of motion.

29.8 Rehabilitation

Early range of motion is critical for elbow dislocations treated both operatively and nonoperatively, to prevent stiffness. The elbow should be immobilized after an elbow dislocation and reduction for 3–10 days. Unstable elbow dislocations are repaired surgically to allow for early motion. Supervised early range of motion with a hinged brace with or without an extension block should begin within 2 weeks from injury. If an extension block is used, it should be set at 5° from the point at which the elbow becomes unstable, and extension should increase gradually as stability increases [7]. Early motion does not result in redislocation or late instability of the elbow [16].

Early motion is critical in restoration of range of motion and prevention of contracture. Multiple studies have evaluated duration of immobilization, noting that early elbow range of motion is associated with better motion, fewer flexion contractures, less pain, less valgus deformity, and earlier return to work [13]. Patients immobilized for more than 3 weeks are more likely to develop contractures [16]. Because studies have indicated that outcomes are superior when motion is started earlier, active range of motion should begin as soon as possible, given the patient's stability and symptoms.

Rehabilitation is also important for patients with minimal residual elbow subluxation following nonoperative or operative treatment. Duckworth et al. found that active flexion exercises and avoidance of varus stress resulted in stability at 2 year followup in 23 patients with mild radiographic subluxation [19]. Patients with residual motion deficits in 2 months will require another intervention. Patients will continue to improve up to 18 months.

Early active motion within the first few weeks helps prevent arthrofibrosis. If the elbow does develop stiffness, dynamic elbow splints and patient-adjusted progressive static splints should be tried if motion has not improved by 4–6 weeks [1].

Flexion returns before extension, with the maximum amount of flexion returning at 6–12 weeks. Extension can take up to 3–5 months to return [1]. Pronation and supination are usually not affected in simple elbow dislocations.

It is important to obtain follow-up radiographs 3–5 days and again at 2 weeks post reduction to ensure continued concentric reduction of the joint. Extension blocks, if used, should be discontinued by 4–6 weeks. Use of a hinged brace should be discontinued at 6 weeks for patients who underwent ligamentous repairs and at 2 months for those undergoing ligamentous reconstruction [7].

29.9 Advantages/Pitfalls/Complications

Most patients who suffer simple elbow dislocations regain a functional arc of motion and report good to excellent results in 75–100 % of cases. Simple elbow dislocations have better results than those associated with fractures [1].

The most common complication following a simple elbow dislocation is loss of terminal extension, averaging 10–20°; however, the average loss of motion in those immobilized for less than a week is less than 10° [7, 12, 13]. Elbows that require surgical treatment often have more soft tissue injury and usually have a greater loss of motion.

Residual instability is very rare; however, symptoms of instability have been reported in up to 35 % of patients. Most commonly this is valgus instability, and these patients tend to have a worse outcome [7].

Heterotopic ossification is seen in up to 55 % of patients, although it usually occurs in the collateral ligaments and is asymptomatic. Most follow-up studies have not shown a correlation between presence of heterotopic ossification and loss of motion [4, 7]. Bridging heterotopic ossification is very rarely seen (less than 5 %) in patients who undergo surgical treatment of an unstable elbow [2]. Subsequently, prophylaxis for heterotopic ossification is not indicated for patients with simple elbow dislocations. The authors noted that patients with residual motion deficits at 2 months required further intervention [7].

Neuropraxia of the ulnar nerve is the most common neurological injury associated with elbow dislocations. Symptoms usually resolve with elbow reduction; however, 10 % of patients have persistent symptoms [2, 13]. Median nerve injuries are rare, but can occur from stretch injuries or entrapment of the nerve in the joint. If paresthesia begins post reduction and there is a widening of the joint, further investigation and treatment are mandated [7].

Brachial artery injuries are rarely seen with elbow dislocations and are associated with higher-energy injuries such as open injures and severe fracture-dislocations. They are not seen with simple elbow dislocations; however, one must be careful to not entrap the brachial artery in the joint during reduction of an anterior dislocation.

Essex-Lopresti injuries can occur with elbow dislocations, so it is important to always evaluate the ipsilateral wrist and interosseous membrane for tenderness to palpation and instability. If distal radial ulnar joint (DRUJ) injury is suspected, it should be worked up and treated appropriately [2].

The force required to dislocate the elbow causes more than soft tissue damage even if no fractures are appreciated on radiographs. Osteochondral injury is common, and long-term functional status is affected by the degree of osteochondral injury that occurred at the time of dislocation [7].

Although most patients with simple elbow dislocations do well, a minority have residual instability requiring operative ligament reconstruction and external fixation [4].

29.10 Experience in Treatment of Athletes

Forty percent of all elbow dislocations occur during sports. Gymnastics, wrestling, basketball, and football are the sports most commonly associated with elbow dislocation [5].

In a study evaluating upper extremity injuries in the NFL by Carlisle et al., 58 % of injuries were to the elbow, and 5 % of the injuries to the upper extremity were elbow dislocations. Offensive and defensive lineman had the highest incidence of upper extremity injury and 75 % of their injuries were to the elbow. Those players with joint instability lost a mean of 18 days to injury [20].

Early mobilization after a simple dislocation, while protecting the elbow from potential instability events, is the key to management in athletes. The athlete is typically allowed to begin early range of motion but without resistance. Sometimes this requires hinged bracing for compliance.

Operative fixation and rehabilitation in athletes does not differ from the standard regiment described above.

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Valgus Extension Overload Syndrome

30

Jin-Young PARK and Seok Won Chung

30.1 Introduction

Valgus extension overload (VEO) syndrome is a condition that causes problems and pains within the elbow and is commonly seen in overhead athletes due to overuse and repetitive throwing forces [1]. This condition develops over time rather than with one particular throw or event and leads to progressive changes within the elbow joint causing pain and athletic impairment. These changes typically present with medial elbow laxity and the posteromedial impingement by the diffuse osseous changes within the elbow joint. Andrews and Timmerman [1] reported the posteromedial olecranon impingement to be the most common diagnosis (78 %) requiring surgery in baseball players.

In recent years, the number of participants in overhead throwing sports has been rapidly increased, which in turn brought a concurrent increase in the VEO syndrome. These injuries most commonly occur in baseball pitchers; however, athletes who participate in other sports with similar mechanics of overhead motion of excessive valgus and extension forces, such as tennis, badminton, volleyball, softball, or swimmers, can be likewise affected. Without appropriate diagnosis and treatment, VEO syndrome can be a career-threatening injury for an overhead athlete.

In caring for athletes who participate in overhead throwing sports, the physician must have the ability to accurately diagnose and appropriately treat the VEO syndrome. A thorough understanding of the pathophysiology involved in overhead activities is essential to the recognition, diagnosis, and treatment of these specific elbow injuries.

Recent advances in the treatment of VEO syndrome based on the understanding of the pathomechanism have led to the successful return of most injured overhead athletes to competitive activities [2]. In this chapter, we will discuss the current knowledge regarding the diagnosis and treatment of VEO syndrome, especially focusing on the posteromedial impingement (pure VEO) rather than the ulnar collateral ligament. The issues of ulnar collateral ligament injuries were addressed on the previous chapter.

30.2 Pathophysiology

The elbow joint is a hinge joint, and the bony ulnohumeral articulation provides stability at the extremes of motion. In the middle range, approximately 20–100° of flexion, the anterior bundle of

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the ulnar collateral ligament is the primary restraint to valgus stress, and tensile stresses approaching the failure point are generated during the acceleration phase of high-velocity throwing. The elbow is subjected to tremendous valgus and extension stresses during the throwing motion. Fleisig et al. [3] demonstrated that the valgus forces have been estimated as high as 64 Nm at the elbow during the late cocking and early acceleration phases of throwing with compressive forces of 500 N at the lateral radiocapitellar joint in their biomechanical study. The combination of large valgus loads with rapid elbow extension produces tensile stress along the medial compartment and shear stress in the posterior compartment, and compression stress in the lateral compartment. Repetitive valgus stress applied during throwing results in microtrauma and inflammation of the anterior band of ulnar collateral ligament and may lead to eventual ligament attenuation or insufficiency. Injury to the ulnar collateral ligament can precipitate or exacerbate VEO due to increased loading of the articular surfaces. Any increased laxity or injury to the ulnar collateral ligament will create compensatory increase in compression on the medial aspect of the olecranon and olecranon fossa articulation as the elbow is forcibly extended. In this condition, continued valgus and extension forces may produce olecranon osteophytes at its posteromedial tip and articular damage in the olecranon fossa and posteromedial trochlea caused by the olecranon osteophyte (kissing lesion). The posteromedial compartment lesions including olecranon osteophytes and loose bodies have been reported as the most common diagnoses that require surgery in baseball players as a result of repetitive valgus extension overload forces [4] (Fig. 30.1).

Surgeons who treat the cases of elbow pain by the impingement of posteromedial compartment in throwing athletes should consider the underlying valgus laxity resulting from the injury of ulnar collateral ligament as an underlying cause. Overhead throwing athletes, especially those who performed with sudden and forceful elbow extension with improper throwing mechanics and with poor physical conditioning of strength and flexibility will have increased risk of disease progression of VEO.

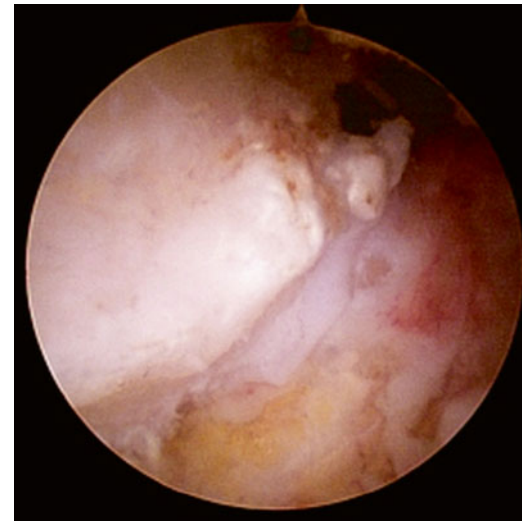

Fig. 30.1 Arthroscopic image viewed from the posterior aspect of the elbow which shows olecranon osteophytes produced by excessive valgus and extension forces

30.3 Clinical Presentation and Essential Physical Examination

The diagnosis of VEO is based on the athlete's thorough history, physical examination, and radiographic studies. Throwers with VEO typically complain of pain at the tip of the elbow in the deceleration and follow-through phases of throwing and inability to throw at full speed and loss of ball control. This is different from the throwers only with medial instability symptoms, who experience pain at the medial side during the acceleration phase of throwing. Throwers with VEO will notice a sharp posterior pain exacerbated by forced extension or even a snapping or locking sensation as they release the ball. This pain typically increases over time and only manifests with throwing and not with other activities of daily living. Occasionally, there is associated stress on the ulnar nerve, which is vulnerable to the same stresses and overload that lead to VEO. This can present as numbness or tingling in the ring and little fingers, with clumsiness and weakness in gripping.

Physical examination begins with careful inspection. Any swelling of the elbow, change of

the carrying angle (increased valgus), or loss of normal extension should be inspected. However, elbow flexion contracture has been seen in up to 50 % of professional throwers and should not be considered indicative of injury [5]. Palpation of the posterior aspect of the elbow is an important aspect of the physical examination of the thrower's elbow. Tenderness on the olecranon tip and in the olecranon fossa in full extension suggests the presence of VEO. On the other hand, tenderness in the posterior region that is more proximal or distal to the olecranon tip may present in triceps tendinitis or olecranon stress fracture, respectively. The end-feel to range of motion test is also important in examining thrower's elbow. The normal end-feel in extension should be the firm sensation, and the end-feel in flexion should be that of soft tissue. If a throwing athlete has a bony end-feel in terminal flexion, a bony osteophyte or loose bodies should be considered, and if a soft end-feel in extension, a soft tissue contracture should be considered [4].

We typically perform the extension jerk test (valgus extension overload test) and the extension impingement test to diagnose VEO. The extension jerk test is performed with the patient seated and the shoulder in slight forward flexion. The examiner repeatedly forces the slightly flexed elbow rapidly into full extension while applying a valgus stress. This maneuver attempts to reproduce pain with impingement of the posteromedial tip of the olecranon on the medial wall of the olecranon fossa. A positive finding often indicates the presence of a posteromedial olecranon osteophyte, which may occasionally be palpable at the time of physical examination or inflammation around olecranon fossa. In addition, the extension impingement test is performed by applying continuous extension forces to the elbow. If the pain occurs at the posterior aspect of elbow and it reproduces the pain or symptoms they experience during throwing, the posterior impingement by the olecranon tip osteophytes can be considered.

In addition, evaluation of elbow medial stability could be performed to check if there is any laxity or injury in the ulnar collateral ligament. Specific test for medial stability includes the moving valgus stress test and the milking maneuver. The moving valgus stress test is the test which the examiner applies a constant valgus force to the elbow and then quickly extends the elbow starting with the arm in full flexion. The milking maneuver is performed by having the patient reach under his injured arm with the opposite hand and grab the thumb of the injured arm. Continued pulling will place a valgus stress on the elbow under examination with palpating the ulnar collateral ligament in approximately 60° of flexion. Reproduction of painful symptoms with an apprehension-like response during the test suggests a problem in the ulnar collateral ligament. In such a case, the staged treatment for the insufficient ulnar collateral ligament such as ligament reconstruction can be considered.

Careful examination of the other susceptible structures of the elbow should be performed. Specifically, palpation of the ulnar nerve, the ulnar collateral ligament, the distal medial triceps, the flexor-pronator muscle mass, the radial head, and the capitellum should be conducted to ensure that these structures are not involved in the process. The ulnar nerve can also be the source of posteromedial pain within the cubital tunnel. The examiner should ensure that the ulnar nerve is stable within the cubital tunnel throughout the range of motion and that no ulnar nerve-distribution symptoms are present.

30.4 Essential Radiology

Standard plain radiographs of the thrower's elbow includes AP, lateral, axial, and two oblique views of the affected side. An oblique axial view with the elbow in 110° of flexion is helpful to demonstrate posteromedial olecranon osteophytes [6]. The presence of olecranon osteophytes, osteochondral damage, or loose bodies can be visualized on these plain radiographs. However, the absence of osteophytes or loose bodies cannot rule out VEO, as the condition of posteromedial impingement predates the formation of osteophytes. We typically perform three-dimensional CT scan of elbow joint to

evaluate the extent of posteromedial impingement accurately and decide the excision level of the olecranon before surgery if indicated. In addition, the CT scan can be helpful for detecting stress fractures of the olecranon and avulsion fractures of medial epicondyle. In cases where the injury of ulnar collateral ligament is suspicious, MRI can be obtained to evaluate the status of ulnar collateral ligament and other pathology within the elbow. The attenuation of the ulnar collateral ligament may accompany the VEO.

30.5 Disease-Specific Arthroscopic Pathology

The arthroscopic pathology of VEO syndrome includes the presence of posteromedial olecranon osteophyte, chondromalacia, osteochondral damage, or loose bodies. During arthroscopic treatment, the olecranon osteophyte should be resected, loose bodies should be removed, and unstable cartilage flaps or cartilage defects may be debrided.

30.6 Treatment Options

Initial treatment consists of active rest and anti-inflammatory medication to relieve the pain and modification of the activity that initially caused the problem. Active rest includes discontinuing throwing and avoiding any exercise that causes discomfort. Strengthening exercise to increase the flexor-pronator strength and gradual return to throwing through an interval throwing program will be allowed as symptoms resolve. For those with long duration and multiple episodes of symptoms, a more prolonged period of rest followed by a more gradual interval throwing program may be indicated.

If conservative management is not effective in relieving the symptoms or if there is locking and catching due to loose bone fragments within the joint, surgical management is recommended. Surgery is performed to remove bone spurs, bone fragments, and loose cartilage.

30.7 Authors' Preferred Treatment

The most important consideration in surgical intervention is identifying those individuals whose ulnar collateral ligament also may require surgical treatment to reconstruct the damaged ligament. For these individuals, we typically reconstruct the ulnar collateral ligament using contralateral palmaris longus tendon autograft at 2 weeks after arthroscopic spur resection for the posteromedial impingement. The issue of ulnar collateral ligament reconstruction in overhead athletes has been addressed in the previous chapter.

In surgical management, elbow arthroscopy has replaced open arthrotomy treatment in the treatment of VEO syndrome in the throwing athlete and has been shown to have good results with low complication rates [7, 8]. The arthroscopic procedure for the treatment of VEO syndrome, especially posteromedial impingement, is as follows.

After appropriate anesthesia, arthroscopic surgery may be performed with the patient in the supine, prone, or lateral decubitus position. We prefer the prone position with the arm in 90° of abduction and the elbow in 90° of flexion after general anesthesia. A tourniquet is routinely used and a pressure-sensitive arthroscopic pump is helpful to allow adequate visualization and prevent overdistension of the elbow. Initially, all bony landmarks and ulnar nerve are marked with a methylene blue pen (Fig. 30.2), and the elbow is distended with 20–30 ml of saline, injected through the soft spot of the elbow joint. The distension of elbow will facilitate the introduction of instruments and the shift of the neurovascular structures away from the penetrating instruments. A detailed knowledge of the elbow anatomy is critical for proper portal placement which is the first step for the successful arthroscopic treatment. We typically establish proximal medial portal to check any pathology at the anterior compartment in advance. Anterolateral portal is then established by using a spinal needle to assist with proper placement. The anterior compartment is thoroughly evaluated for loose bodies; evidence

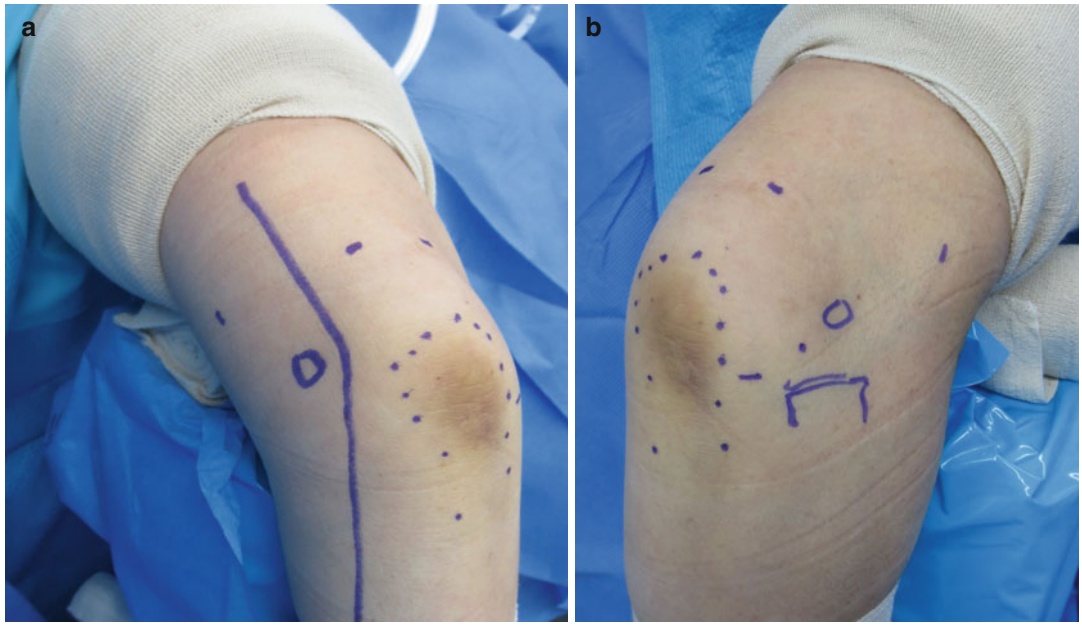

Fig. 30.2 Preparation for the elbow arthroscopy in a prone position. (**a**) Medial view, (**b**) lateral view. All bony landmarks and ulnar nerve are marked with methylene blue pen before surgery

of chondral damage to the coronoid process, capitellum, or radial head; or osteophyte formation in the coronoid fossa. Loose bodies from the posterior or lateral compartments often migrate to the anterior compartment or vice versa; therefore, all compartments must be thoroughly evaluated. A lateral soft spot portal is then established for the arthroscope at the site of initial elbow injection, which is the center of a triangle formed by the radial head, lateral epicondyle, and olecranon tip. A second posterolateral portal may be placed approximately 1 cm proximal to the first lateral portal for instrumentation of the lateral compartment or removal of loose bodies. In addition, a posteromedial portal and a transtriceps portal are established under spinal needle localization. This posteromedial portal is typically made as close to the medial border of the distal triceps as possible to avoid inadvertent injury to the nearby ulnar nerve. Through these portals, a shaver is introduced to debride areas affected by synovitis and soft tissues from the olecranon tip and olecranon fossa so that the entire bony margin of the olecranon tip can be visualized and loose bodies may be identified. The kissing lesion is also debrided. The olecranon osteophyte is then excised. We typically use an arthroscopic burr to remove the posteromedial tip of the olecranon. The amount of olecranon osteophyte that can safely be excised is not established yet; however, up to 8 mm of the olecranon may be resected safely without increase in strain on the ulnar collateral ligament by valgus stress [9]. We remove only enough bone to allow full elbow extension without bony impingement, approximately 3–5 mm; the excision level of the olecranon is decided preoperatively by the measurement using the CT scans (Figs. 30.3 and 30.4). Once the resection is completed and no further intra-articular pathology remains, the arthroscopic instruments are removed, and the portals are closed. A compressive dressing is applied, and the arm is iced and elevated postoperatively. No immobilization device is needed.

30.8 Rehabilitation

Postoperative rehabilitation is begun early to maintain range of motion as well as to strengthen the elbow gradually. For the first 2 weeks, the

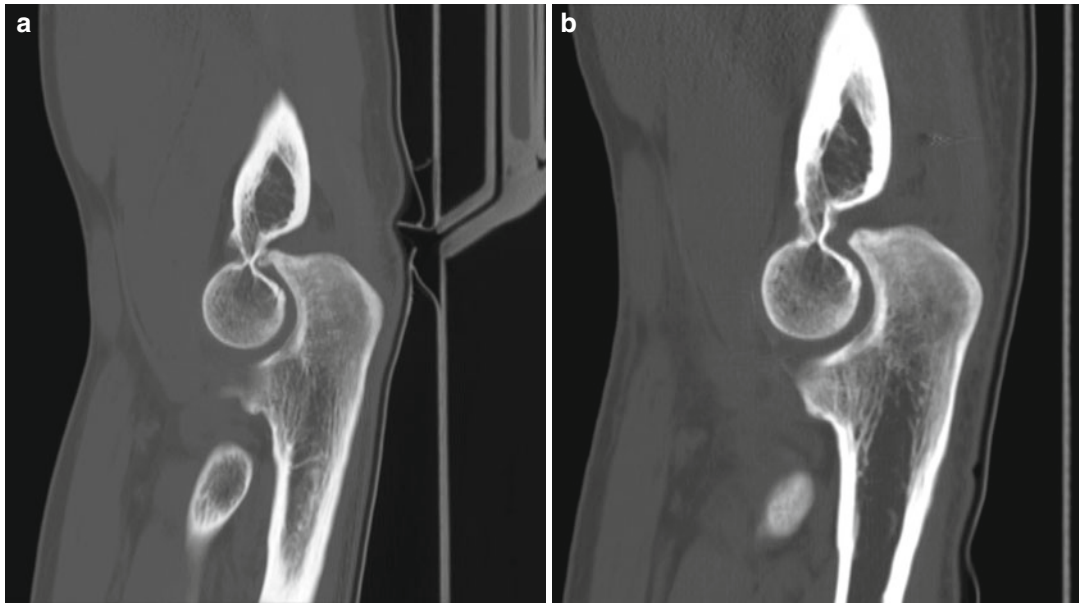

Fig. 30.3 Sagittal view of the elbow CT scans. Before (**a**) and after (**b**) spur resection of posteromedial olecranon tip

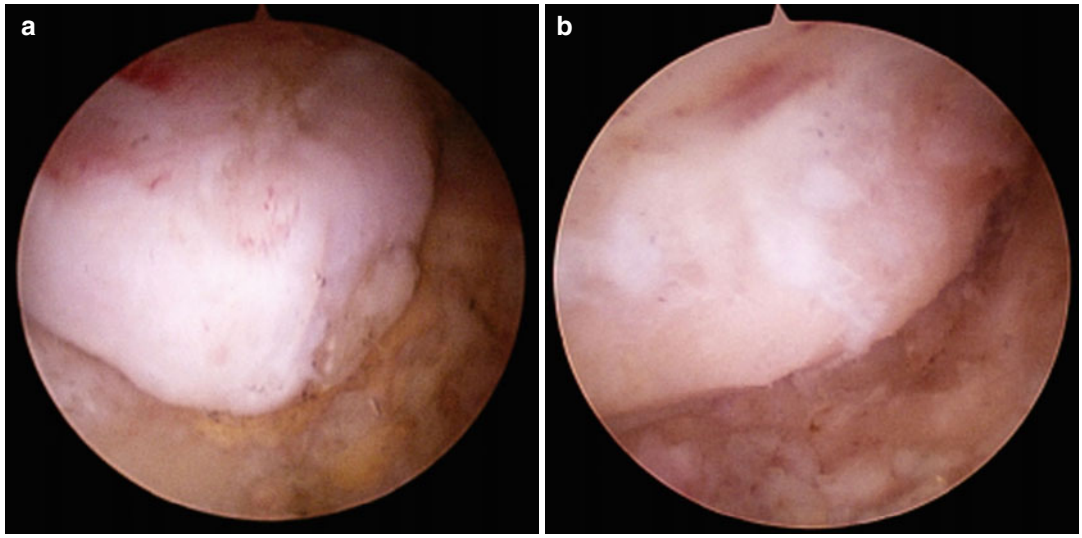

Fig. 30.4 Arthroscopic images viewed from the posterior aspect of the elbow. Before (**a**) and after (**b**) spur resection of posteromedial olecranon tip

recovery from arthroscopic resection is centered on edema control and regaining range of motion. We permit gentle range of motion exercise from the first day postoperatively. Generally, range of motion will be back to normal around 2 weeks. The throwing motion is restricted for 3 months postoperatively, and during this period, the muscle-strengthening exercise is performed. At 3 months, the interval throwing program is started for the athletes. Completion of the interval throwing program may take several weeks to several months depending on the level of competition

and the ability of the individual athlete. Generally, the time to return to sports will be approximately 4–6 months after surgery for the athletes undergoing uncomplicated arthroscopic spur resection for the treatment of VEO [6].

30.9 Advantages, Pitfalls, and Complications

Results after arthroscopic treatment of VEO have shown to be satisfactory, even though it has been reported for many different applications including posteromedial impingement, loose body removal, capsular release, osteochondritis dissecans, and degenerative joint diseases. Since Andrew and Carson [10] showed good results after treating elbow pathology by arthroscopy in 1985, many authors have reported similar good results with high return to play [7, 8]. Reddy et al. [7] reported 87 % of good to excellent results in 187 arthroscopic treatments (posterior impingement in 51 %, loose bodies in 31 %, and degenerative joint disease in 22 %) and showed that 47/55 baseball players (85 %) were able to return to the same level of competition. Complications are quite rare; however, the ulnar collateral ligament failure by the increased strain after olecranon osteophyte resection or recurrence of posteromedial impingement is reported [9].

30.10 Experience in Treatment of Athletes

The careful treatment of VEO has lead to successful return to competitive throwing or overhead sports. When we analyzed 41 baseball players who had undergone arthroscopic spur resection due to pure VEO syndrome (mean age, 20.7 year; mean follow-up, 30.2 months), all patients except 1 (40/41, 97.6 %) were successively returned to sports with the average time for return being 3.8 months. Pain VAS score was decreased from 6.4 to 1.0 point, and subjective satisfaction was mostly excellent (20 patients; 48.8 %) or good (20; 48.8 %). Modified elbow scoring system showed excellent outcome 39 patients (95.1 %) and good outcome 2 patients (4.9 %).

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Rehabilitation: Part I. Basic

31

Jin-Young PARK and Kyung-Soo Oh

31.1 Before Starting the Exercise

31.1.1 Stage of Recovery After Shoulder Surgery

Generally recovery after shoulder surgery follows the stages below:

1. Immobilization
2. Range of motion
3. Strengthening
4. Return to previous level of activity

Trainers should help the patients with the following objectives:

1. To recover range of motion
2. To improve muscle power in all directions
3. To reeducate normal movement patterns and timing of muscle activity/control
4. To recover proprioception
5. To return back to normal life, sports, and recreation activity

31.1.2 Shoulder Exercise Intensity

This book indicated level of strength for all exercises. It would help the patients and the trainers.

★	Level 1	<20 % strength
★	Level 2	21–40 % strength
★	Level 3	>40 % strength

31.1.3 Equipments for the Exercise

The following equipments will be needed for exercises introduced in this book:

1. 65 cm gym ball, for patients taller than 170 cm
2. 55 cm gym ball, for patients shorter than 170 cm
3. Rubber bands of three different strengths (for muscle exercise)
4. Exercise bar
5. Pulley and strap (cord)

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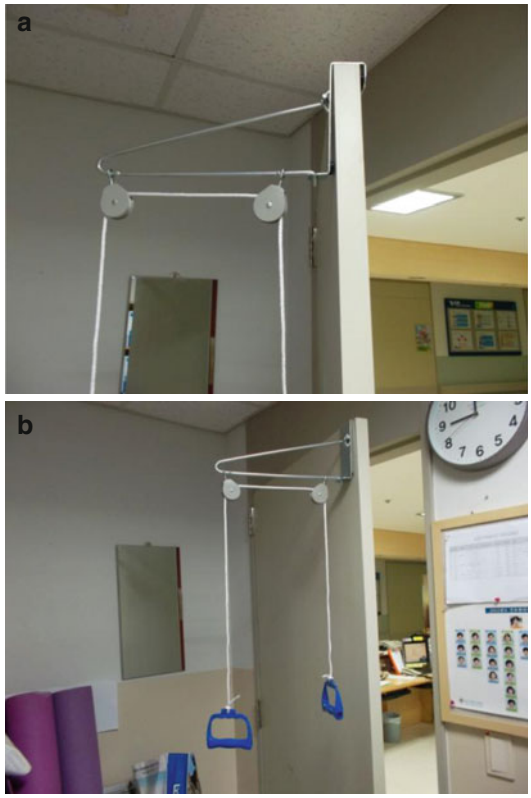


Fig. 31.1 Pulley

Fig. 31.2 Exercise bar

31.1.4 Equipment Installation/ Instructions

1. Pulley
   Install on a door (Fig. 31.1).
2. Exercise bar
   Assemble the separated bar (Fig. 31.2).
3. Use a rubber band by fixing it to a door handle or to your foot by stepping on it (Fig. 31.3).

31.2 Shoulder Exercise Book

31.2.1 Range of Motion Exercise

Exercise for recovery of ROM is made with simple level 1 exercises. Progress after the following:

31.2.1.1 Pendular Exercise

Bend forward and place the uninjured arm on the table for a support. The injured arm should be outside the table toward the ground (Fig. 31.4a). Move your injured arm back and forth to make a small circle (Fig. 31.4b).

31.2.1.2 Forward Flexion with the Opposite Hand

The palm of the injured extremity should be facing your head (Fig. 31.5a). Using the opposite hand, grab the wrist of the injured extremity and move toward your face (Fig. 31.5c) (120°). Never exert your injured extremity, only make effort on the opposite arm. When returning to the initial position, make sure no effort is done on the injured arm.

31.2.1.3 Pulley Exercise

Prepare the exercise on a sitting position holding the pulley. The palm of the injured extremity should face you (Fig. 31.6a), and always exercise with the opposite arm to lift the injured arm till possible range is achieved (Fig. 31.6c). The

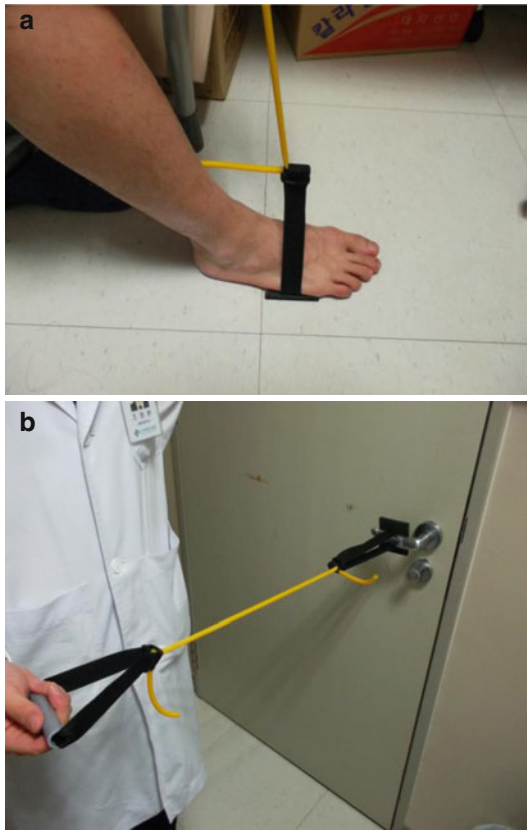


Fig. 31.3 Rubber band

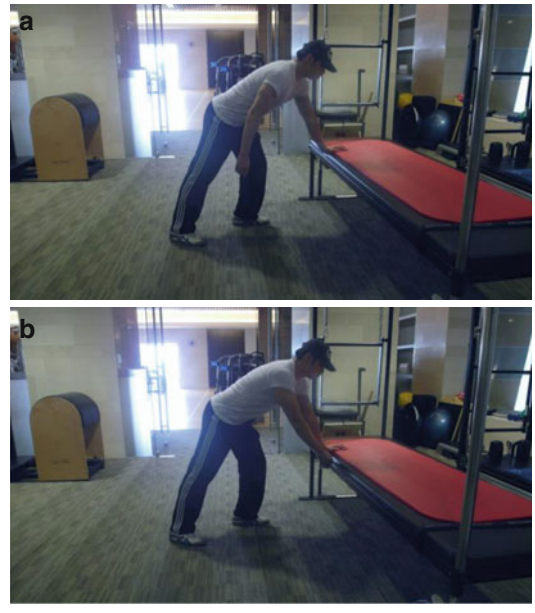


Fig. 31.4 Pendular exercise

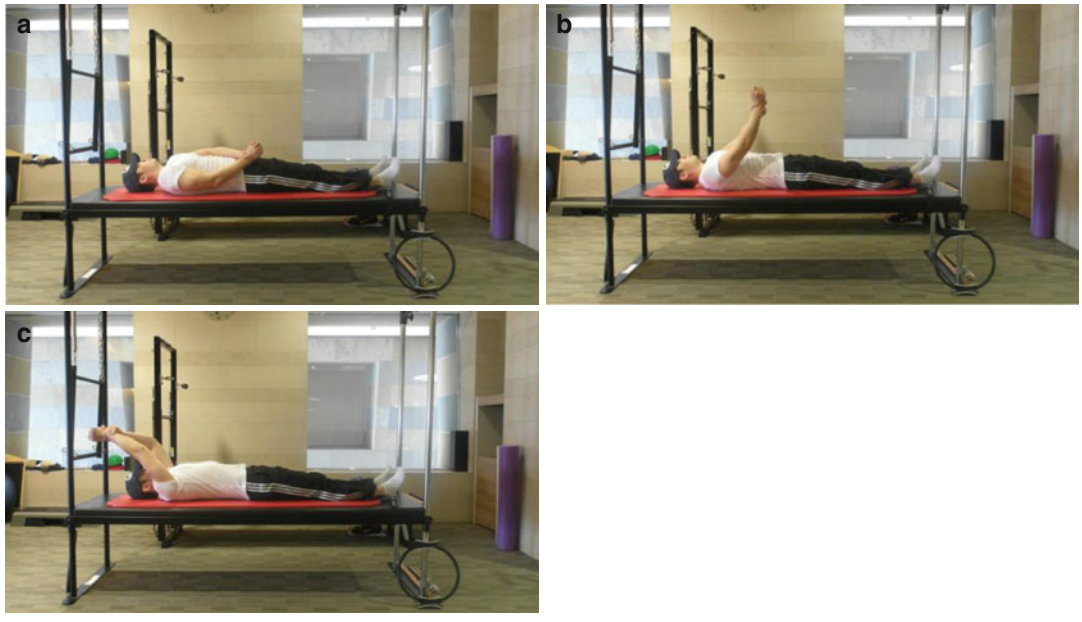


Fig. 31.5 Forward flexion with the opposite hand

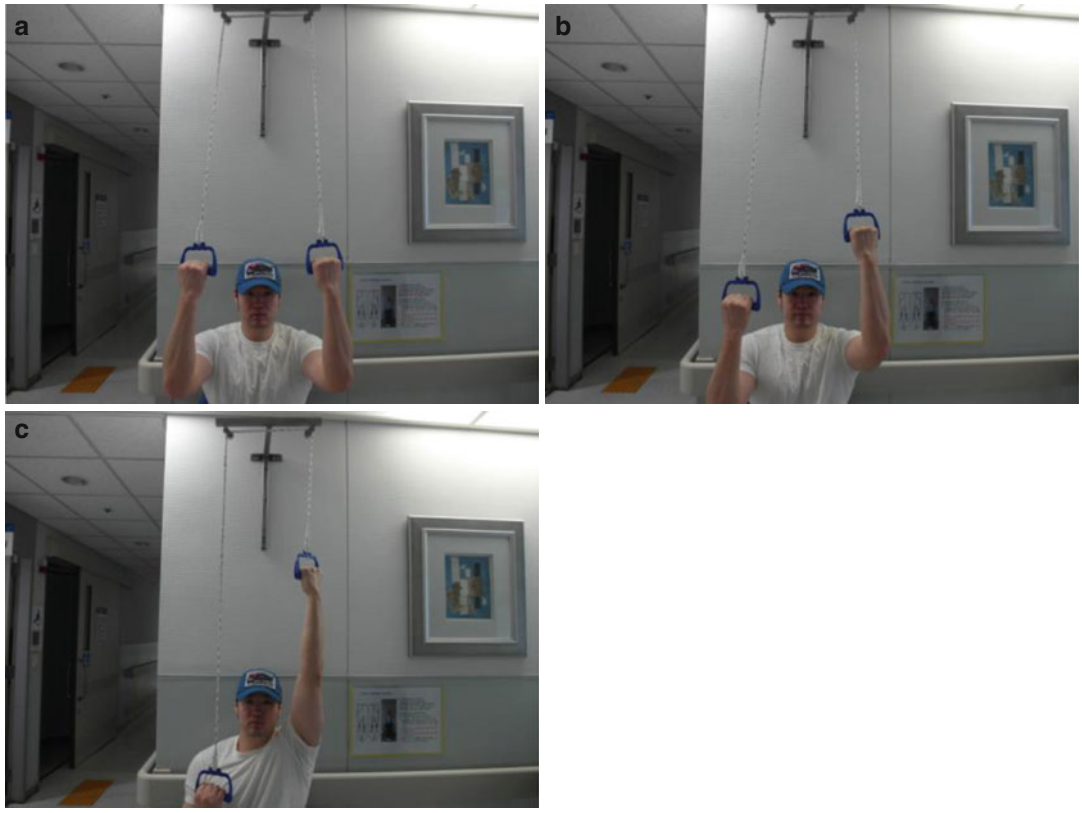


Fig. 31.6 Pulley exercise

injured arm should never be exerted during the exercise. When coming down, the opposite arm should lead the way down.

31.2.1.4 Flexion on the Table

Sitting in front of a table, place both your hands on the table (Fig. 31.7a). By sliding your hands forward, bend down on the table to increase range of motion (Fig. 31.7b).

31.2.1.5 Abduction on the Table

Sitting beside a table, place your hand of injured extremity on the table (Fig. 31.8a). Push your hand laterally away from your body (Fig. 31.8b).

31.2.1.6 Forward Flexion with Exercise Bar

Prepare by lying down on the floor, holding the exercise bar with your shoulder width (Fig. 31.9a). Lift your arm over your head. During the exercise, extend your elbow as much as is possible (Fig. 31.9c, d).

31.2.1.7 Internal and External Rotation: Shoulder in Neutral with Exercise Bar

Prepare by lying down on the floor, holding the exercise bar with your shoulder width (Fig. 31.10a). Elbows should be flexed and maximally rotate both ways (Fig. 31.10b).

31.2.1.8 Internal and External Rotation: Shoulder in 90° with Exercise Bar

Prepare by lying down on the floor, holding the exercise bar with your shoulder width (Fig. 31.11a). Forward flex your shoulders with elbows flexed. Maximally rotate both ways (Fig. 31.11b).

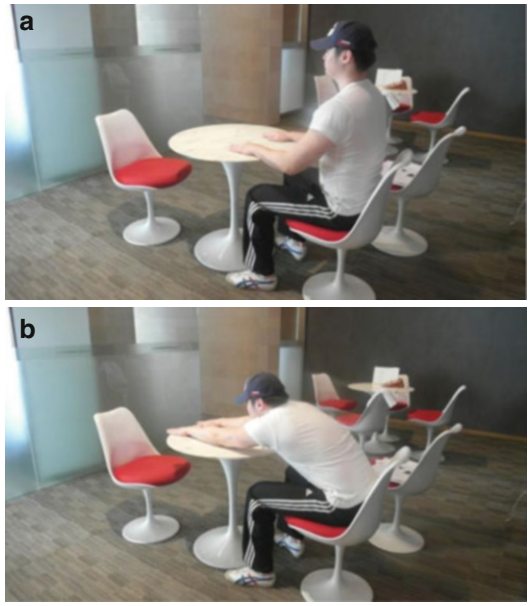


Fig. 31.7 Flexion on the table

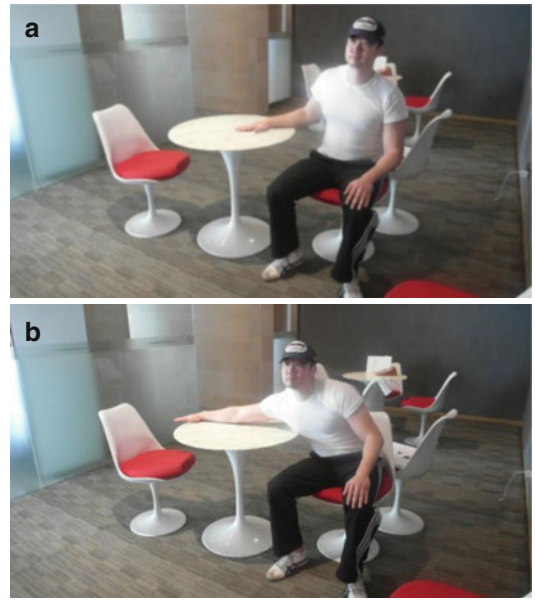


Fig. 31.8 Abduction on the table

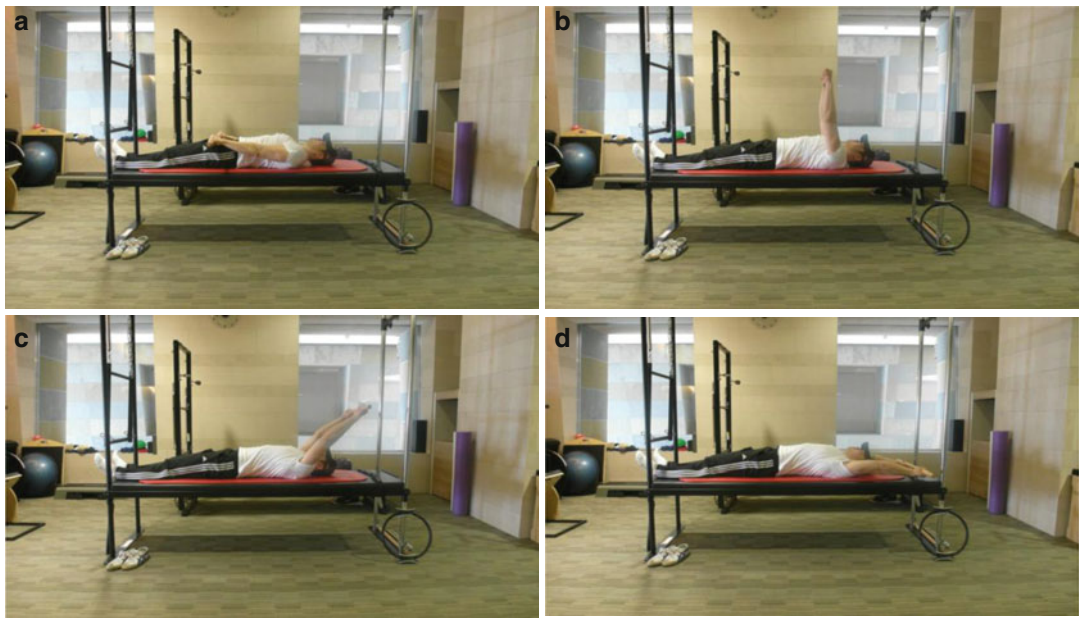


Fig. 31.9 Forward flexion with exercise bar

31.2.1.9 Abduction and Adduction with Exercise Bar

Prepare by lying down on the floor, holding the exercise bar with your shoulder width (Fig. 31.12a). Forward flex your shoulders with elbows extended. Maximally rotate both ways (Fig. 31.12b, c).

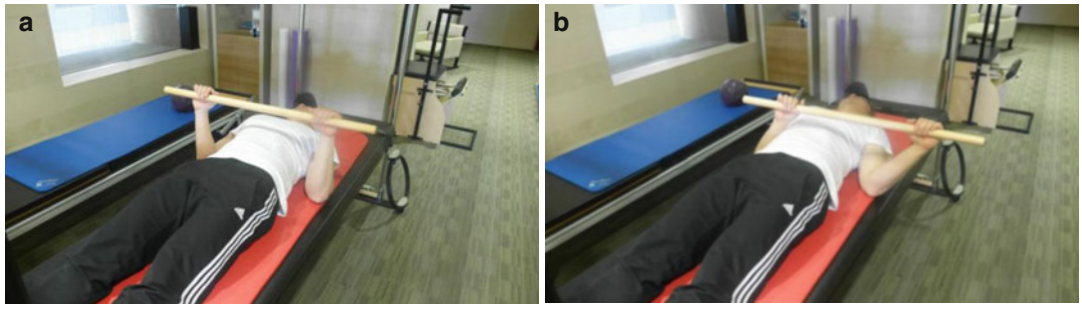


Fig. 31.10 Internal and external rotation: shoulder in neutral with exercise bar

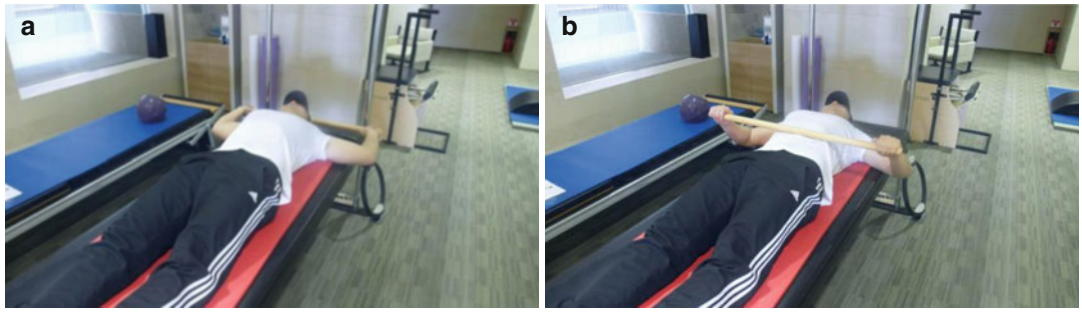


Fig. 31.11 Internal and external rotation: shoulder in 90 degree with exercise bar

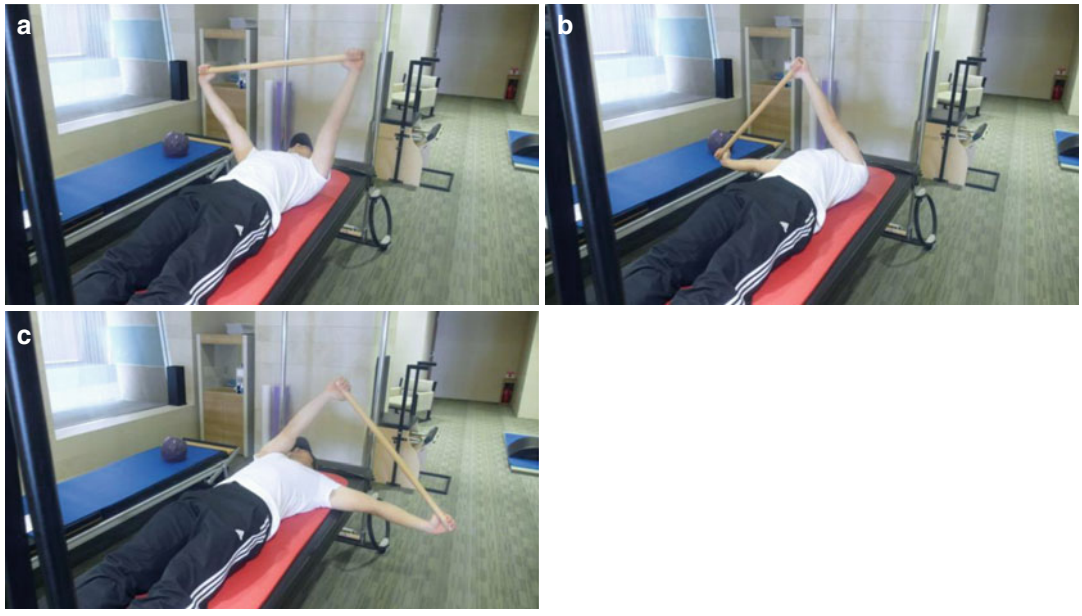
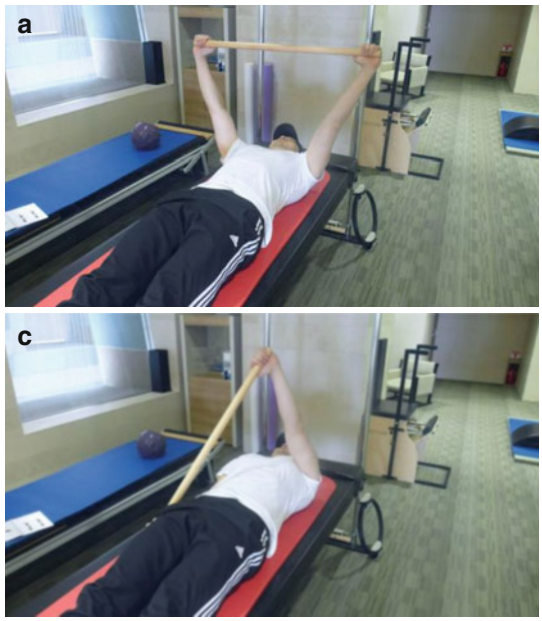


Fig. 31.12 Abduction and adduction with exercise bar

31.2.1.10 Diagonal Side to Side with Exercise Bar

Prepare by lying down on the floor, holding the exercise bar with your shoulder width (Fig. 31.13a). Gently move the bar from the hip (which is on the same side of your injured shoulder) to the opposite shoulder (Fig. 31.13b, c).



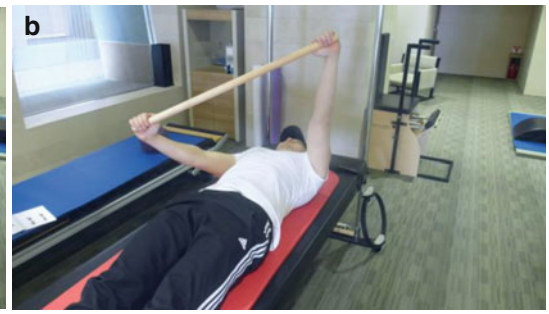
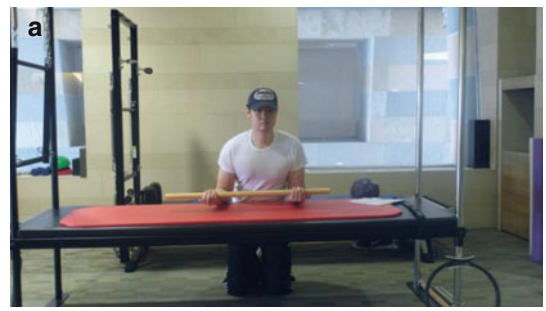


Fig. 31.13 Diagonal side to side with exercise bar

31.2.1.11 External Rotation on the Table

Prepare by sitting down on a table, holding the exercise bar with your shoulder width (Fig. 31.44a). Both palms should face upward. Externally rotate in both ways (Fig. 31.14b).



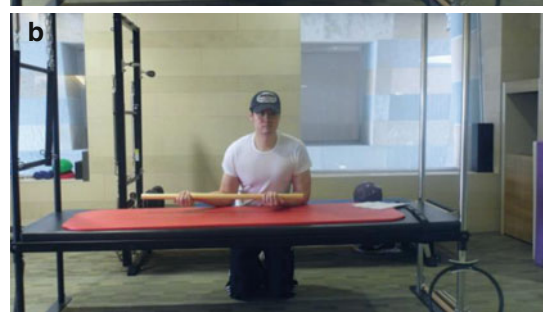


Fig. 31.14 External rotation on the table

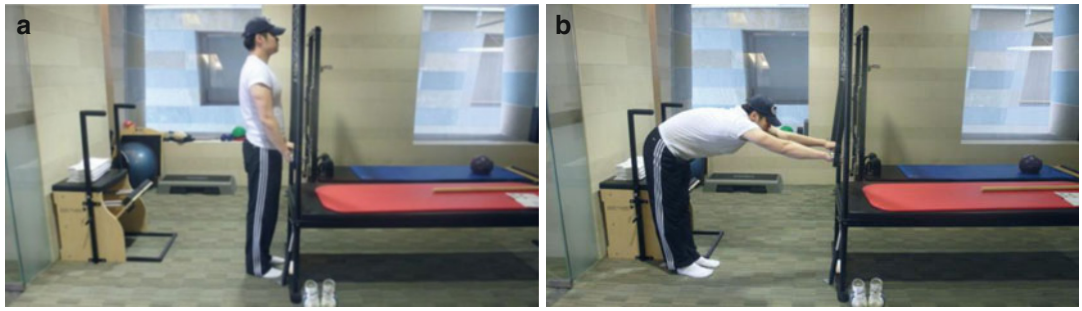


Fig. 31.15 Flexion in standing

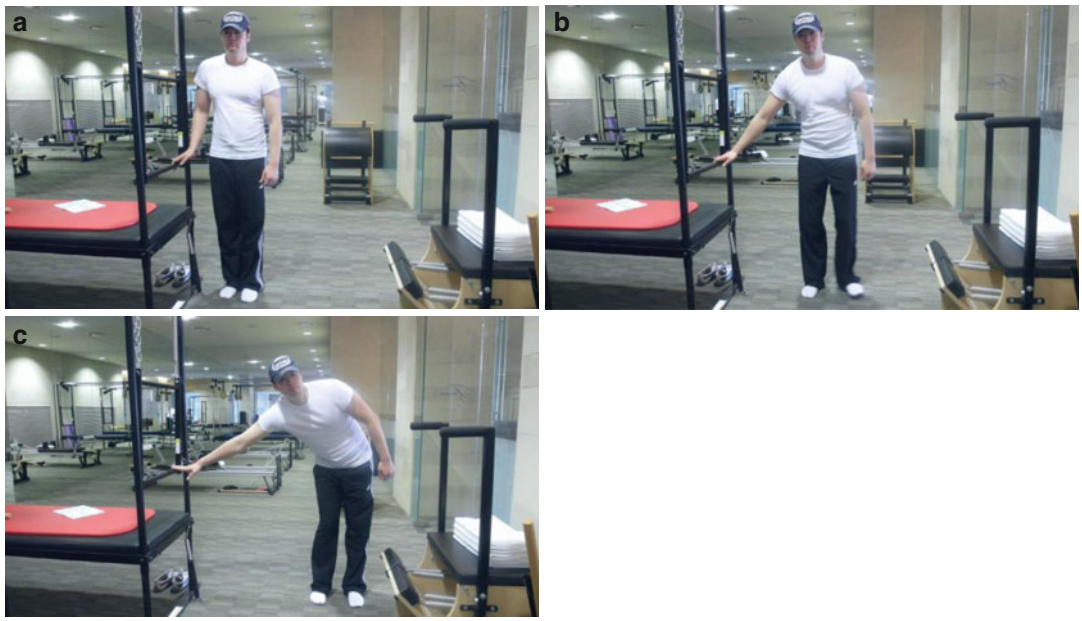


Fig. 31.16 Abduction in standing

31.2.1.12 Flexion in Standing

Standing in front of a table, place both your hands on the table (Fig. 31.15a). Fix your hands on the table and move away from the table with elbows fully extended to flex your shoulders (Fig. 31.15b). Try to stand away from the table as much as you can.

31.2.1.13 Abduction in Standing

Standing beside a table, place your hand of injured extremity on the table (Fig. 31.16a). Fix your hand on the table and push your body laterally away from the table to abduct your shoulder (Fig. 31.16b, c).

31.2.1.14 End-Range External Rotation with Exercise Bar in Lying Position

Prepare by lying down on a bed, holding the exercise bar with your shoulder width. The injured shoulder should be located outside the bed. Using your uninjured extremity, push down

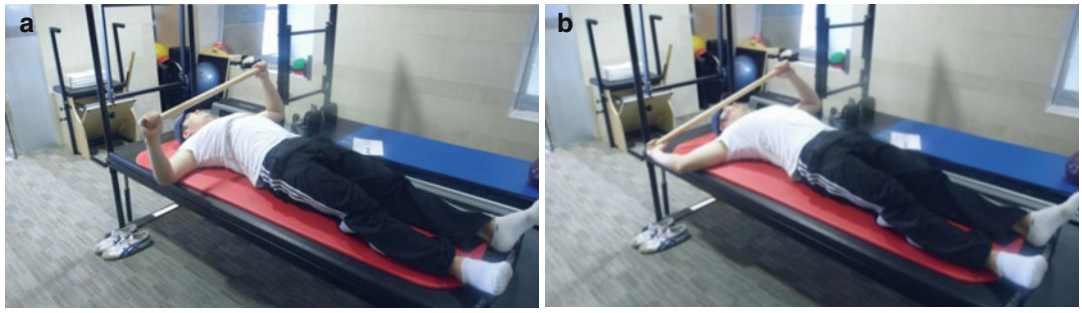


Fig. 31.17 End-range external rotation with exercise bar in lying position

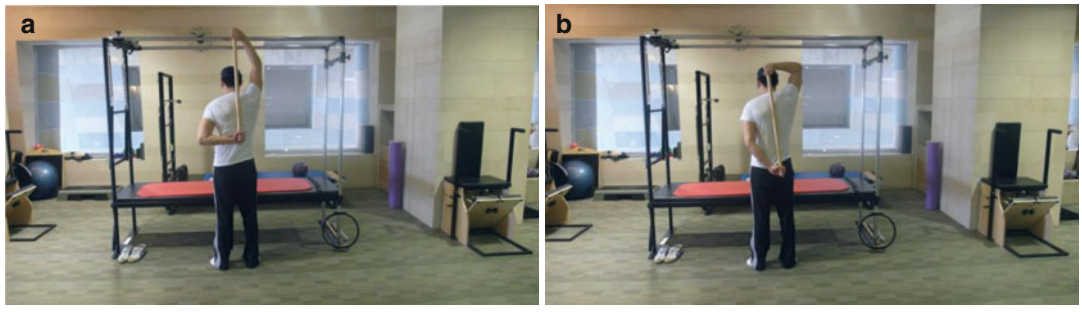


Fig. 31.18 External rotation: stretch with exercise bar in standing position

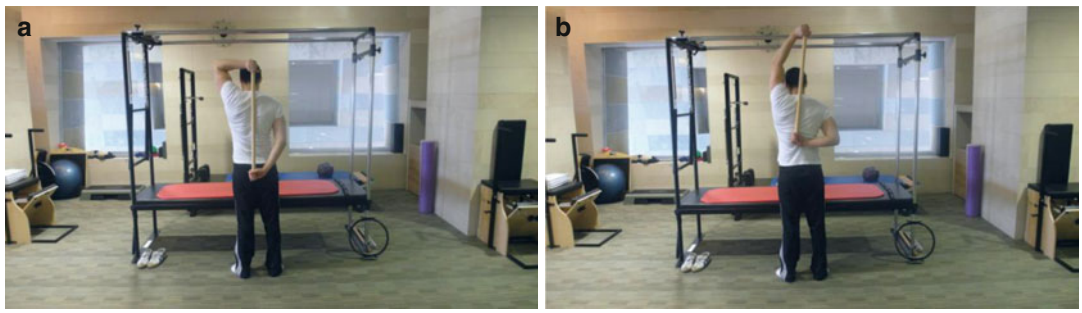


Fig. 31.19 Internal rotation: stretch with exercise bar in standing position

the bar to elevate your injured shoulder. Continue till the back of your hand reaches the floor (Fig. 31.17).

31.2.1.15 External Rotation: Stretch with Exercise Bar in Standing Position

Hold the exercise bar behind your back (Fig. 31.18a). The injured extremity should be placed at the top, and using your uninjured extremity, pull down maximally (Fig. 31.18b).

31.2.1.16 Internal Rotation: Stretch with Exercise Bar in Standing Position

Hold the exercise bar behind your back (Fig. 31.19a). The injured extremity should be placed at the top, and using your uninjured extremity, push upward maximally (Fig. 31.19b).

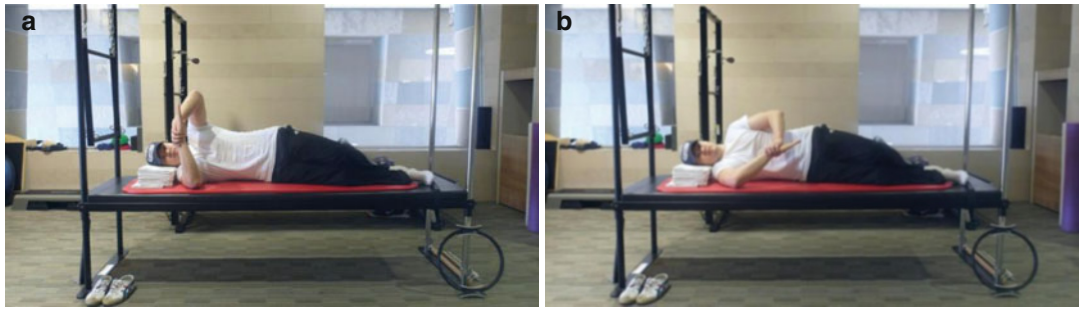


Fig. 31.20 Sleeper stretch

31.2.1.17 Sleeper Stretch

Lie down laterally with the injured shoulder facing the floor. The elbow of the injured extremity must be in level with your shoulder (Fig. 31.20a). Grab the wrist with your opposite hand and push downward toward the floor (Fig. 31.20b). Gently perform for 30 s. After 30 s of exercise, withdraw your opposite hand's power and return to the initial state.

31.2.2 Chapter 2: Proprioceptive Exercise

Proprioception is an information about the present motion state of your body that is told to your brain through nerves in the muscles, ligaments, and articular capsules.

This nervous system controls the amount of muscle stimulation when making a posture or a movement.

When your shoulder is injured, this proprioception does not work well. So the information that is transported to your brain is not accurate, which makes your muscle movement too much or too less.

31.2.2.1 Prayer Position

Kneel down and sit on your heel. Place both hands just outside your knees (Fig. 31.21a). Lean forward, keeping the elbows extended (Fig. 31.21b).

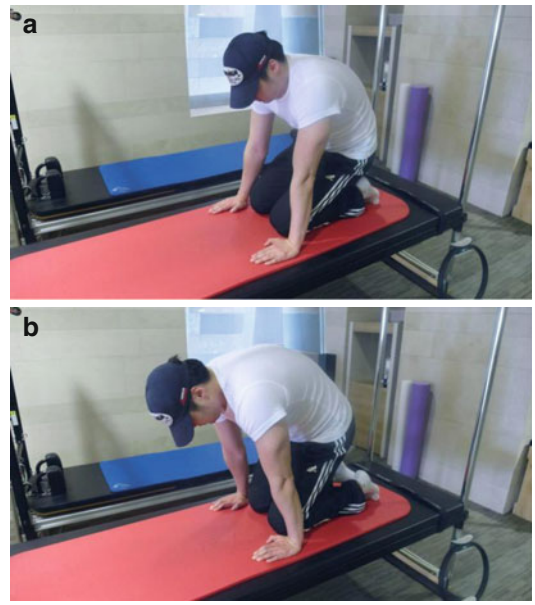


Fig. 31.21 Prayer position

31.2.2.2 Balance Point in Lying Flexion

Prepare by lying down on the floor, holding a lightweight dumbbell with your injured arm. Flex your shoulder 90° (Fig. 31.22a) and move back and forth in a range of 60° (60–120° of movement) with the elbow extended (Fig. 31.22b, c).

31.2.2.3 Balance Point in Lying Abduction

Prepare by lying down laterally on the floor, holding a lightweight dumbbell with your injured arm. Your injured shoulder should be above. Abduct your shoulder 90° (Fig. 31.23a). Push up and pull down in a range of 60° (60–120° of movement) with the elbow extended (Fig. 31.23b, c).

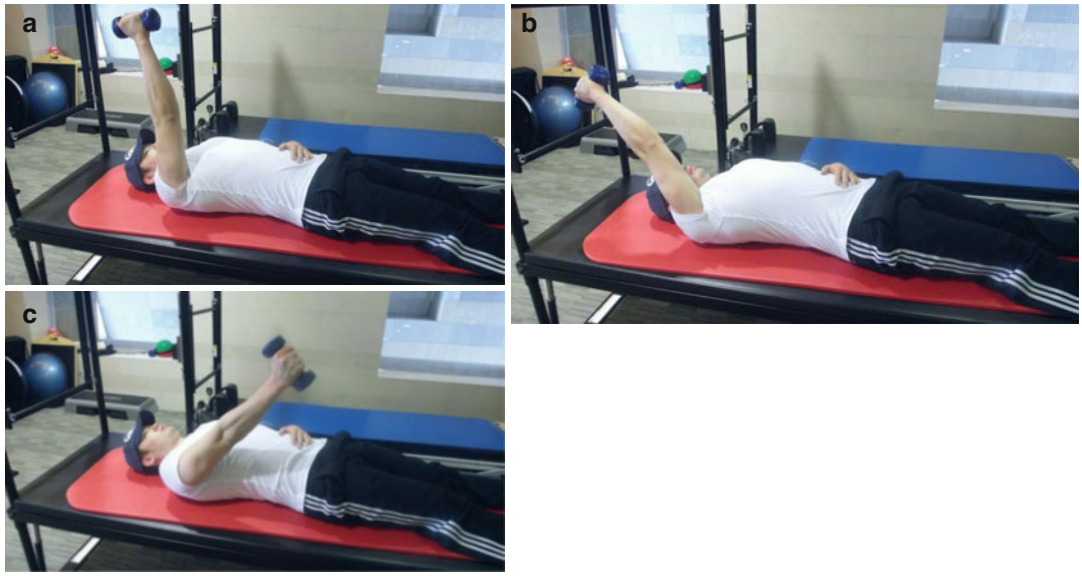


Fig. 31.22 Balance point in lying flexion

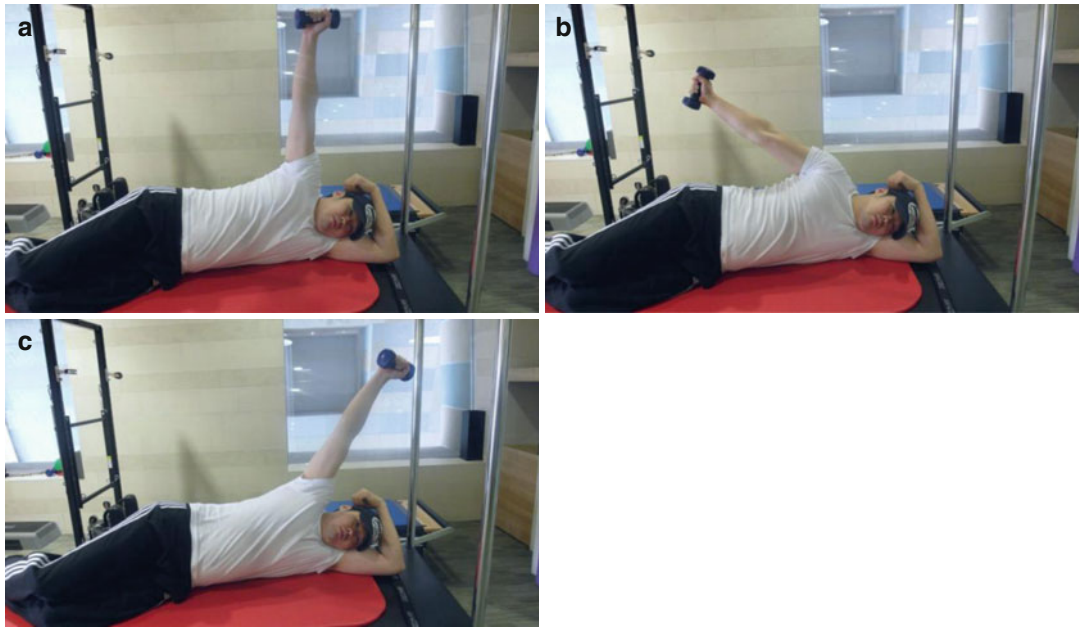


Fig. 31.23 Balance point in lying abduction

31.2.2.4 Point Kneeling over a Physioball

This exercise is using a physioball. Kneel down over the ball. Fix both your hands to the floor with elbows extended. Gently exercise by moving your weight to the injured shoulder (Fig. 31.24).

31.2.2.5 Point Kneeling with Alternate Arm and Leg Elevation

Lie with your face down with both hands and knees fixed to the floor (Fig. 31.25a). Elbows should be extended, and keep your spine straight. Lift up your uninjured arm and your opposite leg

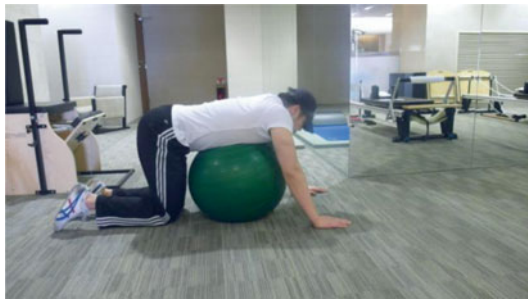


Fig. 31.24 Point kneeling over a physioball

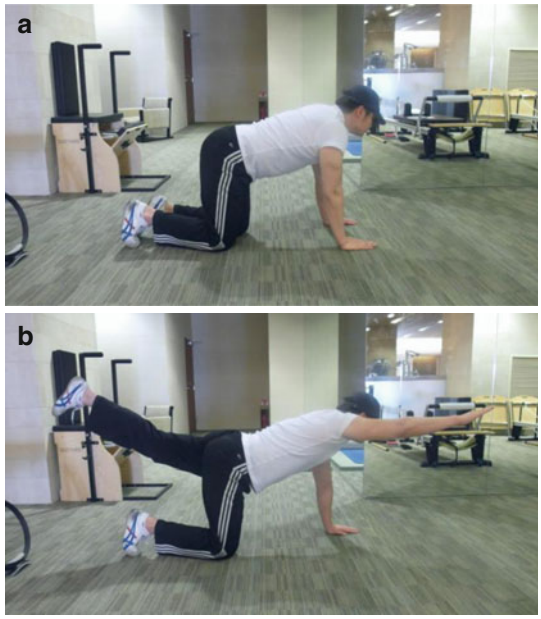


Fig. 31.25 Point kneeling with alternate arm and leg elevation

(Fig. 31.25b). Gently move your weight to your injured arm on the floor.

31.2.3 Chapter 3: Strengthening Exercise

Rehabilitation or strengthening exercise is easy to understand. It is to strengthen the weakened muscle around your shoulder.

31.2.3.1 Isometric Exercises in Sitting External Rotation

Prepare by sitting down on a chair. Hold a towel with your injured shoulder keeping the elbow flexed 90° (Fig. 31.26a). Grab the wrist of your injured shoulder with the opposite hand. Try to externally rotate your injured shoulder with a small amount of resistance (Fig. 31.26b).

31.2.3.2 Isometric Sitting Abduction

Prepare by sitting down on a chair. Hold a towel with your injured shoulder keeping the elbow flexed 90°. Grab the upper arm of your injured shoulder with the opposite hand (Fig. 31.27a). Try to abduct your injured shoulder with a small amount of resistance (Fig. 31.27b).

31.2.3.3 Isometrics Sitting Internal Rotation

Prepare by sitting down on a chair keeping the elbows flexed 90°. Grab the wrist of your injured shoulder with the opposite hand (Fig. 31.28a). Try to internally rotate your injured shoulder with a small amount of resistance (Fig. 31.28b).

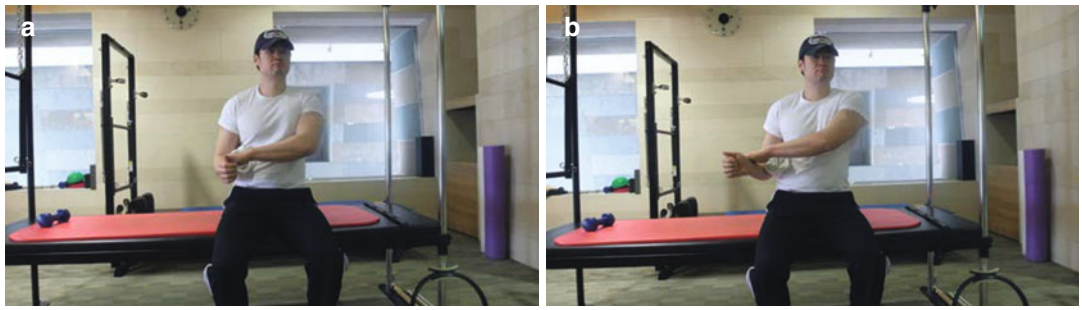


Fig. 31.26 Isometric exercises in sitting external rotation

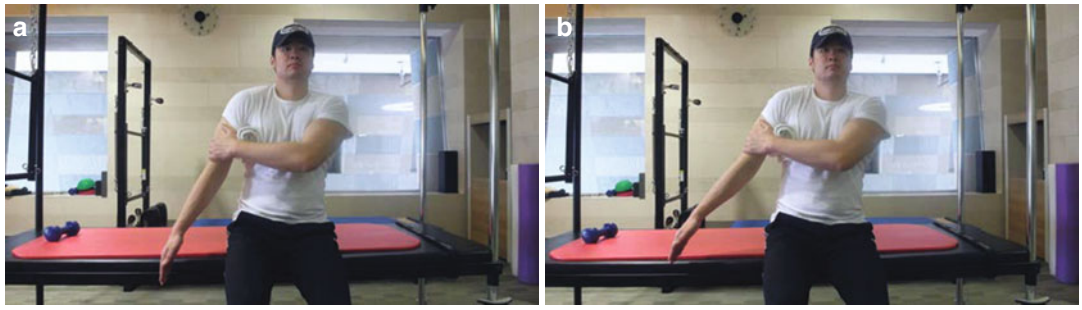


Fig. 31.27 Isometric sitting abduction

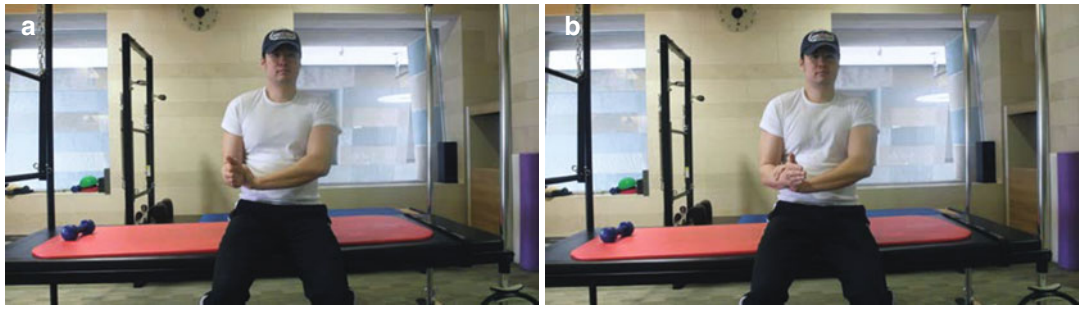


Fig. 31.28 Isometric sitting internal rotation

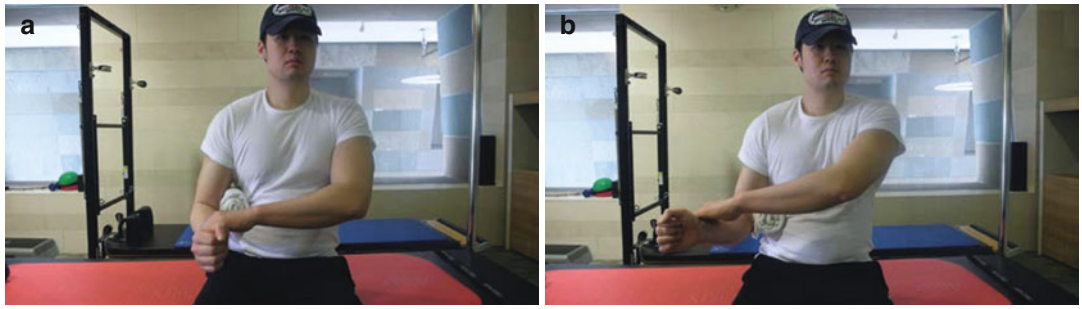


Fig. 31.29 Isometric sitting ER through range

31.2.3.4 Isometrics Sitting ER Through Range

Prepare by sitting down on a chair. Hold a towel with your injured shoulder keeping the elbow flexed 90°. Grab the wrist of your injured shoulder with the opposite hand (Fig. 31.29a). Try to externally rotate your injured shoulder with a small amount of resistance (Fig. 31.29b). Change your external rotation angle and rotate against the resistance.

31.2.3.5 The Exercise Band: Anterior Deltoid Strengthening Exercise

Fix a rubber band to a door handle. Stand with your back toward the wall (Fig. 31.30a). From an elbow flexed position, exercise by pushing the rubber band (Fig. 31.30b).

Fig. 31.30 The exercise band: anterior deltoid strengthening exercise

Fig. 31.31 The exercise band: posterior deltoid strengthening exercise

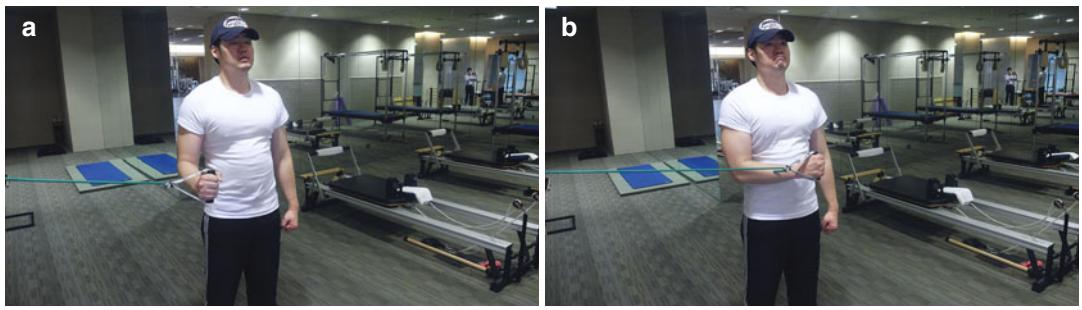


Fig. 31.32 The exercise band: deltoid and subscapularis strengthening exercise

31.2.3.6 The Exercise Band: Posterior Deltoid Strengthening Exercise

Fix a rubber band to a door handle. Stand facing the wall (Fig. 31.31a). From an elbow flexed position, exercise by pulling the rubber band (Fig. 31.31b).

31.2.3.7 The Exercise Band: Deltoid and Subscapularis Strengthening Exercise

Stand with your injured shoulder facing the door (Fig. 31.32a). From an elbow flexed position, exercise by internally rotating your shoulder while holding the rubber band (Fig. 31.32b).

31.2.3.8 The Exercise Band: Deltoid and Teres Minor Strengthening Exercise

Stand with your injured shoulder far from the door (Fig. 31.33a). From an elbow flexed position, exercise by externally rotating your shoulder while holding the rubber band (Fig. 31.33b).

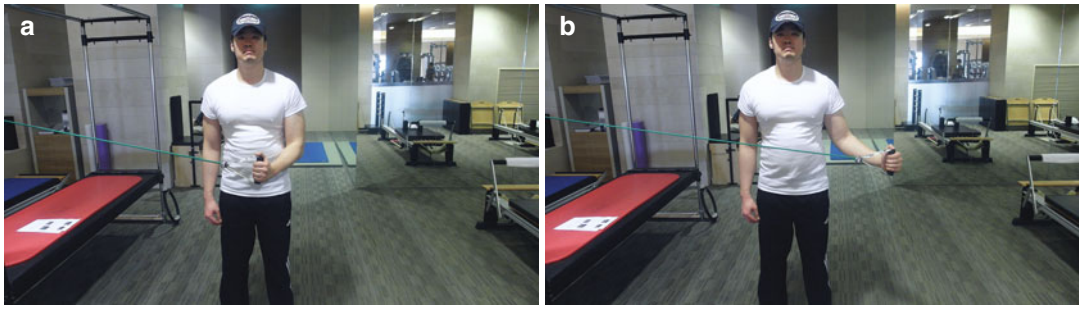


Fig. 31.33 The exercise band: deltoid and teres minor strengthening exercise

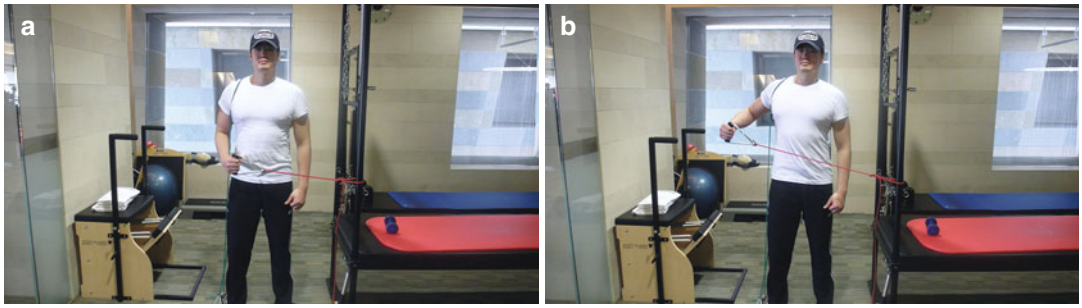


Fig. 31.34 The exercise band: lateral deltoid strengthening exercise

Fig. 31.35 Theraband isometric external rotation short lever: level II

31.2.3.9 The Exercise Band: Lateral Deltoid Strengthening Exercise

Stand with your injured shoulder far from the door (Fig. 31.34a). From an elbow flexed position, exercise by abducting your shoulder while holding the rubber band (Fig. 31.34b).

31.2.3.10 Theraband Isometric External Rotation Short Lever: Level II

Prepare by sitting down or standing up. Hold a towel with your injured shoulder with elbows in a 90° flexed position. Hold a rubber band with both palms facing upward (Fig. 31.35a). Simultaneously externally rotate your shoulders (Fig. 31.35b).

31.2.3.11 Theraband Isometric External Rotation Long Lever: Level II

Prepare by sitting down or standing up. Hold a towel with your injured shoulder with elbows in a 90° flexed position. Hold a rubber band with both palms facing upward (Fig. 31.36a). Simultaneously externally rotate your shoulders

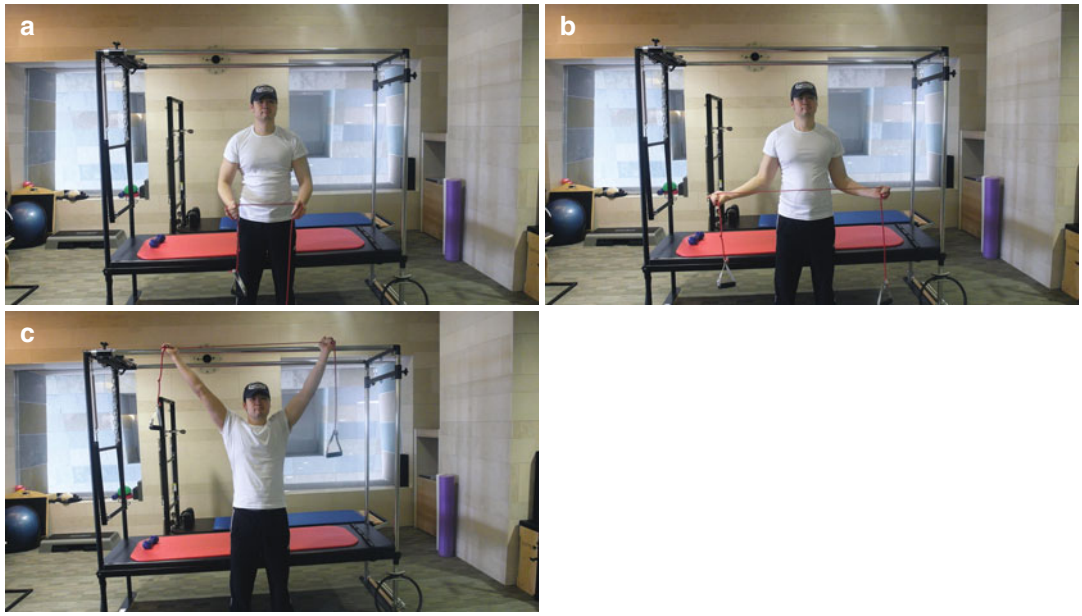


Fig. 31.36 Theraband isometric external rotation long lever: level II

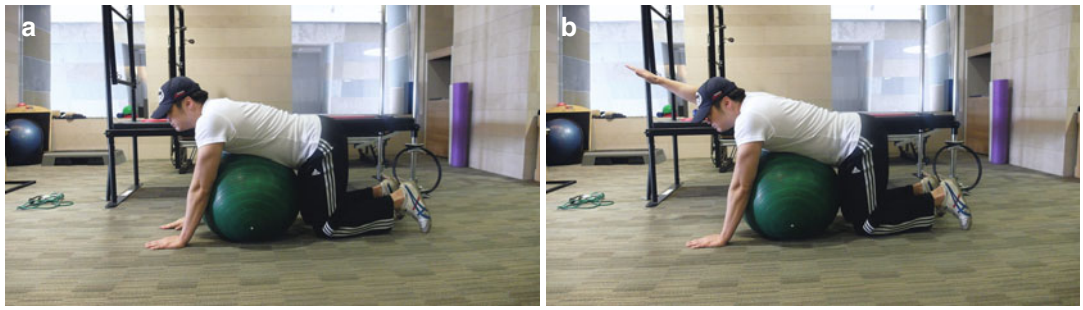


Fig. 31.37 Unilateral shoulder flexion in 4-point kneeling: level I

(Fig. 31.36b). After external rotation is complete, lift up the rubber band over your head (Fig. 31.36c).

31.2.3.12 Unilateral Shoulder Flexion in 4-Point Kneeling: Level 1

Kneel down over the ball. Fix both your hands to the floor with elbows extended (Fig. 31.37a). Gently exercise by lifting your injured shoulder (Fig. 31.37b).

31.2.3.13 Upper Trapezius Strengthening with External Rotation: Level II

Use two rubber bands. Step on a rubber band which is slung over your injured shoulder. Hold the other rubber band with both hands, palms facing upward (Fig. 31.38a). Externally rotate your injured shoulder (Fig. 31.38b).

Fig. 31.38 Upper trapezius strengthening with external rotation: level II

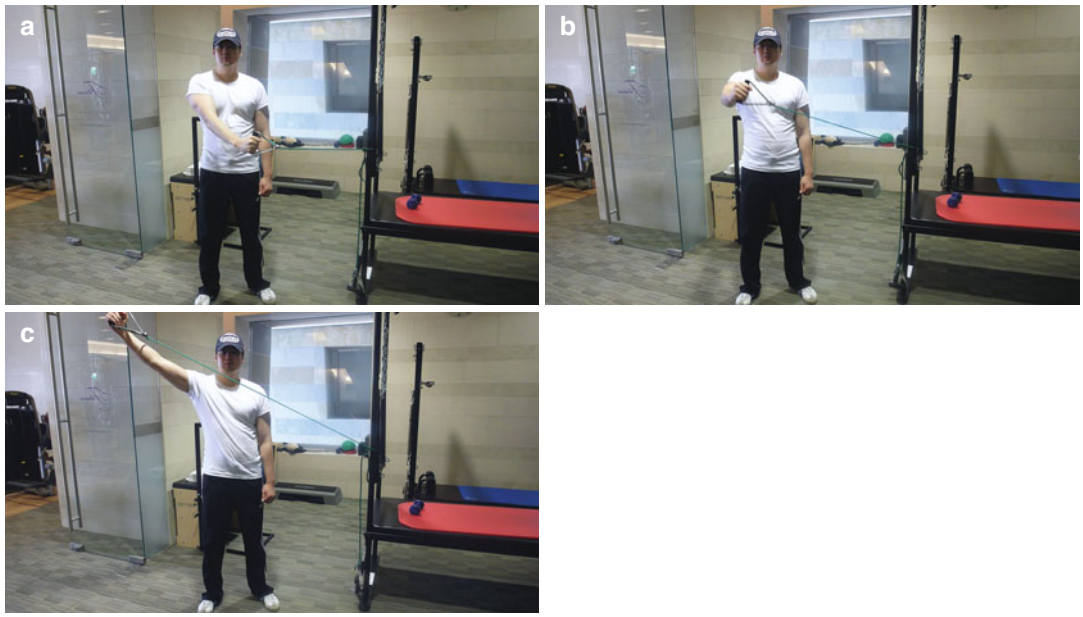


Fig. 31.39 Diagonal pattern exercise with theraband: level II

31.2.3.14 Diagonal Pattern Exercise with Theraband: Level II

Fix a rubber band to a door or a wall. Stand with your injured shoulder far from the door (or wall). Hold the rubber band in front of the opposite hip (Fig. 31.39a). With your elbows extended, pull the band diagonally using your injured shoulder (Fig. 31.39c).

31.2.3.15 Diagonal Pattern Exercise with Free Weight and Step: Level II

This exercise uses dumbbells and a footstep. Gather dumbbells to your chest till arms cross and make a squatting position while stepping out one foot (Fig. 31.40a). Step up on the footstep and externally rotate your shoulder and place

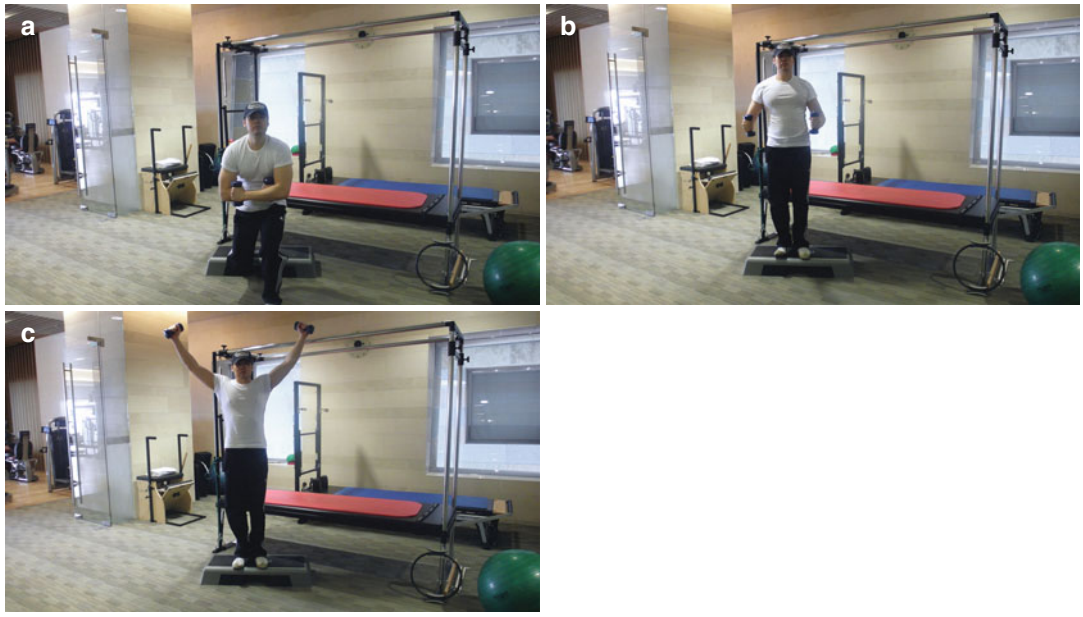


Fig. 31.40 Diagonal pattern exercise with free weight and step: level II

dumbbells in front of your chest (Fig. 31.40b). Lift up both your arms over the head making a "Y" shape (Fig. 31.40c).

31.2.3.16 Forty-Five Degree Raise: Level II

Stand straight and step on a rubber band with your foot which is on the same side of your injured arm. Hold the rubber band, thumb pointing to the ground. With your elbows extended, pull it up sideways (Fig. 31.41a).

31.2.3.17 Forward Flexion with Exercise Band: Level III

Stand straight and step on a rubber band with your foot which is on the same side of your injured arm. Hold the rubber band, thumb pointing to the ground (Fig. 31.42a). With your elbows extended, pull it up in front of your body (Fig. 31.42b).

31.2.3.18 Forward Press: Level II

Fix a rubber band to a door handle at your shoulder height. Stand with your back toward the wall (Fig. 31.43a). From an elbow flexed position, exercise by extending your elbow to forward flex your injured shoulder (Fig. 31.43b).

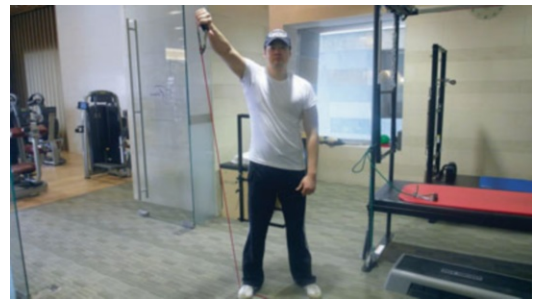


Fig. 31.41 Forty-five degree raise: level II

31.2.3.19 Shoulder Extension: Level III

Fix a rubber band to a door handle. Stand facing the wall. From an elbow extended position (Fig. 31.44a), exercise by pulling the rubber band with your shoulder (Fig. 31.44c).

31.2.3.20 Diagonal Pattern Abduction in Elevation to Adduction: Level III

Fix a rubber band to a door handle at your shoulder height. Stand beside the wall. The injured shoulder should be near the band (Fig. 31.45a). From abduction and flexion state, exercise by pulling the band to your opposite hip with the elbow extended (Fig. 31.45b).

Fig. 31.42 Forward flexion with exercise band: level III

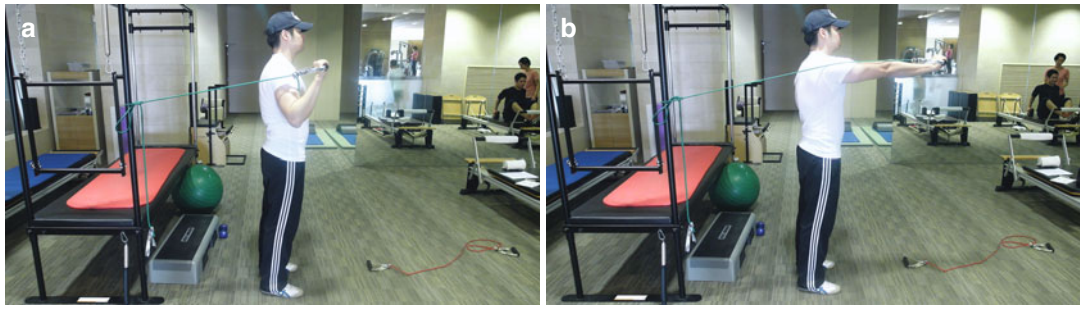


Fig. 31.43 Forward press: level II

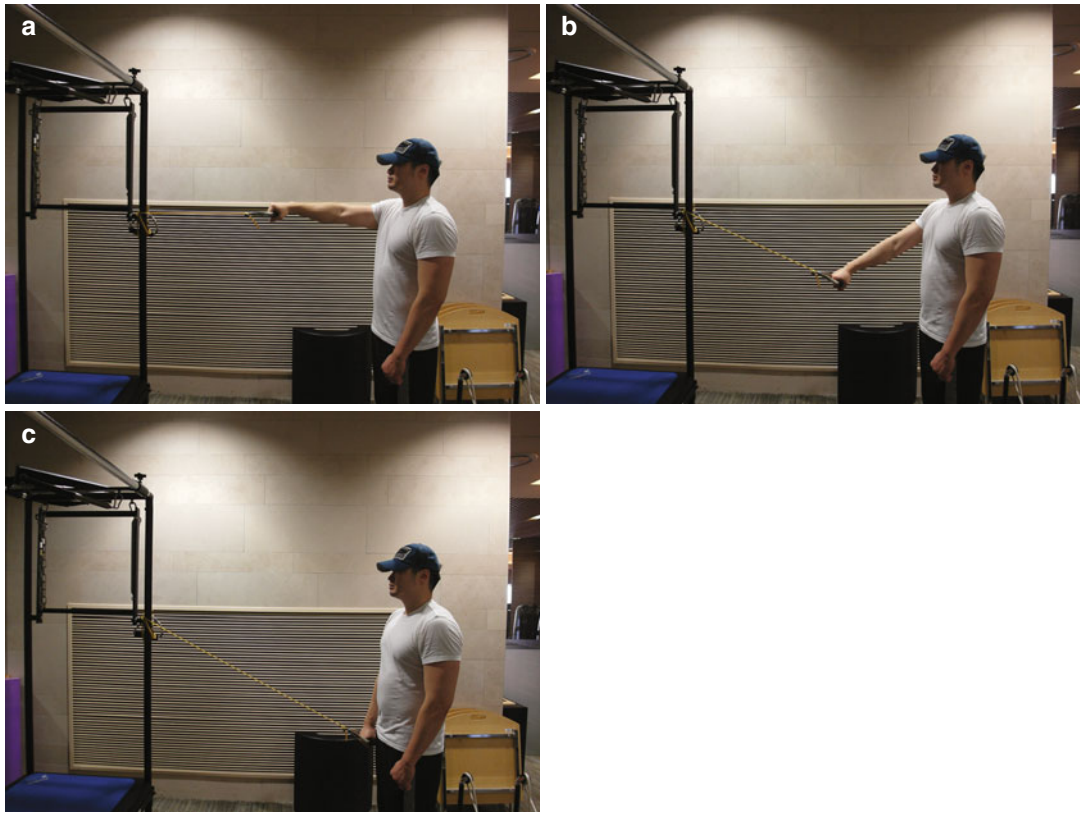


Fig. 31.44 Shoulder extension: level III

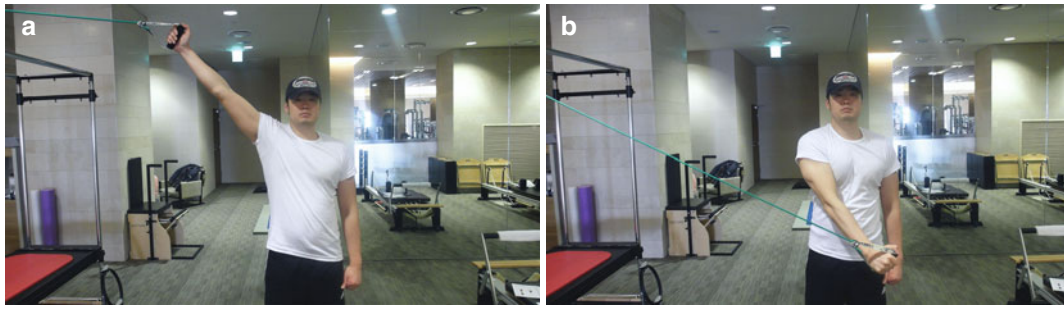


Fig. 31.45 Diagonal pattern abduction in elevation to adduction: level III

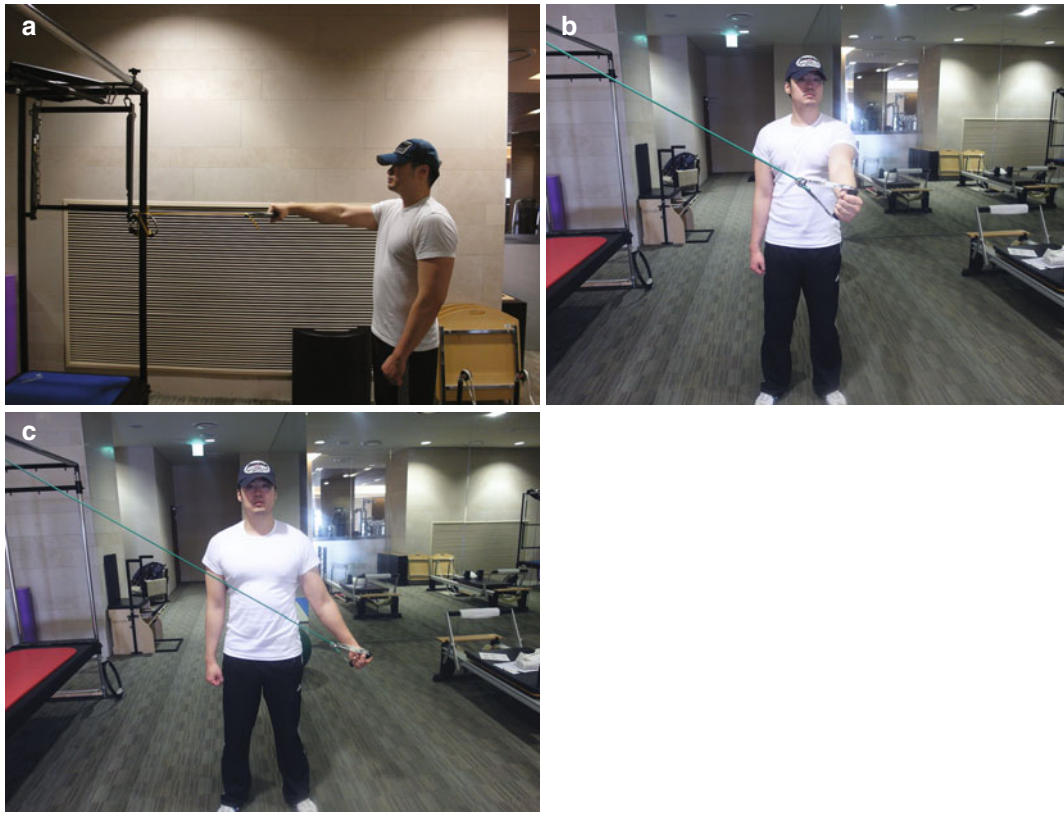


Fig. 31.46 Diagonal pattern adduction in elevation to abduction: level III

31.2.3.21 Diagonal Pattern Adduction in Elevation to Abduction: Level III

Fix a rubber band to a door handle at your shoulder height. Stand beside the wall. The injured shoulder should be far from the band (Fig. 31.46a). From adduction and flexion state, exercise by pulling the band to your opposite hip with the elbow extended (Fig. 31.46c).

31.2.3.22 Diagonal Pattern Adduction to Abduction in Elevation: Level III

Fix a rubber band to a door handle at your waist height. Stand beside the wall. The injured shoulder should be far from the band (Fig. 31.47a). From adduction state, exercise by pulling (abduction, forward flexion) the band to your injured shoulder level with the elbow extended (Fig. 31.47b).

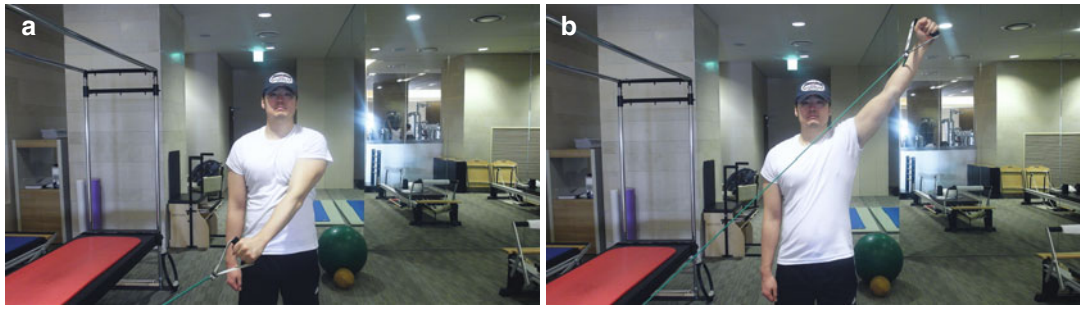


Fig. 31.47 Diagonal pattern adduction to abduction in elevation: level III

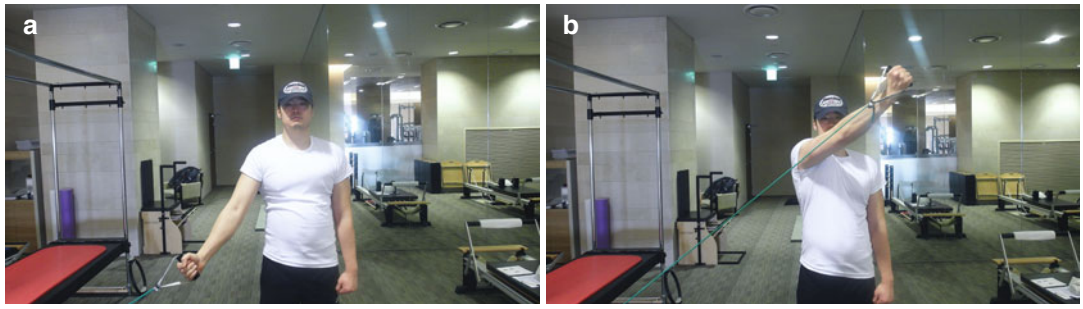


Fig. 31.48 Diagonal pattern abduction to adduction in elevation: level III

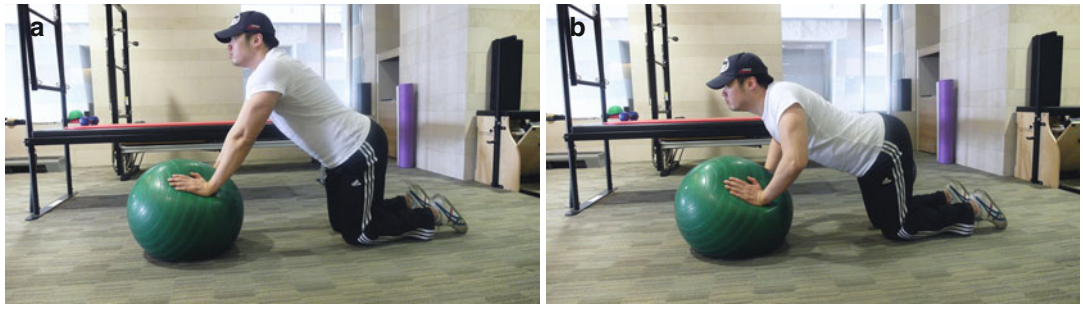


Fig. 31.49 Push-up on the ball: level III

31.2.3.23 Diagonal Pattern Abduction to Adduction in Elevation

Fix a rubber band to a door handle at your waist height. Stand beside the wall. The injured shoulder should be near the band (Fig. 31.48a). From abduction state, exercise by pulling (adduction, forward flexion) the band to your opposite shoulder level with the elbow extended (Fig. 31.48b).

31.2.3.24 Push-up on the Ball: Level II

Fix both your knees on a mat and kneel down placing both hands on a gym ball with both elbows extended (Fig. 31.49a). Exercise by flexing your elbows and returning back to your initial position (Fig. 31.49b). Spine must be kept upright.

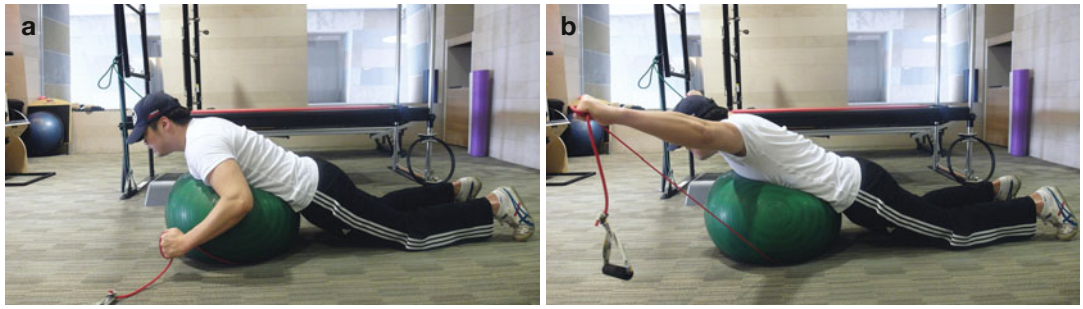


Fig. 31.50 Dynamic hug with theraband ball: level II

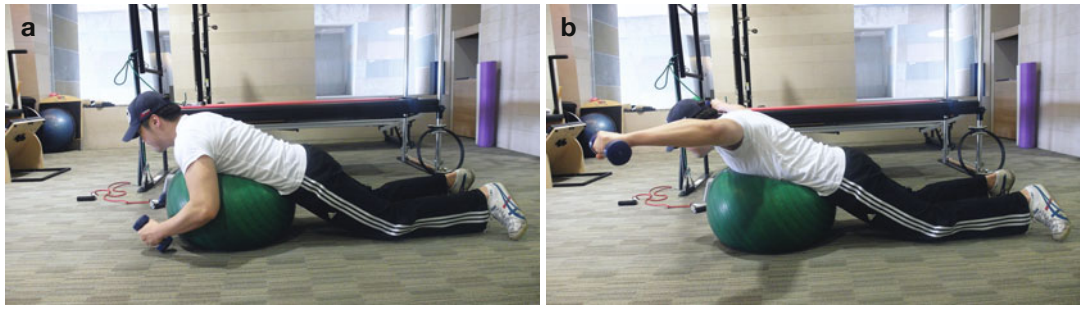


Fig. 31.51 Trunk extension over the ball with or without free weight: level II

31.2.3.25 Dynamic Hug with Theraband Ball: Level II

Bend over a gym ball with both your knees and toes fixed to the floor. Hold a rubber band with both your hands which is fixed under the gym ball (Fig. 31.50a). Exercise by extending your knees and pulling the rubber band with both arms (Fig. 31.50b).

31.2.3.26 Trunk Extension Over the Ball With or Without Free Weight: Level II

Bend over a gym ball with toes fixed to the floor. Comfortably let down both your arms (Fig. 31.51a). Exercise by spreading both your arms (Fig. 31.51b). You can also give weight by using dumbbells.

31.2.3.27 Sitting on the Ball with Arm Elevation: Level II

Sit up straight on a gym ball (Fig. 31.52a). Raise and lower your arm, and once you get used to it, try with both arms (Fig. 31.52b).

31.2.3.28 Prone Lying on the Ball: Level II

Prepare by lying over a gym ball with both feet and hands fixed to the floor (Fig. 31.53a). Pull your chest while the gym ball moves to your lower extremity (Fig. 31.53b).

31.2.3.29 Prone on the Ball with Knee Flexion: Level III

Place your thighs on a gym ball and both your hands to the ground (Fig. 31.54a). Flex your knees 90° (Fig. 31.54b).

31.2.3.30 Prone on the Ball with Hip Extension: Level II

Prepare by lying over a gym ball with both feet and hands fixed to the floor (Fig. 31.55a). Balancing your body using only a single leg, raise up the other leg with the knee extended (Fig. 31.55b).

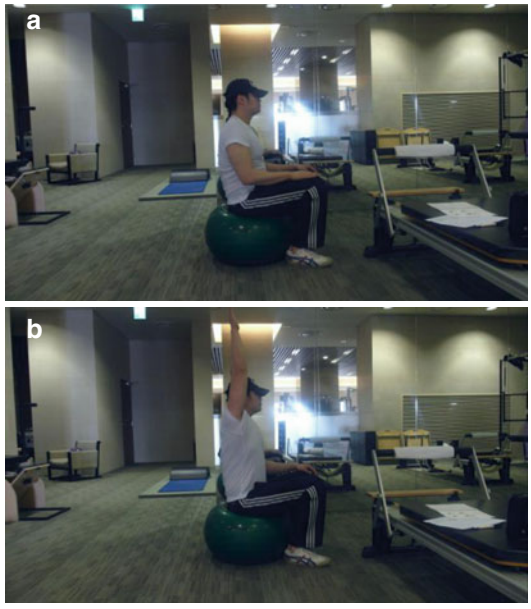


Fig. 31.52 Sitting on the ball with arm elevation: level II

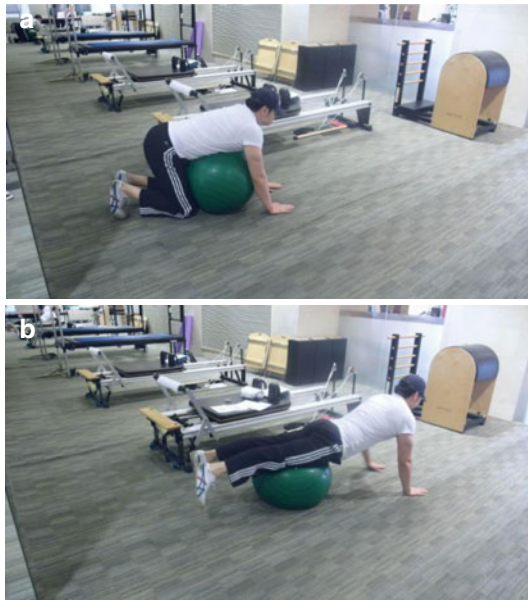


Fig. 31.53 Prone lying on the ball: level II

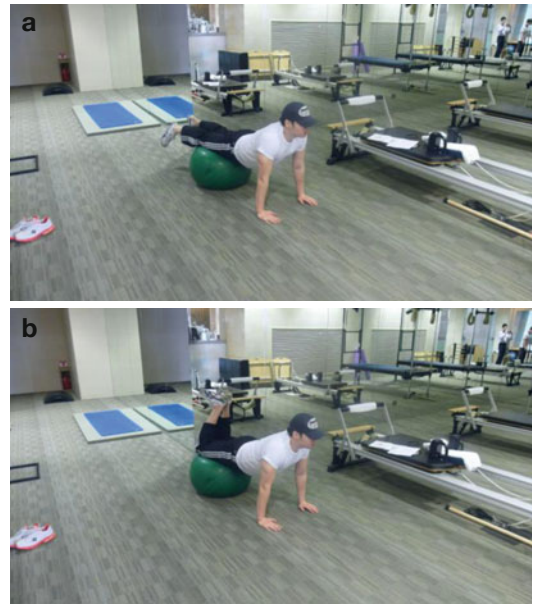


Fig. 31.54 Prone on the ball with knee flexion: level III

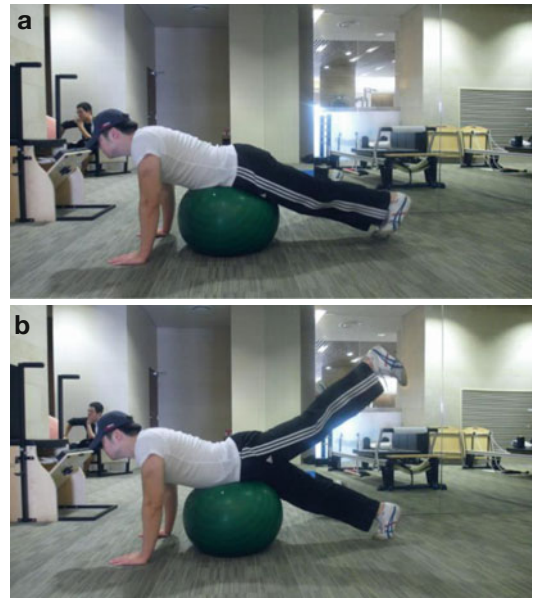


Fig. 31.55 Prone on the ball with hip extension: level II

31.2.3.31 Bridging on the Exercise Ball with Arm Weights: Level II

Hold dumbbells with both hands and sit on a gym ball. Lean back on the ball making balance till the ball is under your upper back and neck (Fig. 31.56a). Exercise by pushing up your dumbbells (Fig. 31.56b).

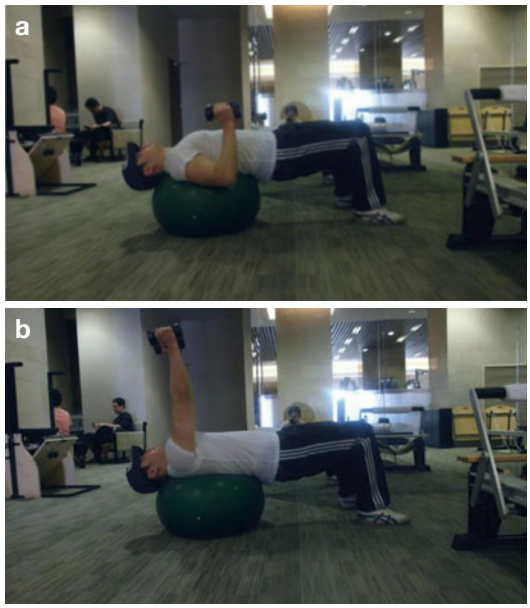


Fig. 31.56 Bridge on the exercise ball with arm weights: level II

31.2.3.32 Bridging on the Exercise Ball with Football

Hold a football with both hands and sit on a gym ball. Lean back on the ball making balance till the ball is under your upper back and neck (Fig. 31.57a). Exercise by pushing up your football (Fig. 31.57b).

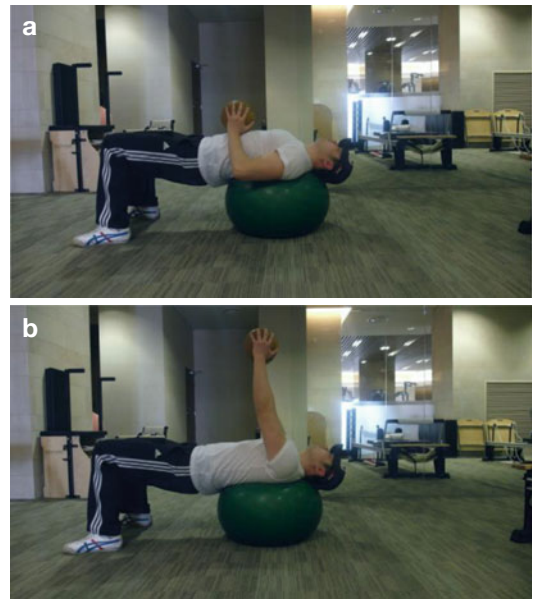


Fig. 31.57 Bridge on the exercise ball with football

Rehabilitation: Part II. Advanced

32

Jin-Young PARK and Young-Min Noh

32.1 Lower Trapezius Muscle Exercise

32.1.1 Functions

1. Scapular depression and adduction
2. Extension of the thoracic vertebra
3. Most important role for shoulder function and posture maintenance
   Origin:
   - External occipital protuberance
   - Medial 1/3 of the superior nuchal line of the occipital bone
   - Ligamentum nuchae
   - Spinous process of the 7th cervical spine
   - Spinous process of all thoracic spines
   - Corresponding supraspinal ligament insertions

   Insertion:
   - Medial end of the scapular spine
   - Tubercle at the apex of the scapular surface

32.1.2 Rehabilitation Exercise

32.1.2.1 Latissimus Pull Down

Method of exercise:

1. Sit in front of the instrument and hold the bar with your shoulder width (Fig. 32.2a).
2. Pull down the bar to your shoulder level while you are breathing out (have a feeling that you are pulling down with your shoulder) (Fig. 32.2b).
3. If you hold the bar with an undergrip or lean backward while pulling the bar, more stimuli will be done on the lower trapezius.

32.1.2.2 Posterior Fly

Method of exercise:

1. Lie prone on the instrument with both your arms hanging down (Fig. 32.3a).
2. With your elbows extended and thumbs pointing upward, lift up your arms to your shoulder level (Fig. 32.3b).
3. When dropping your arm, give resistance to the upper portion of your elbows.

32.1.2.3 Modified Prone Cobra

Method of exercise:

1. Lie prone on a mat (Fig. 32.4a).
2. Extend your upper body and hold your chest to be apart from the floor about 10 cm (Fig. 32.4b).

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3. Both palms should face away from your body and thumbs pointing upwards.
4. Pull both your scapulae to maintain a finger width between the medial borders of the scapulae.
5. When returning to initial position, keep your palm facing upward and give resistance.

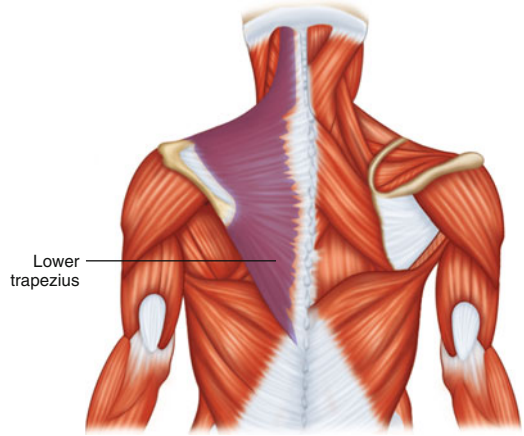

Fig. 32.1 Lower trapezius

32.1.2.4 Prone V-Raise

Method of exercise:

1. Lie prone on a incline bench or a stability ball (Fig. 32.5a).
2. Make your shoulders 180° and abduct 120° with your elbow slightly flexed.
3. With your thumb pointing upward, lift up your arms to the ear level by pulling shoulder and concentrate your scapula together (Fig. 32.5b).
4. While returning to your initial position, keep your arm position, and when the trainer is giving help, give resistance above the elbows only.
5. You can use a dumbbell if you have done sufficient exercise with your bare hands.

32.2 Middle Trapezius

32.2.1 Function

1. Function: scapular retraction, adduction, and upward rotation

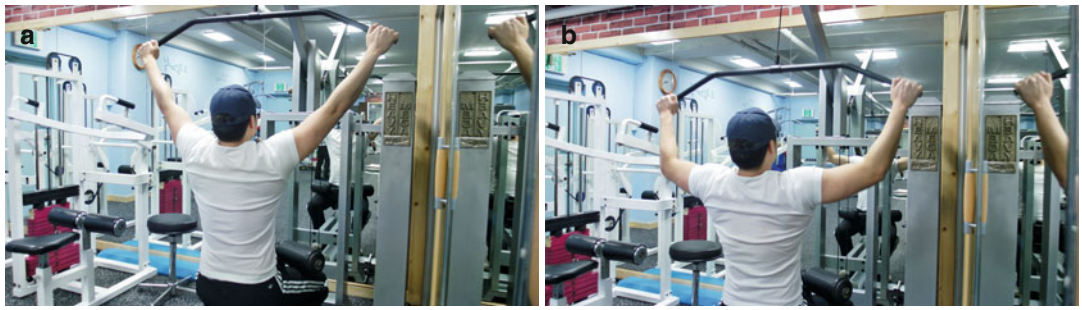

Fig. 32.2 Latissimus pull down

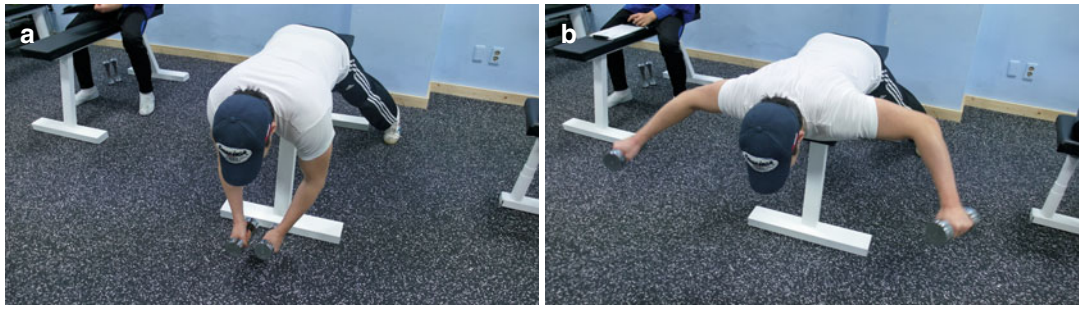

Fig. 32.3 Posterior fly

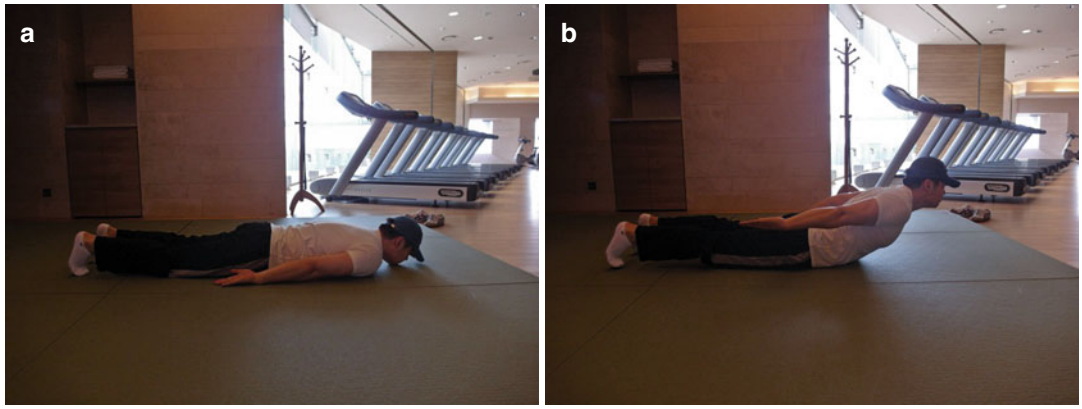

Fig. 32.4 Modified prone cobra

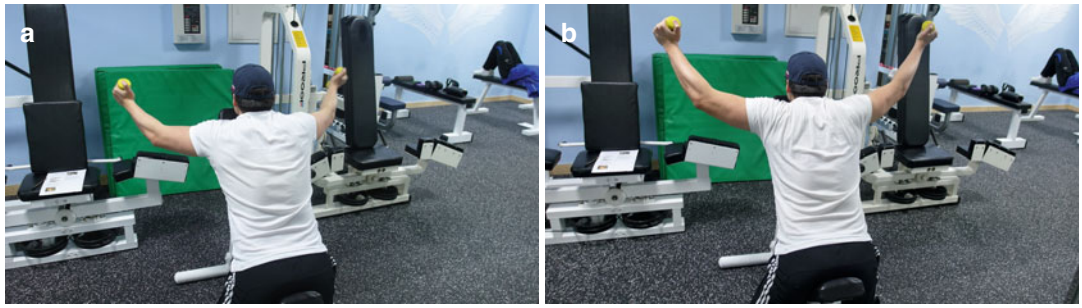

Fig. 32.5 Prone V-Raise

2. Origin:
   - External occipital protuberance
   - Medial 1/3 of the superior nuchal line of the occipital bone
   - Ligamentum nuchae
   - Spinous process of the 7th cervical spine
   - Spinous process of all thoracic spines
   - Corresponding supraspinal ligament insertions
3. Insertion:
   - Medial margin of the acromion
   - Superior lip of the posterior border of the spine of the scapula

32.2.2 Rehabilitation Exercise

32.2.2.1 Seated Cable Row

1. Bend your knees slightly and place your back in an upright position (Fig. 32.7a). Slowly pull the handle to your lower chest without moving your upper body (Fig. 32.7b).
2. Return to the starting position maintaining your upper back muscle tension.
3. When exercising be careful not to bounce on your upper body and try not to load pressure on your lower back by straightening it. When returning, try not to flex your lower back.
4. Maximally squeezing your upper back muscle, pull up the dumbbells with the elbows brushing your body with keeping head up.
5. A bar in front to prevent hyperflexion of the spine (kyphosis) makes it more effective to those with back pain and old age.

32.2.2.2 Bent-Over Dumbbell Row

1. Stand on your shoulder width, draw back hips together, and with straight back bend over 40–50° (Fig. 32.8a).

2. Maximally squeezing your upper back muscle, pull up the dumbbells with the elbows brushing your body with keeping head up (Fig. 32.8b).
3. It is effective to maximally contract your upper back muscle. Try not to spread your shoulders.
4. Bending too much can load pressure on your lower back. So do not bend over exceeding your comfort.

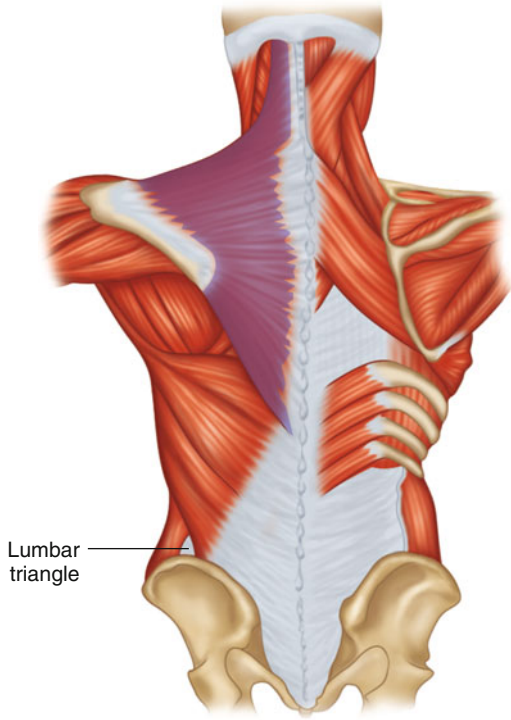

Fig. 32.6 Upper trapezius (please point the upper side or trapezius muscle)

32.2.2.3 Band Rowing

1. This exercise is the same as cable row. The difference is using a rubber band. Fix the band to your feet (Fig. 32.9a), place arms beside the body, and slowly pull the band (Fig. 32.9b); then return.
2. Make sure not to injure your lower back, do not move your back too much (keep it straight). Keep in mind that this exercise is not for your lower back but for your upper back.

32.2.2.4 Reverse Dumbbell Fly

1. This exercise is for the posterior deltoid, but it can also help the trapezius.
2. Try to concentrate on maximally contracting the upper back muscle, not the deltoid.

32.3 Upper Trapezius

32.3.1 Function

1. Elevation and extension of the shoulder
2. Origin:
   - External occipital protuberance
   - Medial 1/3 of the superior nuchal line of the occipital bone
   - Ligamentum nuchae
   - Spinous process of the 7th cervical spine
   - Spinous process of all thoracic spines
   - Corresponding supraspinal ligament insertions
3. Insertion:
   - Posterior border of the lateral 1/3 of the clavicle

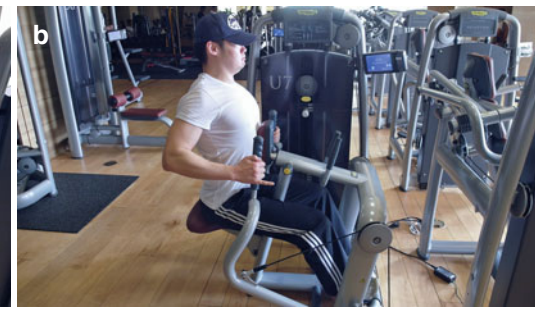

Fig. 32.7 Seated cable row

Fig. 32.8 Bent-over dumbbell row

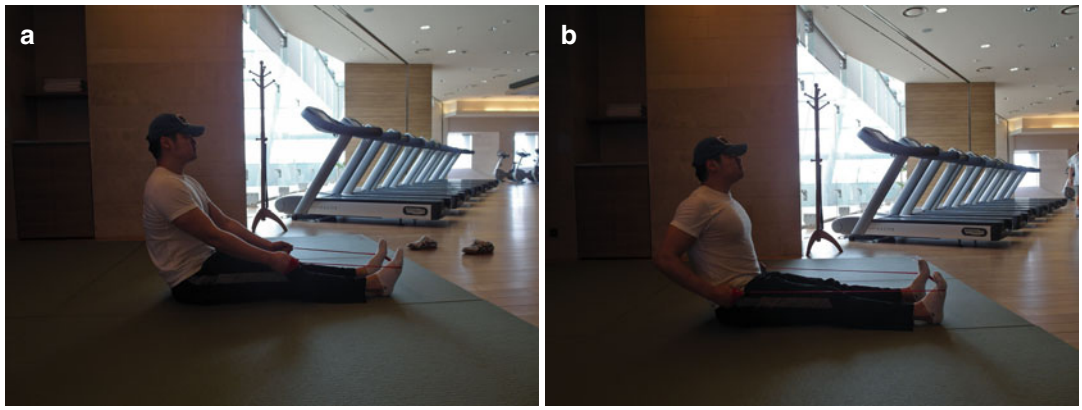

Fig. 32.9 Band rowing

Fig. 32.10 Reverse dumbbell fly

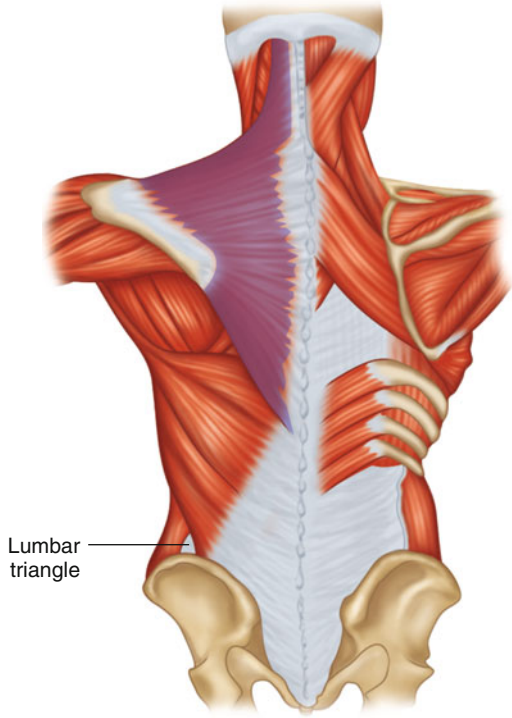

Fig. 32.11 Upper trapezius

32.3.2 Rehabilitation Exercise

32.3.2.1 Band Shoulder Shrug

1. Step on a rubber band with your knees flexed slightly and your back straight (Fig. 32.12a).
2. Keeping your back straight, shrug your shoulders as much as you can (Fig. 32.12b).
3. Do not bounce on your lower back or legs.
4. Breathe in when shrugging up, breathe out when you are returning to initial position.

32.3.2.2 Barbell Shrug

1. This exercise is well known for upper trapezius muscle exercise. Stand on your shoulder width with your knees flexed slightly and your back straight (Fig. 32.13a).
2. Hold the barbell with your shoulder width and shrug as much as you can by contracting the trapezius muscle. Pause for a bit then slowly come back to the initial position (Fig. 32.13b).
3. The barbell can also be held from your back.
4. Breathe in when shrugging up, breathe out when returning to the initial position.
5. Holding the barbell from your back can limit your lower back bending too much (kyphosis) but could be dangerous to your posture. So try with a light weight.

32.3.2.3 Dumbbell Upright Row

1. Stand straight and hold the dumbbells with an overhand grip (Fig. 32.14a).
2. Lift the dumbbells just below your chin, make sure both elbows are above your hands (Fig. 32.14b).
3. Stay still for a while maintaining tension on your trapezius. Breathe in and lower the dumbbells beside your body.
4. It stimulates trapezius muscles more in narrower grip, as opposed to the deltoids.

32.3.2.4 Barbell Upright Row

1. Holding the barbell with a short length between the hands develops the trapezius muscle, and broad length between the hands develops the deltoid muscle.

Fig. 32.12 Band shoulder shrug

Fig. 32.13 Barbell shrug

32.4 Serratus Anterior

Serratus anterior is not a big muscle, but it is very important for maintaining stabilization of the scapula. Under innervation of the long thoracic nerve, this muscle originates from the first to the ninth (or tenth) rib and inserts on the medial border of the scapula. It consists of serratus anterior supe-

Fig. 32.14 Dumbbell upright row

Fig. 32.15 Barbell upright row

rior, serratus anterior intermediate, and the serratus anterior inferior. All three parts pull the scapula toward the thorax. To restate it, these muscles act together to hold the scapula in the right place.

The lower portion externally rotates and pulls forward the lower part of the scapula. This makes lifting of your arm possible. The upper portion elevates the scapula.

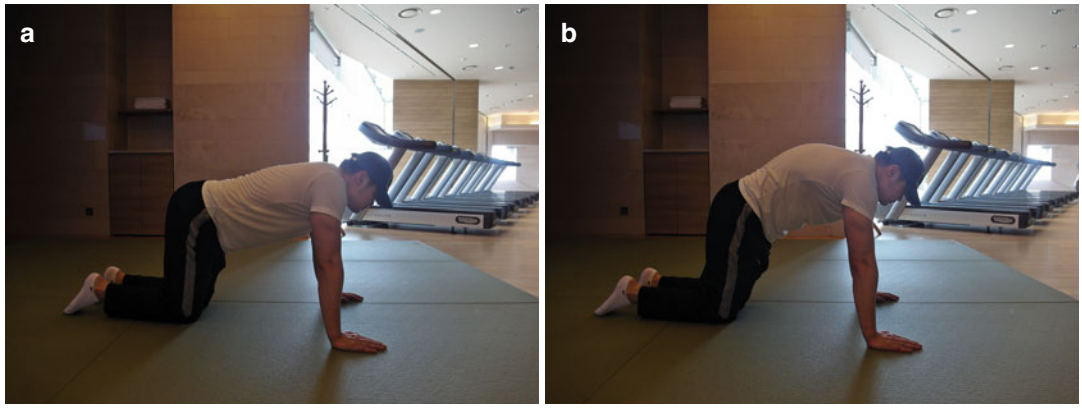

Fig. 32.16 Push-up plus or wall push-up

The serratus anterior plays a major role in scapula protraction, and this action is important in throwing or arm swinging (boxer's muscle).

The serratus anterior stabilizes the scapula on the rib cage against the forward loading, and if the muscle does not act right, scapula winging occurs. If the scapula is not in the right place, it causes pain during ROM exercise. It also causes impingement syndrome or tear of the rotator cuff. For athletes, it causes GIRD and many other problems. Especially for baseball or volleyball players who do the throwing motion too much, weakened muscles around the scapula including the serratus anterior cause major decline for exercise capacity.

Rehabilitation must be preceded to treat the weakened serratus anterior, and many of the cases improved from rehabilitation. Before starting the treatment flexibility of the joint should be retained. Secure flexibility to those with GIRD by sleeper stretch and those with coracoid inflexibility with open book stretch.

Origin:

- Outer surface and superior border of the first 8 or 9 ribs
- Aponeuroses covering the intervening intercostal muscle

Insertion:

- Ventral surface of the medial border of the scapula

32.4.1 Push-up Plus or Wall Push-up

This is the main exercise. This exercise is a usual push-up including scapula protraction. It can be done on several postures and also can be done standing with your hands leaning on the wall (Fig. 32.16a). Width between your hands and height of your hands affect different parts of the muscle. Slowly exercise and unlike the usual push-up, do not use your elbow but use your scapula (Fig. 32.16b). If you don't have sufficient muscle power, exercise against the wall or knees and elbows on the floor. Once you retain your muscle power, exercise like the usual push-up with knees not touching the floor.

32.4.2 Pull Over

This exercise is for the anterior chest muscles but also has great effect for the serratus anterior. Start with not too much weight before retaining muscle power. The key point is to protract your scapula while lifting up the dumbbell.

32.4.3 Dumbbell Scapula Protraction

Exercise can be done on either one hand or both hands. Dumbbell weight can be controlled depending on your muscle power. This exercise is adequate for an initial state without sufficient muscle power.

Fig. 32.17 Pull over

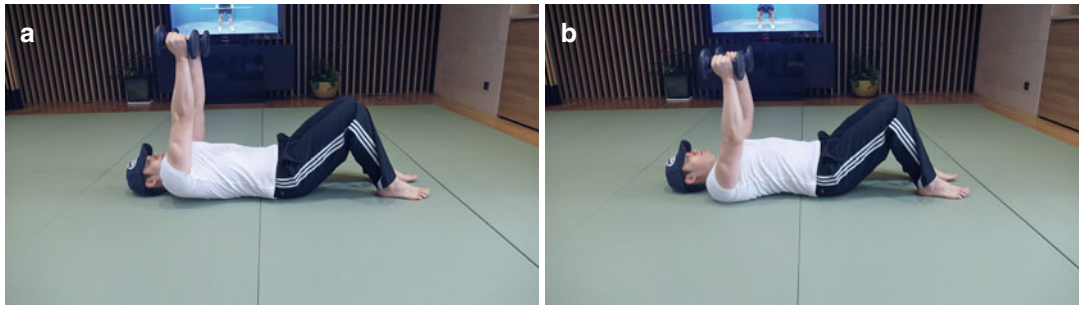

Fig. 32.18 Dumbbell scapula protraction

With your arms fully extended (Fig. 32.18a), protract your scapula (Fig. 32.18b). The opposite scapula should be stuck on the floor tightly.

32.5 Latissimus Dorsi

32.5.1 Functions

1. Adduction, extension, and internal rotation of the shoulder
2. Partly acts as a scapula stabilizer
3. Origin:
   Aponeurotic origin:
   - Spinous processes of the lower 6 thoracic and all lumbar and sacral vertebrae
   - Supraspinous ligament
   - Posterior part of the crest of the ilium

   Muscular origin:
   - Muscular fasciculi from the external lip of the crest of ilium
   - Caudal 3–4 ribs
   - Inferior pole of the scapula

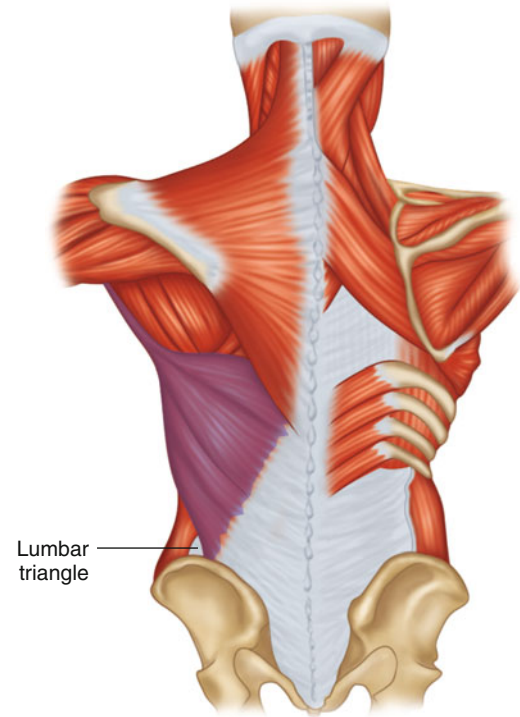

Fig. 32.19 Latissimus dorsi

Fig. 32.20 Lateral pull down

Insertion:
- Distal part of the intertubercular groove of the humerus

32.5.2 Rehabilitation Exercise

32.5.2.1 Lateral Pull Down

1. Sit in front of the instrument and hold the bar just wider than your shoulder width (Fig. 32.20a).
2. Breathe in while pulling the bar to your shoulder level with a feeling of using the shoulders (Fig. 32.20b).
3. It is important to make your back straight, and it is more effective when elbows are behind your back.
4. Spreading your shoulders can give more stimulation.

32.5.2.2 Bent-Over Dumbbell Row

1. Spread your legs as wide as your shoulder width, and with knees flexed slightly, and take care of your back keeping straight (Fig. 32.21a).
2. Breathe out while pulling the dumbbells to the lower abdomen or pelvis by bending your elbows (Fig. 32.21b).
3. Slowly return with resistance.

32.5.2.3 Body Row

1. Hang on a horizontal bar, pull up to the midsternum of your body (Fig. 32.22a).
2. Slowly lower your body with straight back keeping parallel to the floor (Fig. 32.22b).

32.6 Pectoralis Minor Muscle Exercise

32.6.1 Anatomy

1. Origin:
   Clavicular part:
   - Anterior surface of the sternal 1/2 of the clavicle

   Sternocostal parts
   - 1/2 the breadth of the anterior surface of the sternum (cartilages of the 6th/7th ribs)
   - Cartilages of all the true ribs

   Abdominal parts:
   - Aponeurosis of the external abdominal oblique muscle
2. Insertion:
   Muscle rotates before its insertion
   - Lower end of the lateral lip of the intertubercular sulcus of the humerus (3 laminae)

   Anterior part: clavicular part
   Middle part: upper sternocostal part
   Posterior part: lower sternocostal part and abdominal part

32.6.2 Function

1. Scapula protraction – Moving the scapula forward
2. Scapula depression – Moving the scapula downward
3. Scapula downward rotation – lowering and rotating the inferior angle of the scapula medially

Fig. 32.21 Bent-over dumbbell row

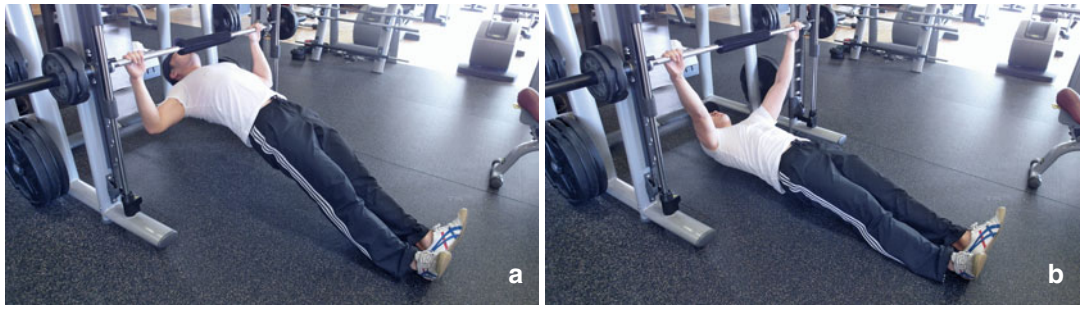

Fig. 32.22 Body row

32.6.3 Explanation

This muscle elongates during arm elevation, upward rotation of the scapula, external rotation, and retraction of the scapula.

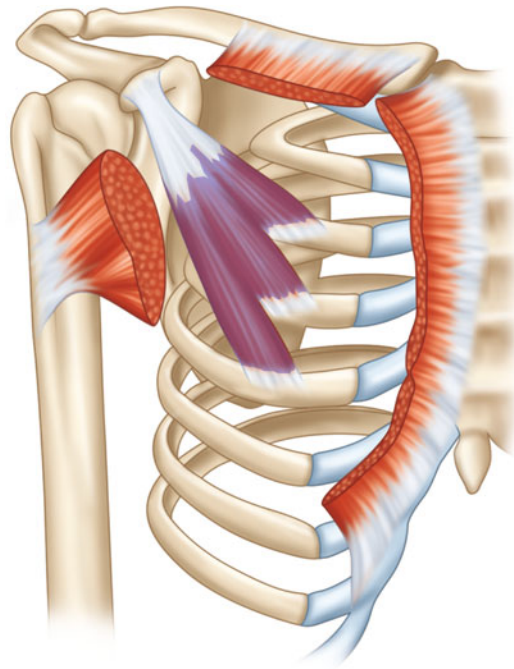

Fig. 32.23 Pectoralis minor

Also this muscle is the cause of bench-pressers shoulder when the exercise is not done in the right position or done too much.

32.6.4 Rehabilitation Exercise

32.6.4.1 Unilateral Corner Stretch

1. Stand up with your back straight (Fig. 32.25a).
2. Flex the elbow 90° and abduct the shoulder 90°.
3. Fix your hand and forearm against the wall, push your upper body to the front for 3 s. (do it like stretching your chest) (Fig. 32.25b)

32.6.4.2 Sitting Manual Stretch

1. Sit on a stool with your back straight.
2. Breathe in deeply.
3. The trainer should hold the inferior border of the scapula and push back the coracoid process of the scapula with the opposite hand.
4. Maintain for 3 s and after stretching, breathe out.

32.6.4.3 Supine Manual Stretch

1. Start with lying down.
2. Use a therapeutic bed or place a towel under your thoracic spine to slightly lift your scapula.
3. The trainer should flex the elbow 90° and abduct the shoulder 90°.
4. Then stretching is done by pushing the coracoid process of the scapula for 3 s.

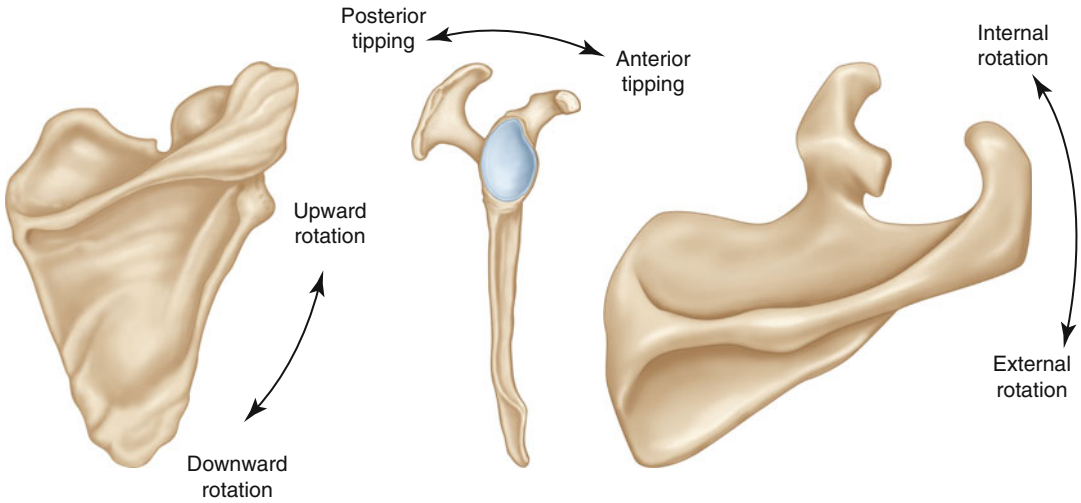

Fig. 32.24 Scapula function

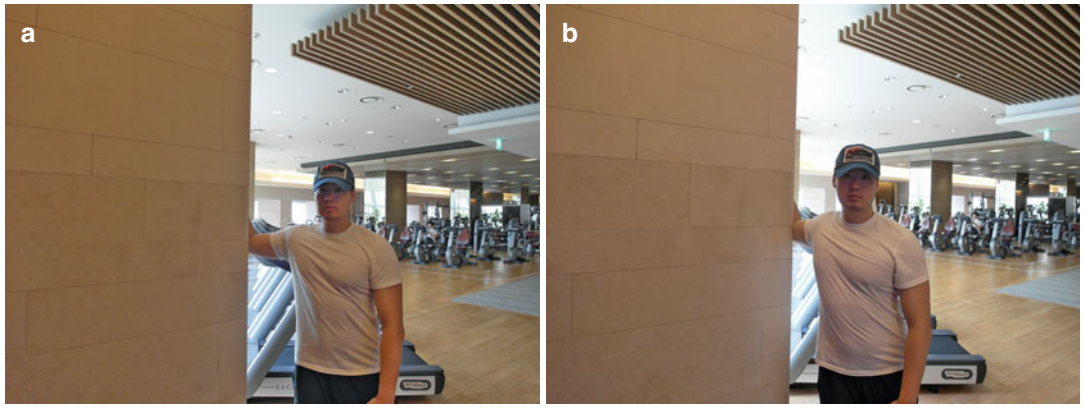

Fig. 32.25 Unilateral corner stretch

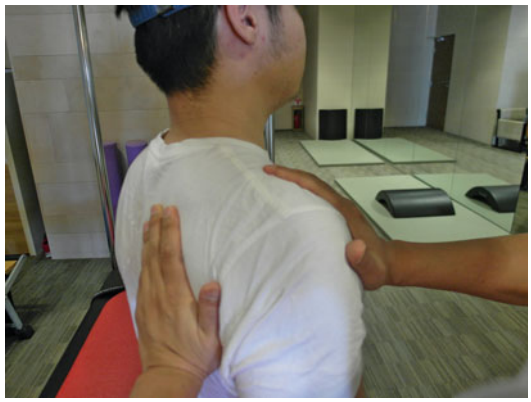

Fig. 32.26 Sitting manual stretch

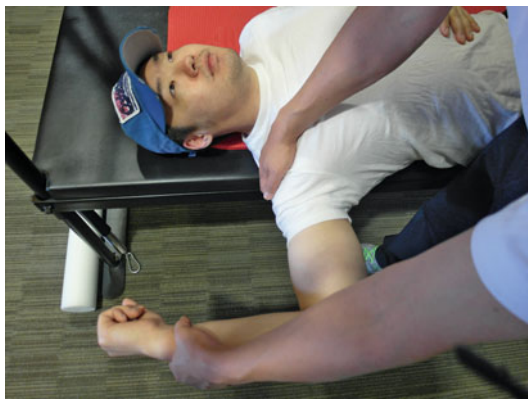

Fig. 32.27 Supine manual stretch

32.7 Pectoralis Minor Muscle Exercise

32.7.1 Rehabilitation Exercise

32.7.1.1 Chest Dips and Assisted Dips

1. Stand between a wide dip bar. Make balance by holding the bar handle.
2. With both arms extended, flex knees and cross your ankles. Lower your arm and lean forward and downward till your upper arm is parallel to the floor (Fig. 32.28a).
3. Then return to the initial position (Fig. 32.28b). To improve muscle endurance, set up a possible number of exercises you can do. Repeat three times. To increase muscle size, repeat three times making a 12–15 exercise a set.
4. If chest dip cannot be done, try assisted dip. Prepare in the same position, lower your arm till your upper arm is parallel to the floor. When returning to the initial position, get help from assistance.

32.7.1.2 Dumbbell Pull Over

1. Lie down with the sole of your foot and your back on the floor (Fig. 32.29a).
2. Hold dumbbells with both thumbs pointing to your head. With elbows slightly flexed, extend arms above your chest (Fig. 32.29b).

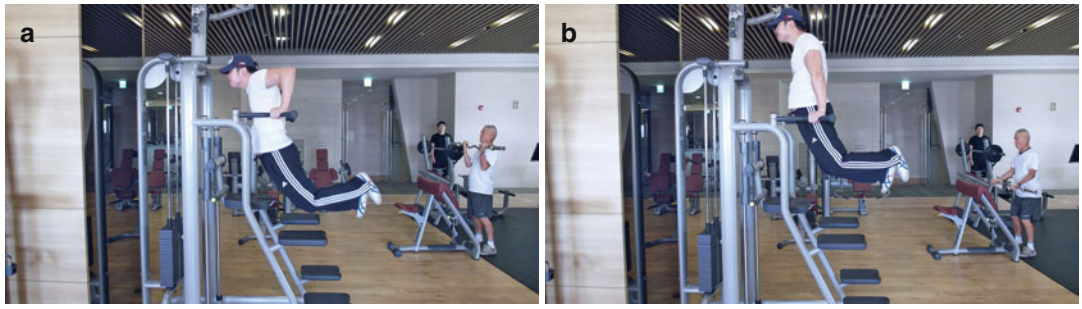

Fig. 32.28 Chest dips and assisted dips

Fig. 32.29 Dumbbell pull over

Fig. 32.30 Pec dec fly

3. Deeply breathe in and lower the dumbbells over your head. Make sure that your hips must be fixed to the floor.
4. If the upper arm lies parallel to your body, stop exercising.
5. While breathing out, raise dumbbells to initial position. Exercise three sets, 12–15 times a set.

32.7.1.3 Pec Dec Fly

1. Sit on the instrument, fix your hips and back to the rear seat.
2. Hold the handle, flex elbows 90°, position upper arms parallel to the floor and keep your forearms close to the pad (Fig. 32.30a).
3. While breathing out, push the pad slowly to contract the pectoralis muscle (Fig. 32.30b).

4. Pause at the maximum contraction point, then spread your arms while breathing in.

32.8 Pectoralis Major Muscle Exercise

32.8.1 Clavicular Head (Upper Head)

32.8.1.1 Anatomy

1. Origin:
   Clavicular part:
   - Anterior surface of the sternal 1/2 of the clavicle

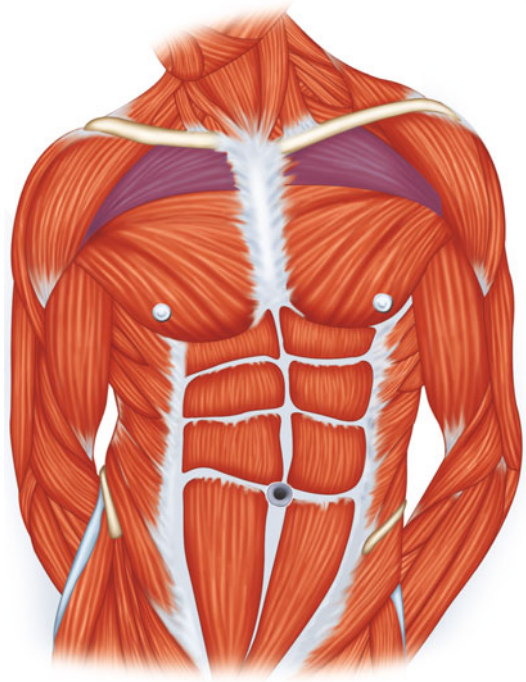

Fig. 32.31 Pectoralis major clavicular head

2. Insertion:
   Muscle rotates before its insertion
   - Lower end of the lateral lip of the intertubercular sulcus of the humerus (3 laminae)

   Anterior part: clavicular part

32.8.1.2 Functions

1. Flexion
2. Transverse adduction
3. Internal rotation
4. Adduction
5. Abduction

32.8.1.3 Incline Push-up

1. Lie prone by supporting the upper body to objects that have some height, with both arms on your shoulder width (Fig. 32.32a).
2. Make your body straight, even when exercising remember to position your hips and legs in a straight line.
3. While breathing in, continue to lower your arms before your chest touches the floor (Fig. 32.32b). Then pause for 1–2 s. Breathe out and extend your arms to return.
4. When lowering your arms, elbows should point away from your body and exercise slowly from fully extended position.
5. Give more resistance using a band. Exercise by coiling around your back and fix it with both hands.
6. Patients with shoulder pain can exercise by externally rotating both hands 45° to lower resistance.

Fig. 32.32 Incline push-up

Fig. 32.33 Incline bench (Dumbbell)press

32.8.1.4 Incline Bench (Dumbbell) Press

1. Lie down on a 15–45° inclined bench (Fig. 32.33a).
2. Hold a barbell or dumbbells on your shoulder width. Raise by pushing your elbow while breathing in and return by breathing out (Fig. 32.33b).

32.8.2 Sternal Head

32.8.2.1 Anatomy

1. Origin:
   Sternocostal parts
   - 1/2 the breadth of the anterior surface of the sternum (cartilages of the 6th/7th ribs)
   - Cartilages of all the true ribs
2. Insertion:
   Muscle rotates before its insertion
   - Lower end of the lateral lip of the intertubercular sulcus of the humerus (3 laminae)

   Middle part: upper sternocostal part

32.8.2.2 Functions

Shoulder:
1. Flexion
2. Transverse adduction
3. Internal rotation
4. Adduction
5. Abduction

Scapula:
1. Downward rotation
2. Depression
3. Abduction

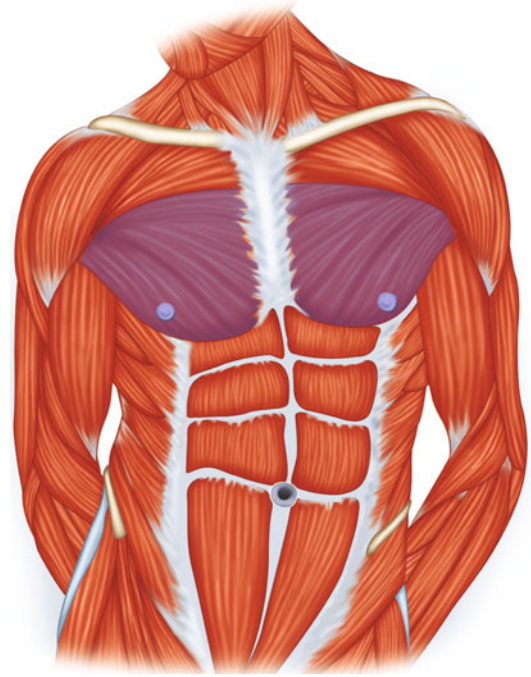

Fig. 32.34 Pectoralis major sternal head

32.8.2.3 Diamond Grip Push-up

It is called a stance push-up. This exercise strengthens the inner portion of the pectoralis major muscle.
1. From a usual push-up position, spread fingers and gather your hands together (Fig. 32.35a).
2. Thumbs and index fingers should be touched to each other, and extend your elbow about 90 % to not give much pressure.

32.8.2.4 Wide Stance Push-up

This exercise is for strengthening the outer portion of the pectoralis and deltoid. The more you

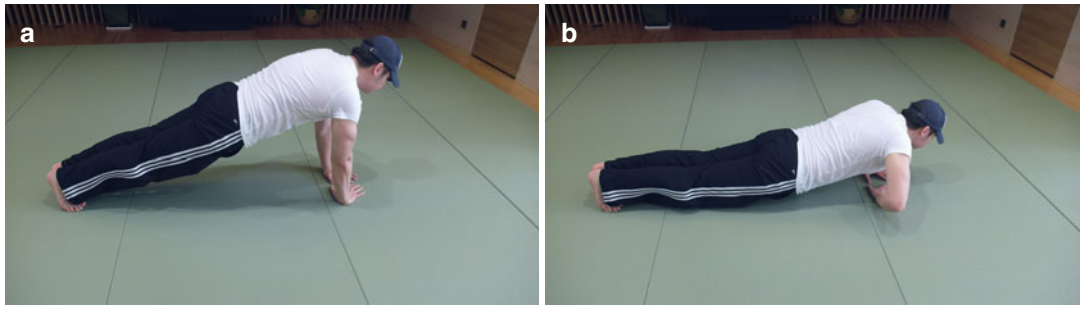

Fig. 32.35 Diamond grip push-up

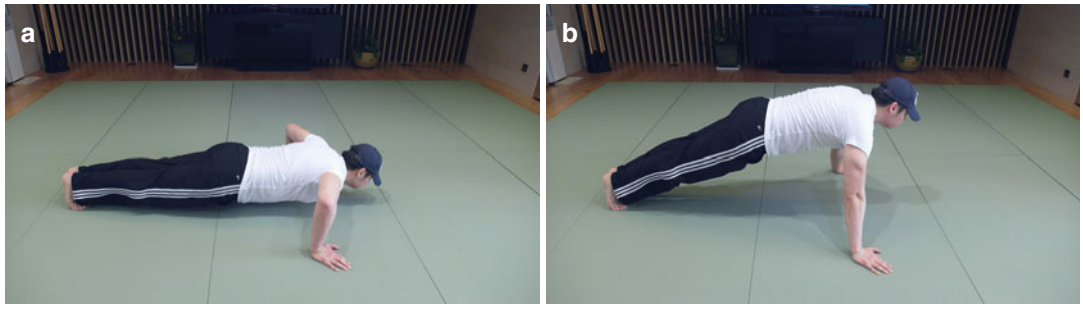

Fig. 32.36 Wide stance push-up

lower down, the more stretching will be done on the target muscle.

1. Spread fingers and place hands 45° outside to your body. Spread your arms as much as you can (Fig. 32.36a). Spreading arms more than 20–25 cm over your shoulder width will make lesser pressure on your rotator cuff.
2. While breathing in, flex arms just before your chest touches the floor. Pause for 1–2 s, then while breathing out, return to the initial position (Fig. 32.36b).
3. With strengthened muscle, try declined position, with the weak muscle exercise at inclined position.

32.9 Inferior Part of Pectoralis Major

1. Origin:
   Abdominal parts:
   - Aponeurosis of the external abdominal oblique muscle
2. Insertion:
   Muscle rotates before its insertion
   - Lower end of the lateral lip of the intertubercular sulcus of the humerus (3 laminae)

   Posterior part: lower sternocostal part and abdominal part

32.9.1 Strengthening Exercise

32.9.1.1 Decline Push-up

1. Lie prone by supporting legs to objects that have some height, with both arms on your shoulder width.
2. Make your body straight, even when exercising remember to position your hips and legs in a straight line (Fig. 32.37a).
3. While breathing in, continue to lower your arms before your chest touches the floor. Then pause for 1–2 s. Breathe out and extend your arms to return (Fig. 32.37b).
4. When lowering your arms, elbows should point away from your body and exercise slowly from fully extended position.

Fig. 32.37 Decline push-up

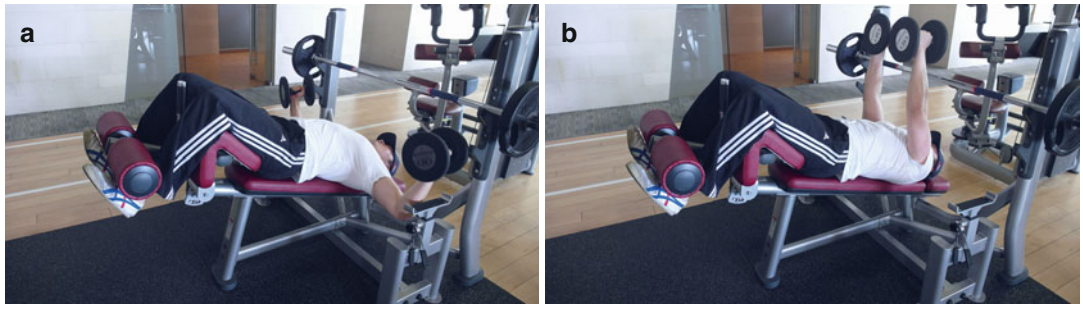

Fig. 32.38 Decline dumbbell fly

5. If you get used to this exercise, a gym ball could be used.
6. *Patients with shoulder pain can exercise by externally rotating both hands 45° to lower resistance.*

32.9.1.2 Decline Dumbbell Fly

Decline dumbbell fly is an exercise that you can do by spreading your arms in an arch position, then gathering them toward your chest like a hugging motion.

1. Sit on a bench with dumbbells on both your hands.
2. Place dumbbells beside your chest and lie down. Draw back your chin.
3. Slightly float your back. Make not too much arch on your back.
4. Hold the dumbbells and spread your arms in an arch position (Fig. 32.38a).
5. Fix your elbow angle, breathe out, and just using your shoulder, gather the dumbbells like a hugging motion (Fig. 32.38b).
6. While breathing in, lower your dumbbells.

32.10 Levator Scapulae Muscles and Rhomboideus Exercise

32.10.1 Anatomy

1. Origin:
   - Transverse processes of the atlas and axis
   - Posterior tubercles of the transverse processes of the 3rd and 4th cervical spine insertion
2. Insertion:
   - Vertebral border of the scapula between superior angle and triangular smooth surface at the root of the spine

32.10.2 Function

1. Scapula elevation, abduction
2. Scapula downward rotation
3. Right to left neck rotation
4. Right to left neck flexion

32.10.3 Explanation

This muscle holds the scapula upward and rotates inferiorly. It prevents scapulae dyskinesis and is important in rehabilitation. Also this muscle plays a major role in neck movement and has relations to referred pain from neck stiffness and around shoulder pain.

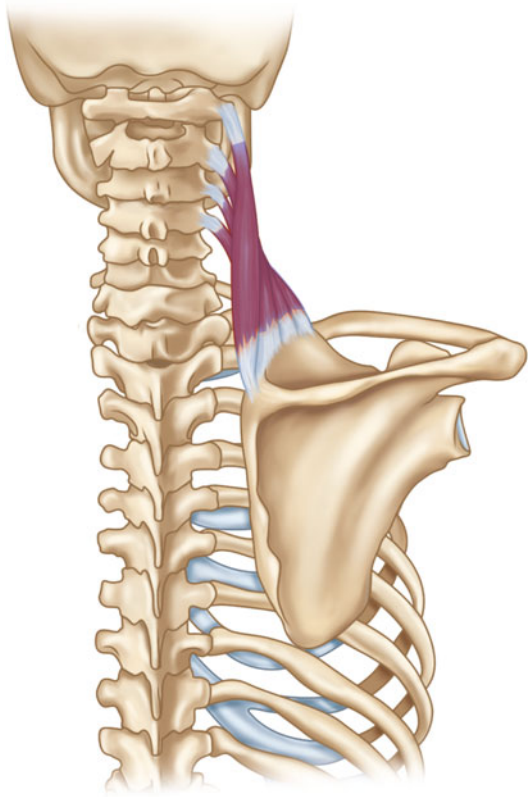

Fig. 32.39 Levator scapulae

32.10.4 Rehabilitation Exercise

32.10.4.1 Levator Scapulae Stretching

1. Sit on a chair with your back straight.
2. Place hand on your back, making elbows pointing to the roof (Fig. 32.40a).
3. With your opposite hand, make your head lean forward or sideways (Fig. 32.40b).

32.10.4.2 Barbell Shrugs

1. Stand with holding a barbell on your shoulder width (Fig. 32.41a).
2. Lift up the barbell by shrugging your shoulders with elbows extended (Fig. 32.41b).
3. Give resistance when lowering back.

32.10.5 Rhomboid Muscle Exercise

Functions

1. Adduction of the scapula by pulling it medially toward the vertebral column
2. Elevate scapula

   Origin:
   - Spinous processes of the 2nd, 3rd, 4th, and 5th thoracic vertebrae and supraspinous ligament insertions (major)
   - Inferior part of the ligamentum nuchae (minor)
   - Spinous processes of the 7th cervical and 1st thoracic vertebrae

   Insertion:
   - Above: medial border of the scapulae (major)

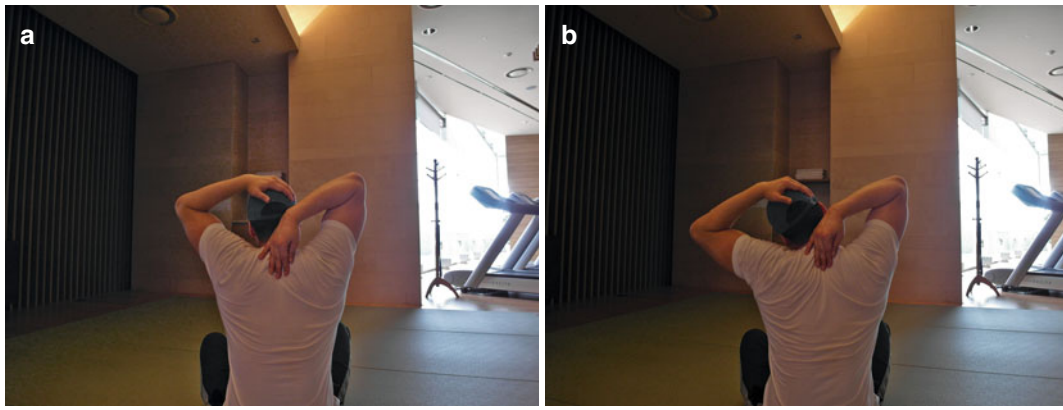

Fig. 32.40 Levator scapulae stretching

Fig. 32.41 Barbell shrugs

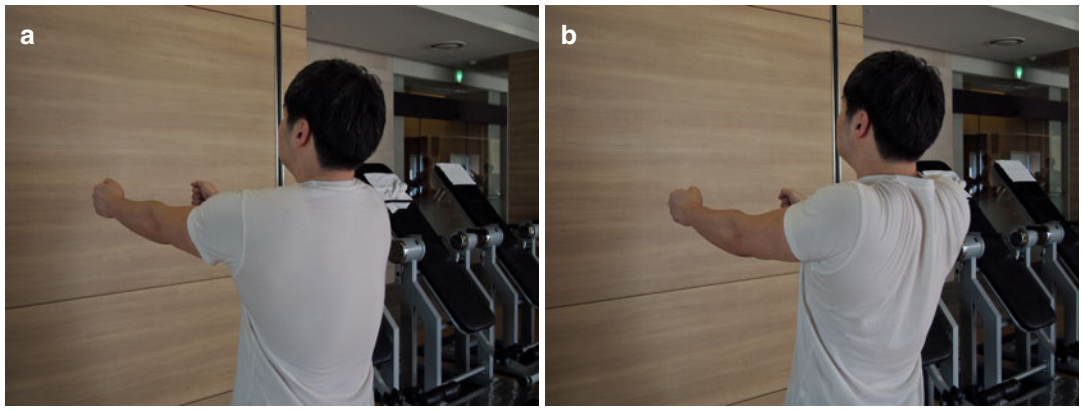

Fig. 32.42 Shoulder blade squeeze

- Below: inferior angle of the scapula (major)
- Base of the triangular smooth surface at the root of the spine (minor)

32.10.6 Rehabilitation Exercise

32.10.6.1 Shoulder Blade Squeeze

1. Get ready for this exercise on a standing position with your back straight.
2. Your chin should be tucked in slightly and your shoulders should be slightly backward (Fig. 32.42a).
3. Slowly tighten your rhomboids by squeezing or gathering your shoulder blades together as hard and much as possible (Fig. 32.42b).
4. Then relax, back to the initial position.

32.10.6.2 Darts (Modified Prone Cobra)

1. Lie prone on a mat (Fig. 32.43a).
2. Extend your upper body and hold your chest to be apart from the floor about 10 cm (Fig. 32.43b).
3. Both palms should face away from your body and thumbs pointing upward.
4. Pull both your scapulae to maintain a finger width between the medial borders of the scapulae.
5. When returning to initial position, keep your palm facing upward and give resistance.

32.10.6.3 Resistance Band Pullbacks

1. Start in a standing position with your back straight and holding a resistance band tied to a secure pole with both elbows in a 90° flexion (Fig. 32.44a).
2. Slowly pull your arms backward, contracting your rhomboids by squeezing or gathering your shoulder blades together (Fig. 32.44b).
3. Then relax and go back to the initial position.

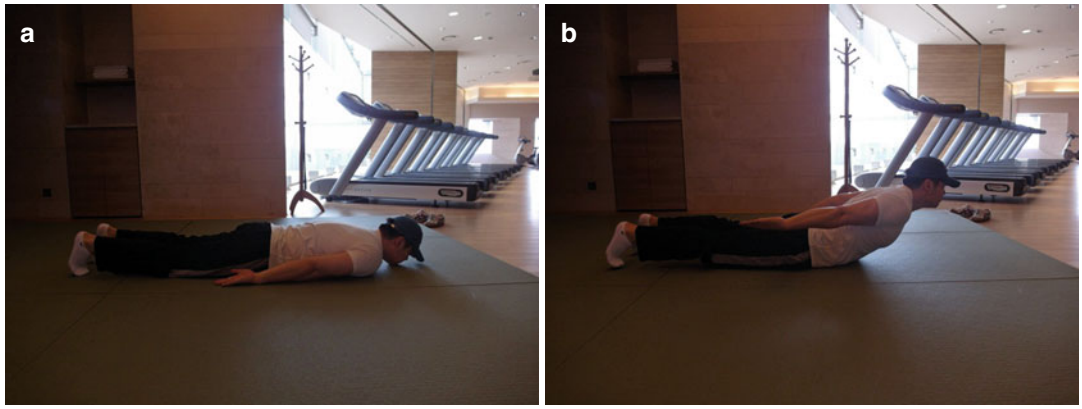

Fig. 32.43 Darts (modified prone cobra)

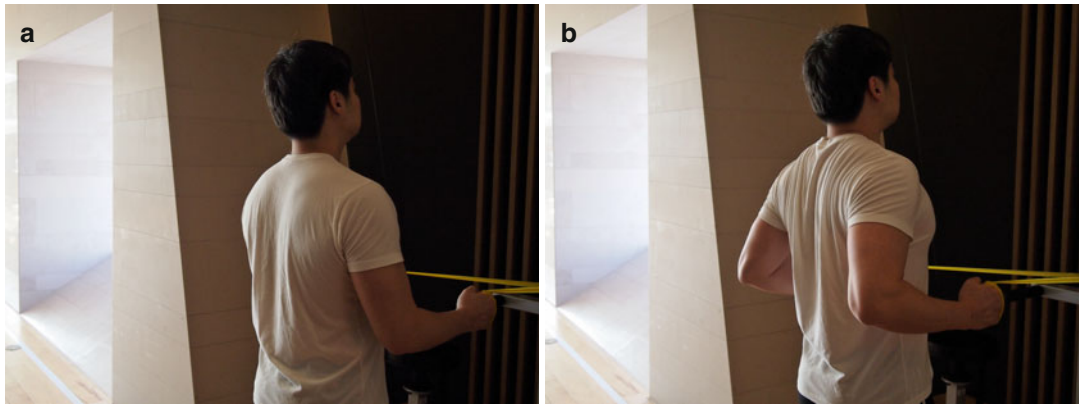

Fig. 32.44 Resistance band pullbacks

32.11 Anterior Deltoid

32.11.1 Functions

1. Abduction of the shoulders. The anterior part acts as parallel adduction and internal rotation of the shoulders.
2. Origin:
   - Anterior and superior surfaces of the lateral 1/3 of the clavicle
   - Lateral margin and superior surfaces of the acromion
   - Lower lip of posterior border of the spine of the scapula

   Insertion:
   - Deltoid tuberosity on the lateral aspect of the body of the humerus

32.11.2 Rehabilitation Exercise

32.11.2.1 Band Frontal Lateral Raise

1. This exercise can be done by keeping a band beneath your feet or by wrapping around a pillar. Exercise done on a sitting position is effective because it prevents bounce on your back or legs (Fig. 32.46a).
2. Spread your feet on your shoulder width and forward flex your shoulder till it reaches 90° (or nose level), then slowly lower to initial position (Fig. 32.46b).
3. Do not bounce on your legs or your back while exercising and feel some resistance when returning.
4. Elbows can be slightly fixed but angles should be fixed.
5. Breathe in while lowering and breathe out while raising.

32.11.2.2 Dumbbell Frontal Raise

1. This exercise is typical for anterior deltoid strengthening. Stand on your shoulder width, slightly flex your knees and make your back straight (Fig. 32.47a) Then raise your arm till it gets parallel to the floor (Fig. 32.47b). The more you raise, the more stimulation will be done on the target muscle.
2. Pause at the top if it is possible, then slowly lower your arm. You can try one arm at a time or both arms.
3. Try to concentrate on your deltoid and avoid shrugging your neck or bouncing your body.

32.11.2.3 Arnold Press

1. This exercise can be done on a sitting or a standing position. Hold dumbbells wider than your shoulder width, and from your hand pointing to your face (Fig. 32.48b), lift up the dumbbells over your head by rotating the wrists 180° (external rotation – > internal rotation) (Fig. 32.48b).
2. Start from the elbows pointing forward and do not lift till your elbows get fully extended.
3. This exercise can give stimulus to anterior and lateral portion of the muscle.
4. It is recommended to patients with impingement syndrome.

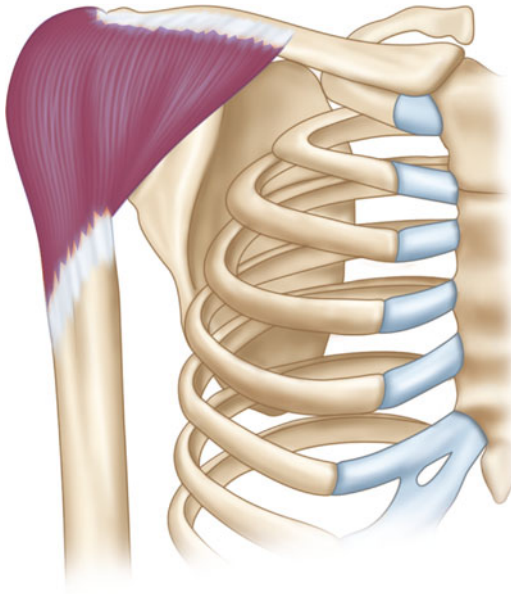

Fig. 32.45 Anterior deltoid

32.12 Posterior Deltoid

32.12.1 Functions

1. The most important muscle to abduct the shoulders. Posterior part acts as horizontal extension and external rotation and overextension of the upper arm.
2. It has a partial role for scapula stabilization.
3. Origin:
   - Anterior and superior surfaces of the lateral 1/3 of clavicle
   - Lateral margin and superior surfaces of the acromion
   - Lower lip of posterior border of the spine of the scapula

   Insertion:
   - Deltoid tuberosity on the lateral aspect of the body of the humerus

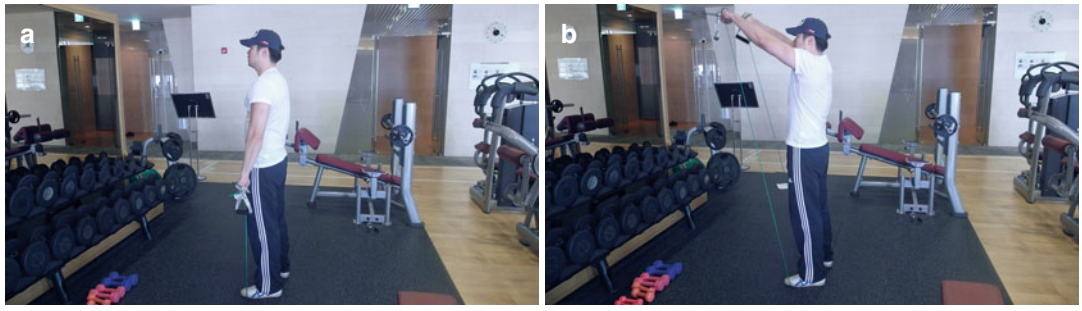

Fig. 32.46 Band frontal lateral raise

Fig. 32.47 Dumbbell frontal raise

Fig. 32.48 Arnold press

32.12.2 Rehabilitation Exercise

32.12.2.1 Band Rear Deltoid Exercise

1. Kneel down on a floor, make your back straight and fix your eyes on the floor.
2. Fix a band on a floor with one hand and hold the band with the opposite hand.
3. Breathe in and raise your arm till your shoulder level. Keep 80–90° abduction position.

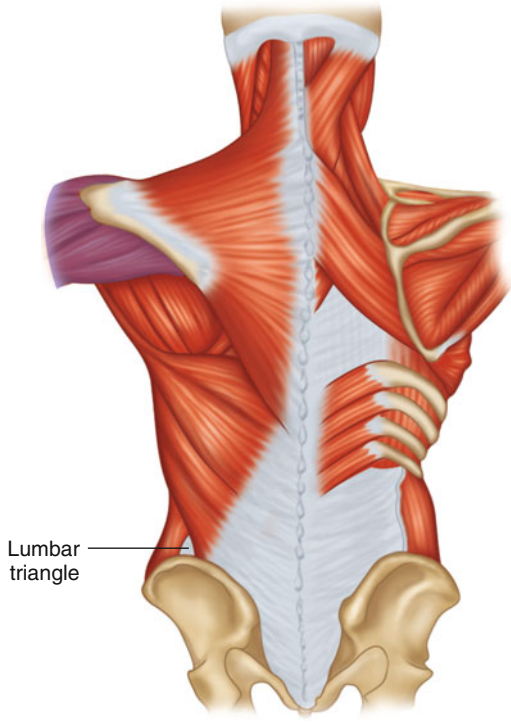

Fig. 32.49 Posterior deltoid

4. If your arm is tilted to your head, the trapezius and lateral deltoid will get more stimulus.
5. Be careful not to bend your back too much.

32.12.2.2 Bent-Over Dumbbell Raise

1. This is the most basic and unique exercise to concentrate on the posterior deltoid.
2. Bend your back till it gets parallel to the floor. While breathing in, lift your arms by making a big circle (Fig. 32.51b).
3. If your arm is tilted to your head, the trapezius and lateral deltoid will get more stimulus.
4. Posterior portion will be strengthened if your thumb is pointing to the floor.

32.12.2.3 Rear Deltoid Row

1. This exercise is to give stimulus to the posterior deltoid. Pulleys or band can be used and pull it to your eye level (Fig. 32.52a).
2. If your arm is dropped too much, the posterior deltoid will not get enough stimuli. Try to make your arms parallel (Fig. 32.52b).

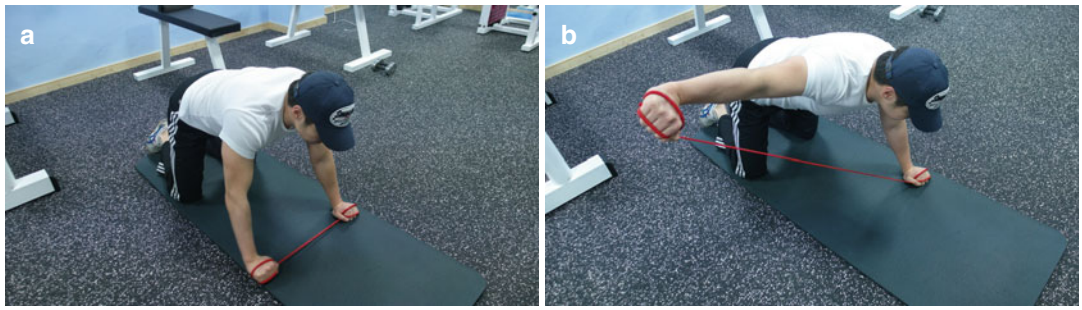

Fig. 32.50 Band rear deltoid exercise

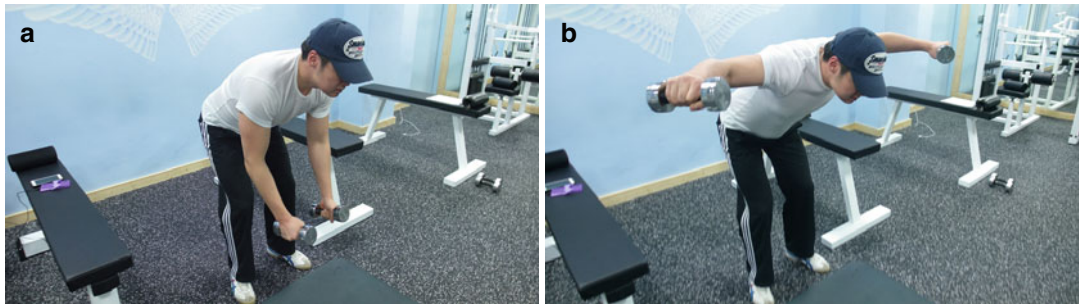

Fig. 32.51 Bent-over dumbbell raise

32.12.2.4 Seated Rear Deltoid Row

1. From a sitting position with shoulders abducted 90°, fix your eyes at the front and make your arms parallel to the floor (Fig. 32.53a). Concentrate on the posterior deltoid and slowly pull the cable and return with some resistance.
2. Make sure not to adduct your shoulders too much and bounce on your back too much.
3. Breathe out while pulling and breathe in while pushing back to initial position.

Fig. 32.52 Rear deltoid row

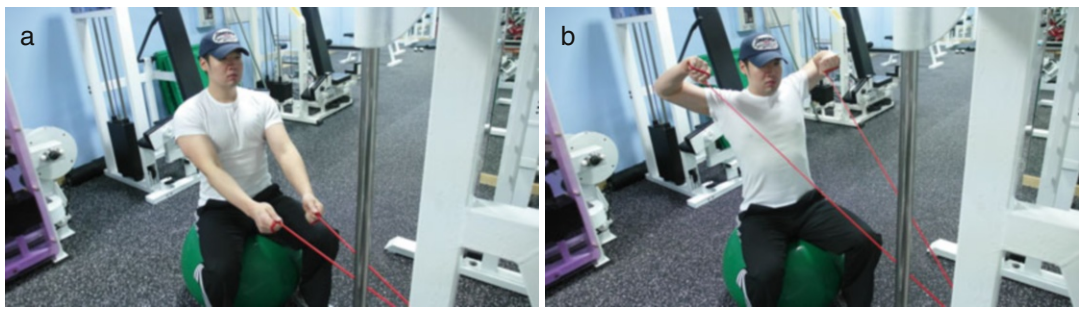

Fig. 32.53 Seated rear deltoid row

32.13 Biceps Brachii

32.13.1 Function

1. Function: forearm supination and elbow flexion
2. Origin:
   Short head
   - Apex of the coracoid process in common with coracobrachialis

   Long head
   - Supraglenoid tubercle at the upper margin of the glenoid cavity
     - 20 %: supraglenoid
     - 50 %: superior labrum
     - 30 %: mixed
3. *Insertion:*
   - *Rough posterior surface of the tuberosity of the radius (bicipital aponeurosis; lacertus fibrosus)*

32.13.2 Rehabilitation Exercise

32.13.2.1 Dumbbell Biceps Curl

1. Stand on your shoulder width and slightly bend your knees. Hold dumbbells with your palm facing forward (supinated position) (Fig. 32.55a).
2. Fix elbows beside your waist and lift up the dumbbells. Back of the wrists should point forward at the final phase of lifting (Fig. 32.55b).
3. Slowly lower the dumbbells with a feeling of resistance to your biceps. Breathe in when lifting up.
4. Do not bounce on your back. Make sure your elbows do not lean forward and keep dumbbells beside your body.
5. It can be also done with palms facing upward. This is called hammer curl, and it gives more stimuli on the lateral biceps.

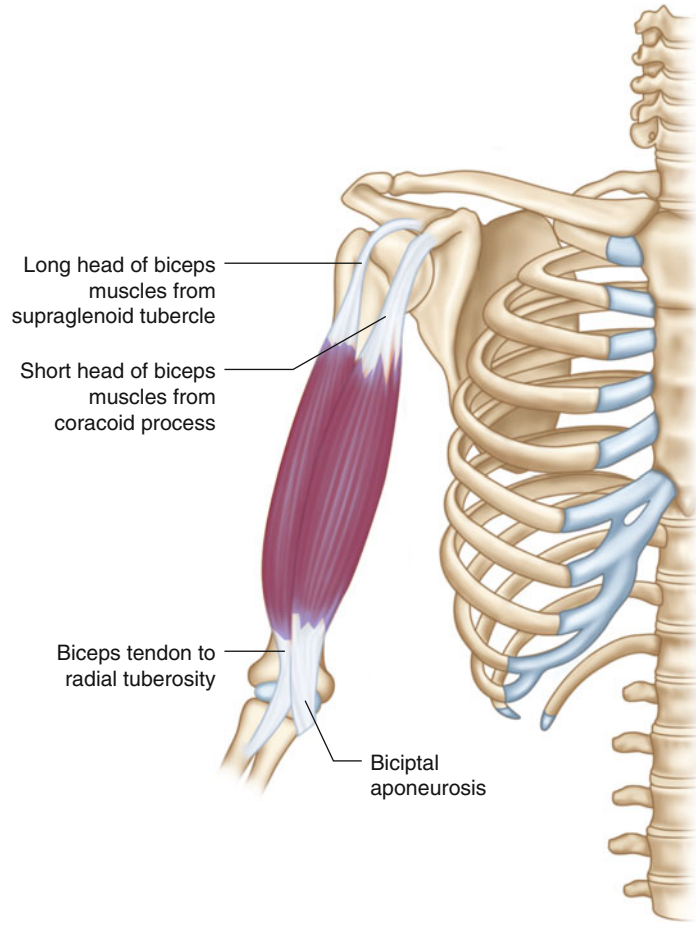

Fig. 32.54 Biceps brachii

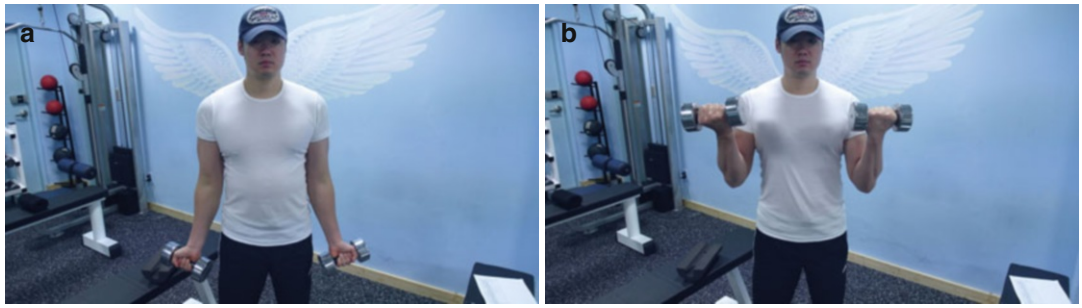

Fig. 32.55 Dumbbell biceps curl

Fig. 32.56 Band biceps curl

32.13.2.2 Band Biceps Curl

1. It is similar to biceps curl but it uses a band fix on your feet.
2. Keep your upper arms close to your body and do not make them lean forward (Fig. 32.56a).
3. When extending your arm, slowly lowering with resistance is effective to muscle stimuli (Fig. 32.56b). Since the band is fixed to both feet, it is more safe to raise the band with both hands.
4. Strength of band should be selected carefully to avoid pressure on your back. A definite stretching-squeezing should be performed.

32.13.2.3 Barbell Biceps Curl

1. Hold a barbell on your shoulder width and slowly lift up till forearm touches your biceps.
2. Like a dumbbell biceps curl exercise, affecting muscle area can be different depending on the grip.
3. Holding the barbell with a wide grip gives more stimuli to the short head of the biceps, while a short grip (short distance between your hands) gives stimuli to the long head of the biceps.

32.13.2.4 Preacher Curl

1. For the reason that you cannot bounce your body in this exercise, it gives stimulus to only the biceps. EZ-bar, barbells, or dumbbells can be used.

32.13.2.5 Concentrated Curl

1. This exercise is done with placing the elbow on the medial part of your thigh. Like a preacher's curl, elimination of bouncing and concentration on the biceps is a great benefit of this exercise. It's efficacy to raise the height of the muscle is well known.

32.14 Triceps Brachii

32.14.1 Function

1. Function: forearm extension and shoulder extension (long head)
2. Origin:
   Long head:
   - Infraglenoid tuberosity of the scapula

   Lateral head:
   - Posterior surface of the body of the humerus between the insertion of the teres minor and the proximal part of the oblique
   - Lateral border of the humerus and intermuscular septum

   Medial head:
   - Posterior surface of the body of the humerus, distal to groove for radial n.
   - Medial border of the humerus
   - Whole length of medial intermuscular septum
3. *Insertion*:
   - Posterior portion of the proximal surface of the olecranon

32.14.2 Rehabilitation Exercise

32.14.2.1 Dips

1. This exercise is effective for developing the mass of the triceps, especially around the elbows.
2. Holding on a parallel bar, fully extend your elbows. When lowering your body by flexing the elbows, try to make your body upright (Fig. 32.61b).
3. Making your body more straight will give more stimuli on the triceps, and the more you lean forward, the more stimuli will be done on the chest muscles.

Fig. 32.57 Barbell biceps curl

Fig. 32.58 Preacher curl

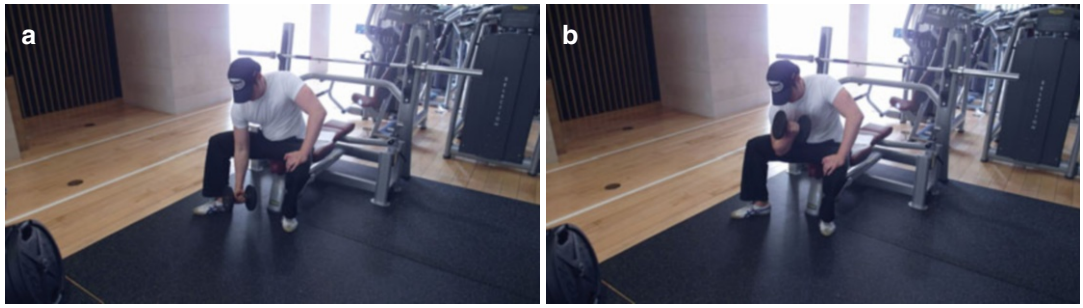

Fig. 32.59 Concentrated curl

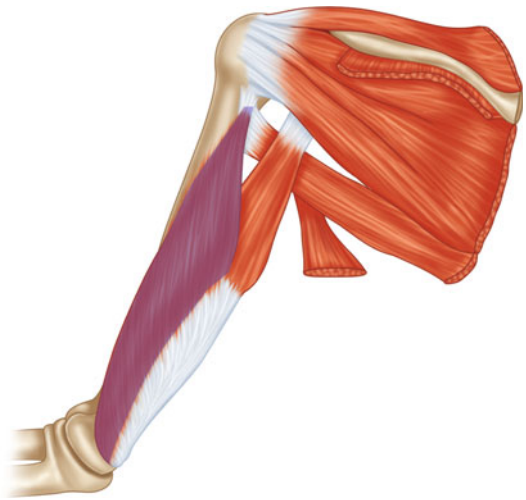

Fig. 32.60 Triceps brachii

4. Lower your body with resistance on the triceps (eccentric concentration) (Fig. 32.61a).
5. More effort can be done to this exercise by raising your body up to 3/4 level.

32.14.2.2 Dips Behind Back

1. This exercise has an advantage of performing it anywhere with a place to lean your hands. Placing your feet on the floor reduces load on this exercise. Therefore, it is recommended to beginners.
2. Lean on the back with both hands and both heels on the floor or a bar (Fig. 32.62a).
3. By flexing the elbows, lower your body as much as you can (Fig. 32.62b). To exercise the triceps, stop before full flexion.
4. By placing a weight on your thigh from a partner (or trainer), exercise strength will be increased.

32.14.2.3 Dumbbell Kick

1. *Develops upper part of the triceps.*
2. With both knees bent, place one leg in front, and the opposite hand on a low bench (to make balance) (Fig. 32.63a).
3. Hold a dumbbell and from an elbow-flexed position, extend your elbow to your shoulder level (make your forearms parallel to the floor) (Fig. 32.63b).
4. Pause for a while at extended position, then slowly lower to the initial state. You can also exercise by using a cable or a pulley.
5. To develop the triceps more, twist your arm inside the body when raising and outside when lowering.

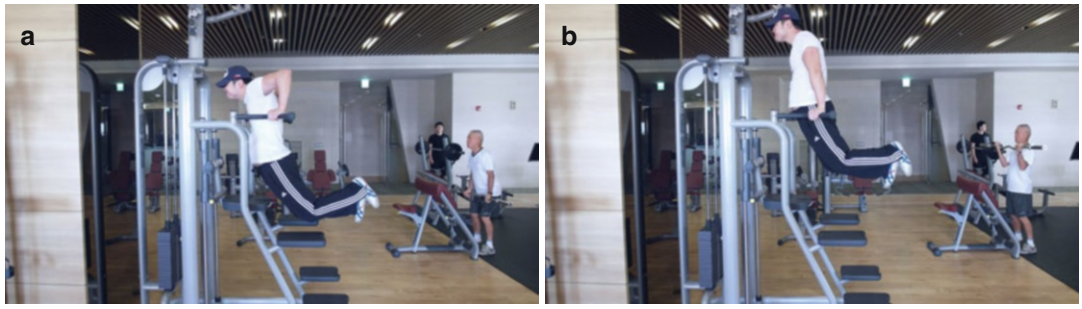

Fig. 32.61 Dips

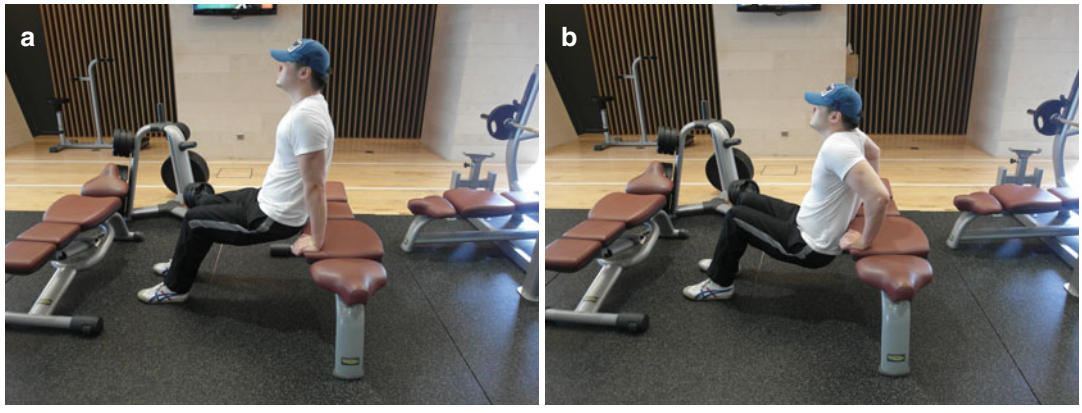

Fig. 32.62 Dips behind back

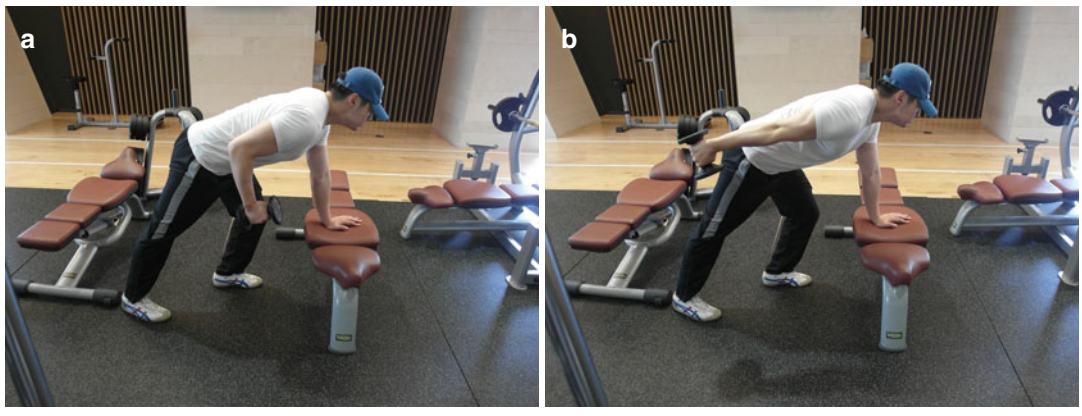

Fig. 32.63 Dumbbell kick

32.14.2.4 Triceps Cable Push-Down

1. This is a basic exercise for the triceps using entire working range of triceps.
2. Stand close to a bar and hold it with an overhand grip. Place the elbows beside your body to make it stable.
3. Press the bar and make the elbow fully extended. Feel the triceps on a maximum contraction.
4. Do not move your entire body and do not lean forward to press the bar with your weight.
5. Change can be made by grip width and stance between your body and the bar. Also underhand grip can be tried.

32.14.2.5 Triceps Extension

1. This exercises the entire triceps and separates the three heads.

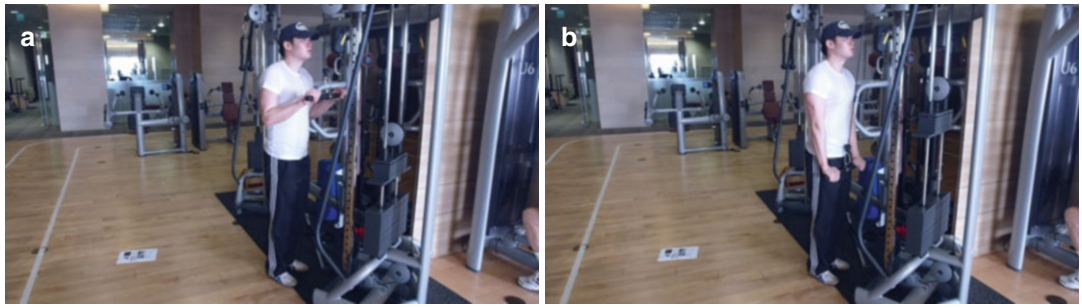

Fig. 32.64 Triceps cable push-down

Fig. 32.65 Triceps extension

Fig. 32.66 Band triceps extension

2. Hold a dumbbell with one hand; from your back extend your arm and pause near your head. Then return by lowering your arm.
3. Feel maximally stretched triceps and return by pressing the muscle.
4. The upper arm should be attached to the ear area. It is good to look at the mirror for checking the right position.

32.14.2.6 Band Triceps Extension

1. Start seated in a chair or bench with your abdomen leaning over.
2. Hold the bands in thumb down with both hands and make elbow fully extended.
3. Be care of your back is not over-bended.
4. Supported by a ball on your abdomen help lowering pressure of the spine.

32.15 Glenohumeral Joint

32.15.1 Rehabilitation Exercise

32.15.1.1 Low Row Tubing

1. Secure a tube or an elastic cable around doorknob of a door or wrap it around a pole (or a post).
2. Stand with your knees slightly bent and facing the door. Grab the handles of the tube (Fig. 32.67a).

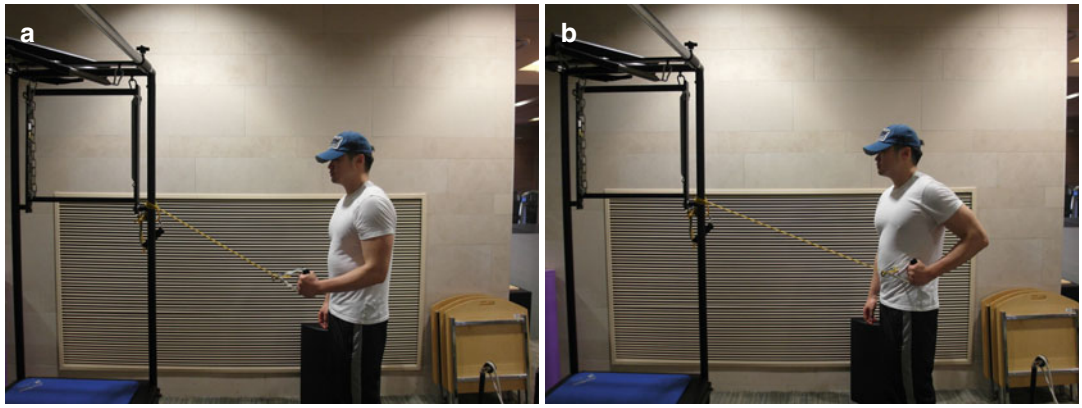

Fig. 32.67 Low row tubing

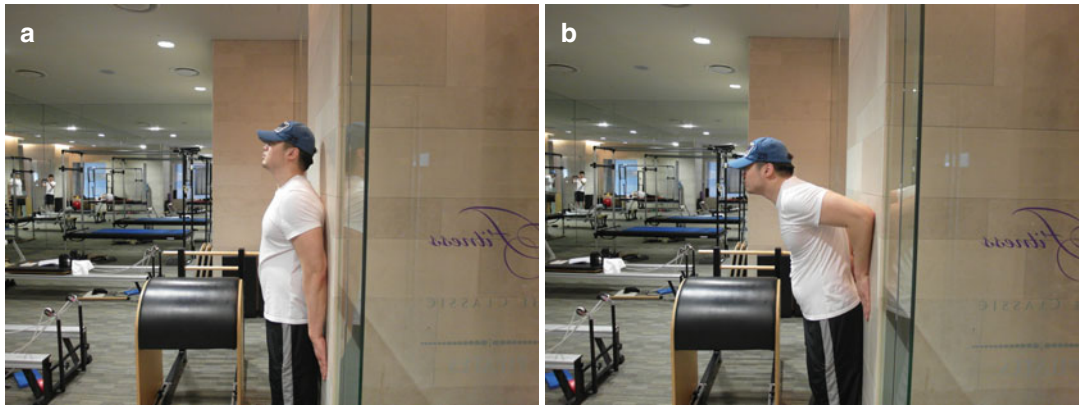

Fig. 32.68 Wall wash exercise

3. Pull from your back first with your elbows following backward. Your elbows should be at a 90° angle of flexion at the maximum pulling phase. Return slowly to the starting position (Fig. 32.67b).

*This exercise primarily strengthens the latissimus dorsi muscle and secondarily the middle trapezius and rhomboids muscles.

32.15.1.2 Wall Wash Exercise

1. Stand on your shoulder width apart with your back against a wall.
2. Step out away from the wall (about a foot width) and keep your head, hips, and back touching the wall by bending your legs. Place your hands, elbows, and forearms on the wall.
3. Breathe in and slide your hands and elbows upward until your hands are even with your shoulder level.
4. Hold this position for a few seconds and then slowly slide your arms back down the wall to the starting position.

32.16 Scapula

32.16.1 Rehabilitation Exercise

32.16.1.1 Rotation

1. Secure or tie a band to a pole or a doorknob and hold the end. Stand back until the band is stretched tight with your arm extended straight forward (Fig. 32.68a).
2. Keeping your arm extended, squeeze your scapula backward. Then draw your arm backward until it is bent at a 90° angle at the elbow (Fig. 32.68b).
3. Finally, rotate your forearm medially (pronation) until your fist points the roof while keeping 90° angle on your elbow (Fig. 32.68c).

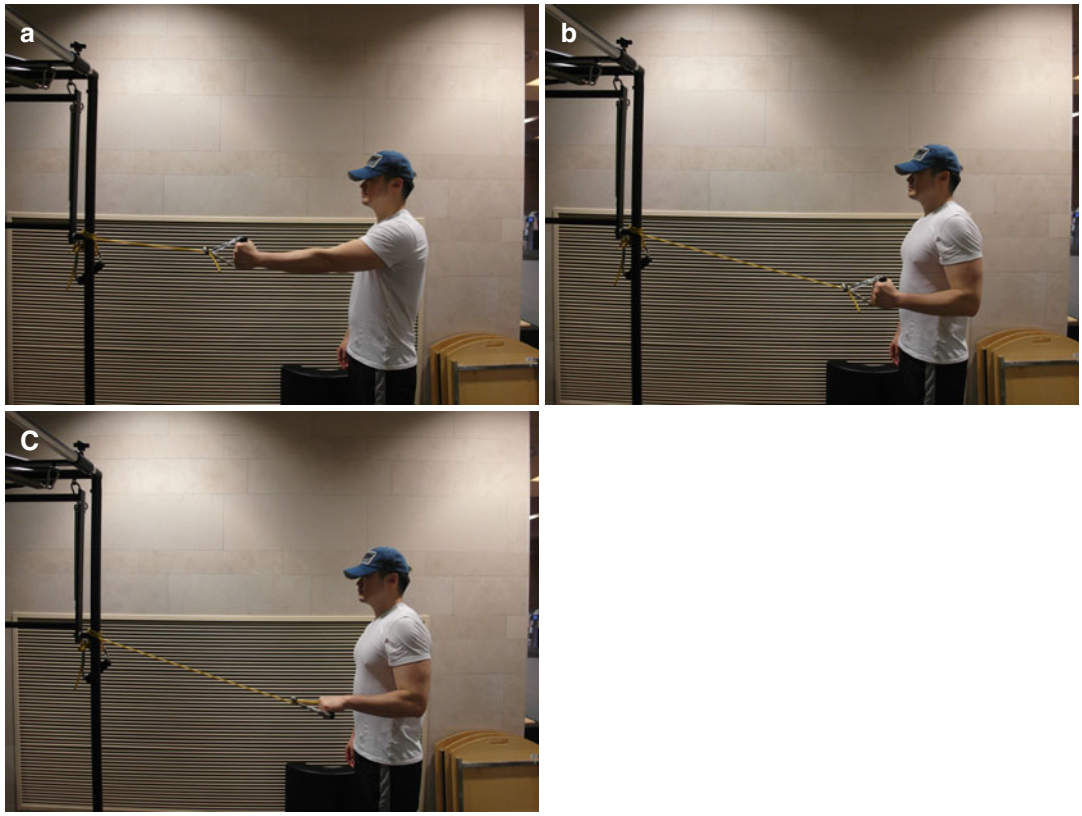

Fig. 32.69 Rotation

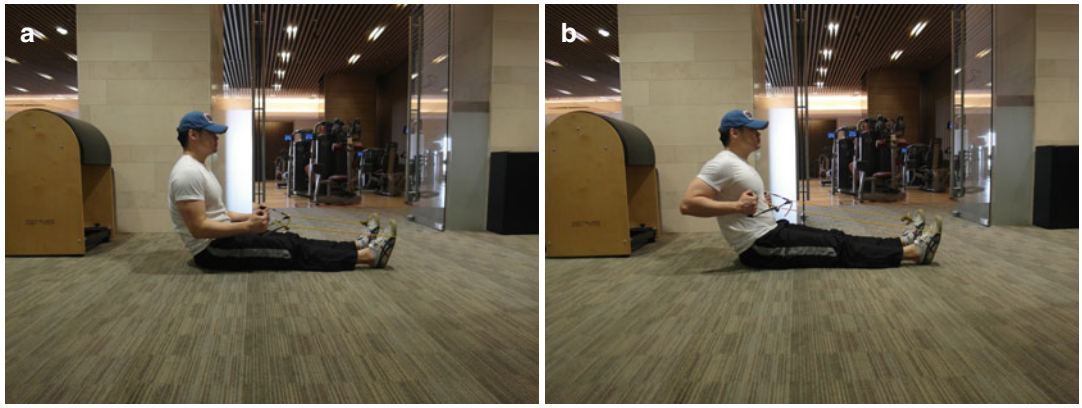

Fig. 32.70 Retraction

32.16.1.2 Retraction

1. Sit straight on a floor, wrap a band around both of your feet, keeping your feet and legs together. Make sure your back is straight during the exercise.
2. Grab the handles of the resistance band, keeping the tension on the band (Fig. 32.70a).
3. Making your body straight and still, pull your arms toward your chest (Fig. 32.70b), and then slowly return your arms to the starting position.

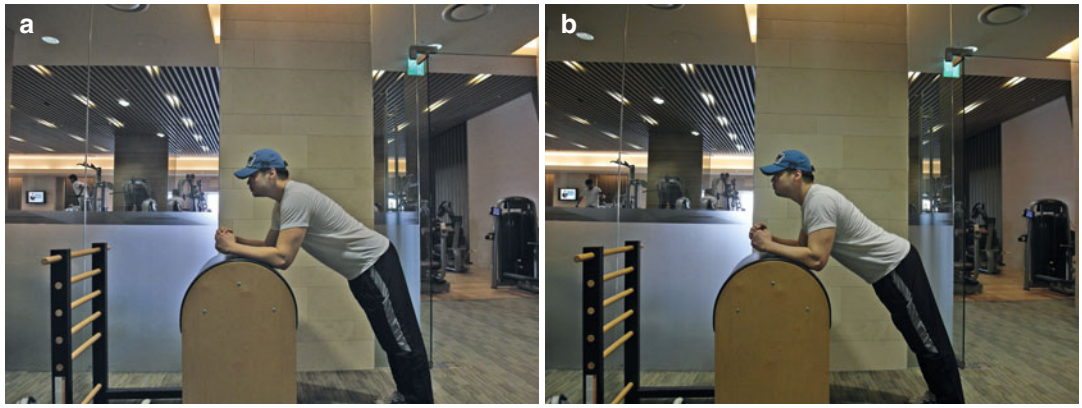

Fig. 32.71 Table retraction/protraction (table elbow push-up)

Fig. 32.72 Clock

32.16.1.3 Table Retraction/Protraction (Table Elbow Push-up)

1. Lean on a table with face down and your forearms on the table to support your body.
   Legs should be straight and elbows should be 90°(Fig. 32.71a).
2. Exercise by flexing your elbow more and lower your torso by feeling the tension on your scapula (Fig. 32.71b).
3. Pause for a while, then slowly return to your starting position. Also feel the tension on your scapula when returning.

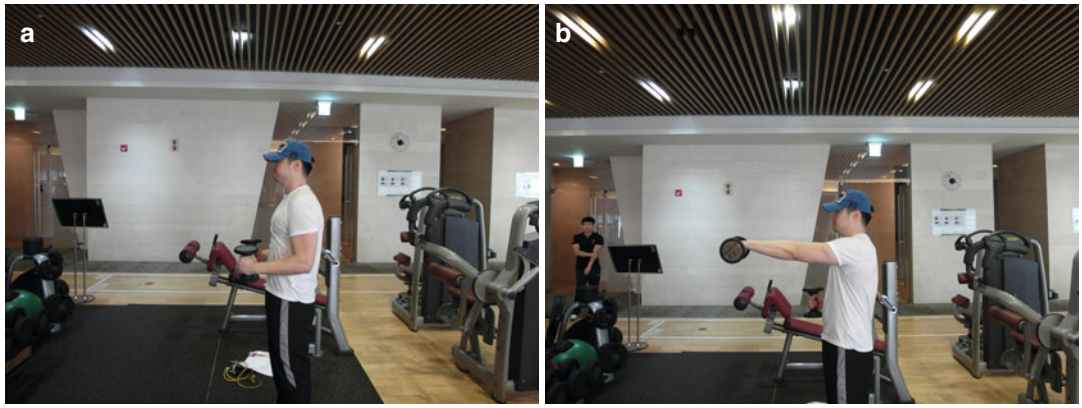

Fig. 32.73 Dumbbell punch

32.16.1.4 Clock

1. Stand with your arms extended and your palms pressing against the wall.
2. Your arms should point at the 8 o'clock position. Press firmly against the wall for 10 s. Then move your arms to the 9 o'clock position and again press against the wall for 10 s.
3. Repeat this motion for every hour (8–4 o'clock) on the clock, finishing at the 4 o'clock position. Feel the retracted scapula during the exercise.

32.16.1.5 Dumbbell Punch

Stand on your shoulder width apart with knees slightly bent. Grab a pair of dumbbells with an overhand grip and bring them up to shoulder level. Your palms should face each other (Fig. 32.73a).

Push your one arm out, rotating the dumbbell to make your palm face the floor (Fig. 32.73b). While pushing your arm out, twist your body slightly for added momentum.

Return to starting position, and repeat the punching with the opposite arm. This should be done in a continuous motion with no intervals between punches.

32.16.1.6 Push-up (Plus)

Get down on your hands and feet on the floor. Hands should be slightly wider than the shoulder width apart (Fig. 32.74a).

Your body should be straight from your ankles to your head. Have some tensions on your abdominals. Hold the tensions throughout this exercise.

Lower your body like a normal push-up until your chest nearly touches the floor. Pause, then return back to the starting position as quickly as you can (Fig. 32.74b).

When you have returned to the starting position, push your upper back toward the ceiling. The movement is very minimal, but-but force your body as much as you can (Fig. 32.74c).

32.17 Gluteus Maximus Muscle

32.17.1 Anatomy

1. Origin:
   - Posterior gluteal line of the ilium
   - Iliac crest
   - Immediately superior and dorsal to the crest
   - Posterior surface of the lower part of the sacrum and side of the coccyx
   - Aponeurosis of the erector spine
   - Fascia (gluteal aponeurosis) covering the gluteus medius

Insertion:

- Iliotibial band of the fascia lata
- Deeper fibers of the lower portion
- Gluteal tuberosity between the vastus lateralis and adductor magnus

32.17.2 Function

External rotation of the hip
Extension of the hip
Abduction of the thigh

Fig. 32.74 Push-up (Plus)

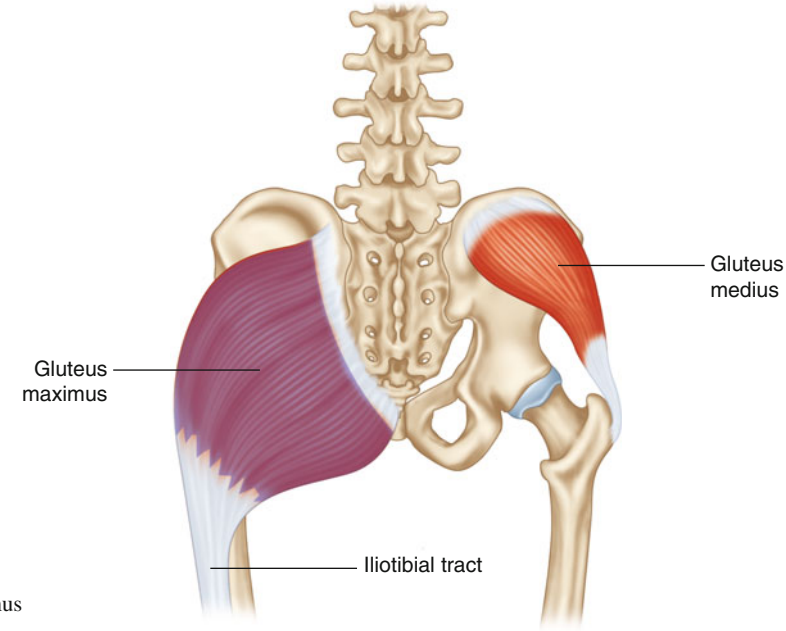

Fig. 32.75 Gluteus maximus muscle

32.17.3 Rehabilitation Exercise

32.17.3.1 Bridge Exercise

1. Lie on your back with your knees bent and your feet on the floor. Place your arms at your sides, palms facing down (Fig. 32.76a).
2. Draw your abdominal muscles in and push your pelvis upward toward the roof.
3. With both shoulder blades(scapula) in full contact with the floor, breathe in, then breathe out and lift your hips up by contracting your gluteal muscles (Fig. 32.76b).
4. Simultaneously push your heels against the floor to stabilize your legs. Your body should be straight from your shoulders to your knees. Hold the position, lower with control, and repeat.

32.17.3.2 Gluteus Kickback

1. Kneel down on a floor with your one leg behind your body slightly raised (Fig. 32.77a).
2. Elevate your leg as high as you can and slowly return to its starting position (Fig. 32.77b).
3. Exercise as the same instruction with your other leg and repeat the steps.

32.17.3.3 Squat

1. This exercise starts from a standing position (Fig. 32.78a). Weights (barbells) can be used.
2. Move your hips back and bend your knees (Fig. 32.78b). Depth of your movement will decide the load of the exercise.

**This exercise strengthens the gluteus maximus, quadriceps, and hamstring muscles.*

Fig. 32.76 Bridge exercise

Fig. 32.77 Gluteus kickback

32.18 Gluteus Medius Muscle

32.18.1 Anatomy

1. Origin:
   - Outer surface of the ilium between the iliac crest and posterior gluteal line
   - Anterior gluteal line ventrally
   - Gluteal aponeurosis covering its outer surface
2. Insertion:
   - Oblique ridge on lateral surface of the greater trochanter

32.18.2 Function

Abduction of the hip
Medial rotation of the thigh

Fig. 32.78 Squat

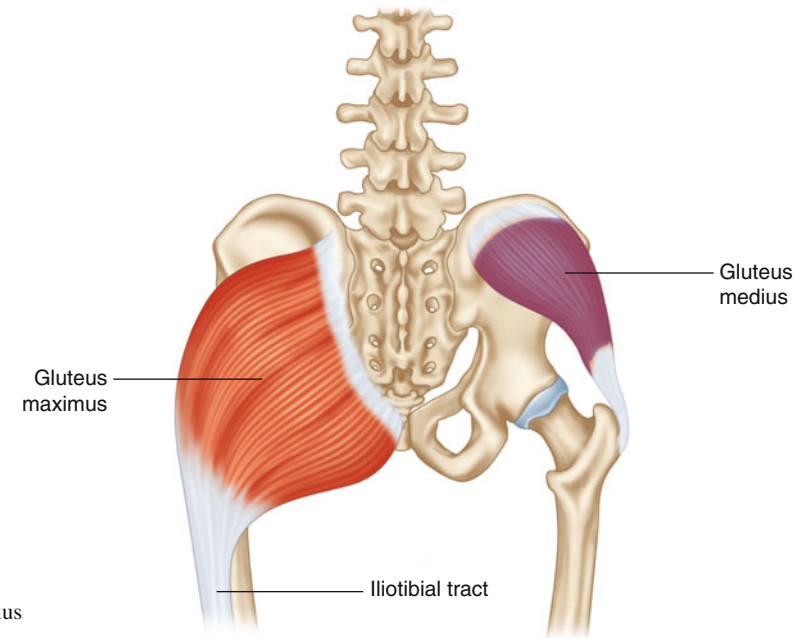

Fig. 32.79 Gluteus medius muscle

32.18.3 Rehabilitation Exercise

32.18.3.1 Side-Lying Abduction/ External Rotation

1. Lie down on your side, keep both knees bent, and flex the hips to 30° (Fig. 32.80a).
2. While keeping both your heels touching and your pelvis still, open your knees(hip abduction) by contracting your gluteus medius (Fig. 32.80b).
3. Repeat the exercise slowly 10–15 times and switch sides.

32.18.3.2 Side-Lying Leg Raises

1. Lie down on your side, keep both knees and hips extended (Fig. 32.81a).
2. Lift up your leg slowly while keeping your knee extended. Feel the contraction on your gluteus medius (Fig. 32.81b).
3. Repeat the exercise slowly 10–15 times and switch sides.

32.18.3.3 Forward Lunge

1. Stand on your shoulder width apart and hands on your waist. Look straight ahead and make your back straight (Fig. 32.82a).
2. Take a large step forward landing on heel first. The step should be long enough for the heel of the back foot to lift off the ground.
3. Lower yourself, while breathing in, until your legs are at a 90° angle with keeping your weight on forward heel (Fig. 32.82b).
4. Raise yourself up to the starting position, through the heel on the forward foot, while breathing out.

**This exercise strengthens the gluteus medius, quadriceps, and hamstring muscles.*

32.18.3.4 Lateral Tube Walk

1. Prepare a resistance band, place it just above each knee and wrap it around both your legs.
2. Stand on your shoulder width apart. Slightly bend your knees and make your back straight.

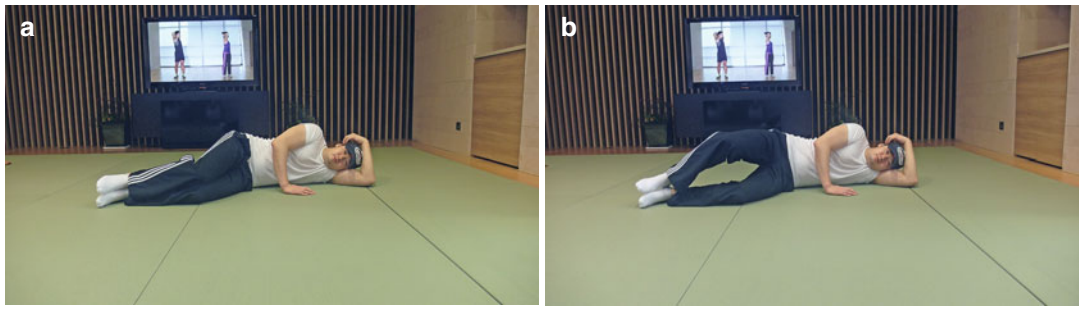

Fig. 32.80 Side-lying abduction/external rotation

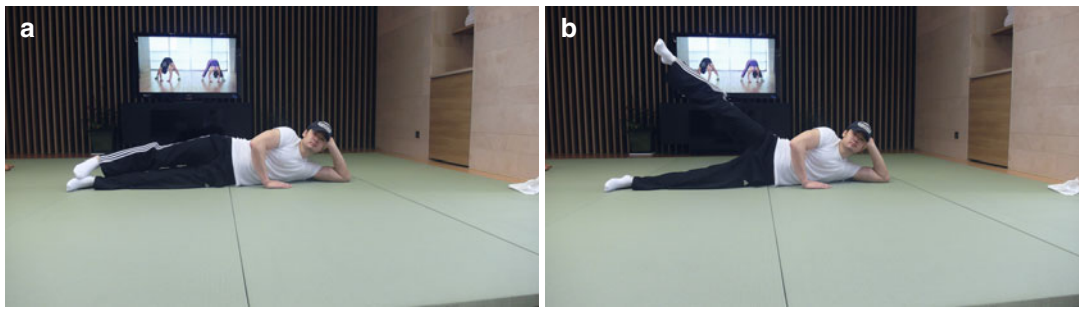

Fig. 32.81 Side-lying leg raises

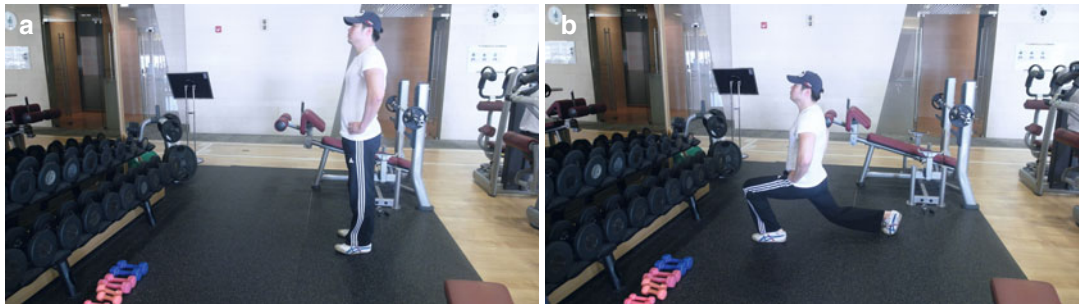

Fig. 32.82 Forward lunge

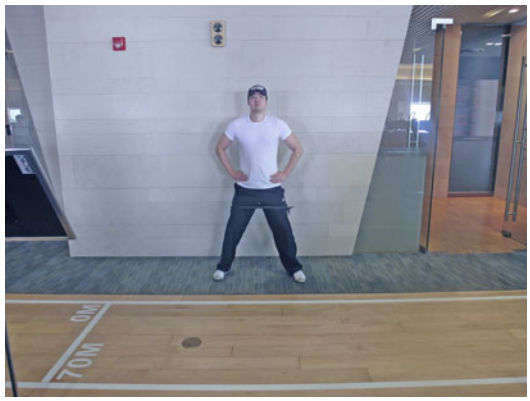

Fig. 32.83 Lateral tube walk

3. Step your right foot out to the side about shoulder widths apart. Then, step your left foot inward until your feet are again shoulder width apart.
4. Your knees should remain aligned over your feet during the exercise. Feel your gluteus muscle contract during the exercise.

32.19 Quadriceps Muscle

32.19.1 Anatomy

Rectus Femoris

Origin:

- Anterior or straight head: anterior inferior iliac spine
- Posterior or reflected head: a groove above the posterior brim of the acetabulum

Insertion:

- Base of the patella

Vastus Lateralis

Origin:

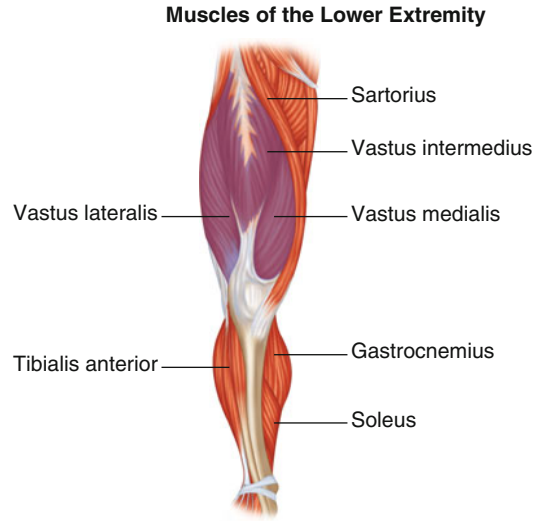

Fig. 32.84 Quadriceps muscle

- Broad aponeurosis from the proximal part of the intertrochanteric line
- Anterior and inferior borders of the greater trochanter
- Lateral lip of the gluteal tuberosity
- Proximal half of the lateral lip of the linea aspera
- Tendon of the gluteus maximus
- Lateral intermuscular septum between vastus lateralis and short head of the biceps femoris

Insertion:

- Lateral part of the patella
- A tendinous expansion to the capsule of the knee joint

Vastus Medialis

Origin:

- Lower half of the intertrochanteric line
- Medial lip of the linea aspera

- Upper part of the medial supracondylar line
- Tendons of the adductor longus and adductor magnus
- Medial intermuscular septum

Insertion:

- Medial border of the patella.
- Quadriceps femoris tendon.
- Expansion of the aponeurosis is sent to the capsule of the knee.

Vastus Intermedius

Origin:

- Anterior and lateral surface of the upper 2/3 of the body of the femur
- Lower part of the lateral intermuscular septum

Insertion:

- Aponeurosis on the anterior surface of the muscle which forms the deep part of the quadriceps femoris

32.19.2 Function

Knee extension
Hip flexion (rectus femoris only)

32.19.3 Rehabilitation Exercise

32.19.3.1 Front Squat

1. This exercise strengthens the gluteus maximus, quadriceps, and hamstring muscles.
2. This exercise starts from a standing position. The weight (usually a barbell) is held in front of the body (Fig. 32.85a).
3. Move your hips back and bend your knees (Fig. 32.85b). Depth of your movement will decide the load of the exercise.

32.19.3.2 Lunge

1. Stand with your shoulder width apart and hands on your waist. Look straight ahead and make your back straight (Fig. 32.86a).

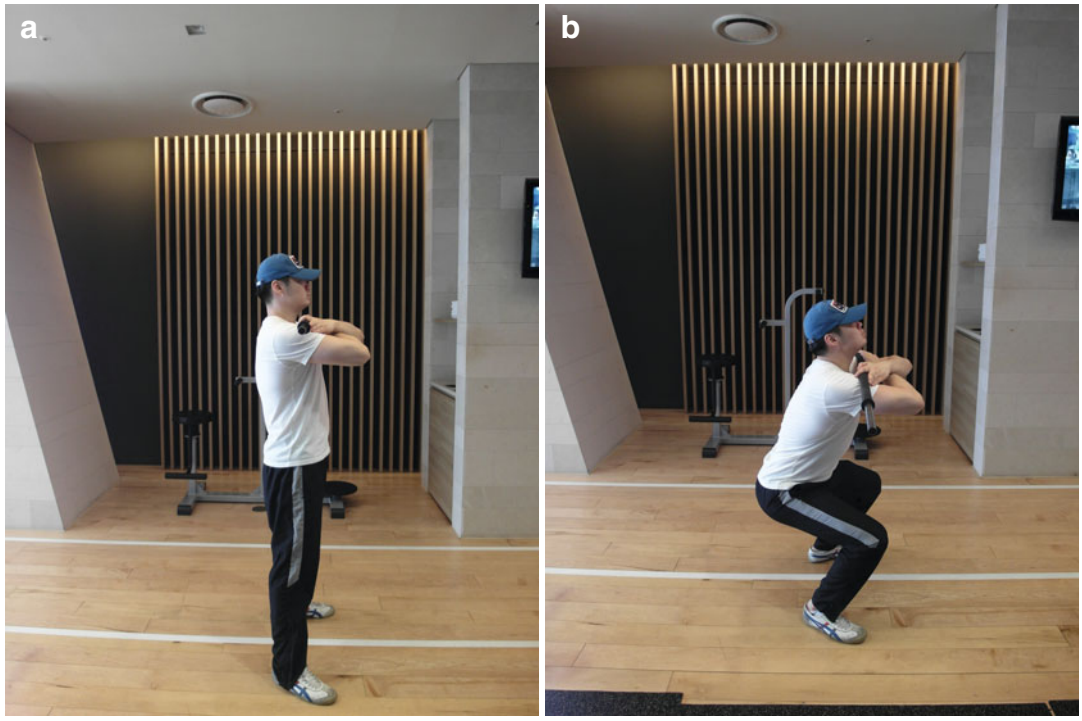

Fig. 32.85 Front squat

2. Take a large step forward landing on heel first. The step should be long enough for the heel of the back foot to lift off the ground.
3. Lower yourself, while breathing in, until your legs are at a 90° angle with keeping your weight on forward heel (Fig. 32.86b).
4. Raise yourself up to the starting position, through the heel on the forward foot, while breathing out.

**This exercise strengthens the gluteus medius, quadriceps, and hamstring muscles.*

32.19.3.3 Leg Extension

1. Sit on the machine with your legs under the pad and hold the side bars. Make sure that your knees are bent 90° angle (Fig. 32.87a).
2. Using your quadriceps, extend your legs to the maximum as you breathe out. During the exercise, the rest of the body should remain still on the seat. Pause for a few seconds on the contracted position (Fig. 32.87b).
3. Slowly return back to the original position as you breathe in. Do not go past the 90° angle limit on your knees.

32.19.3.4 Quadriceps Over Fulcrum

1. Start this exercise by lying on your back with a rolled towel under your knee and your knee relaxed (Fig. 32.88a).
2. Slowly straighten your knee as far as possible by tightening the quadriceps (Fig. 32.88b)
3. Hold for few seconds then slowly lower back down to original state.

32.20 Hamstring Muscle

32.20.1 Anatomy

Biceps Femoris
Origin:
Long head:

Fig. 32.86 Lunge

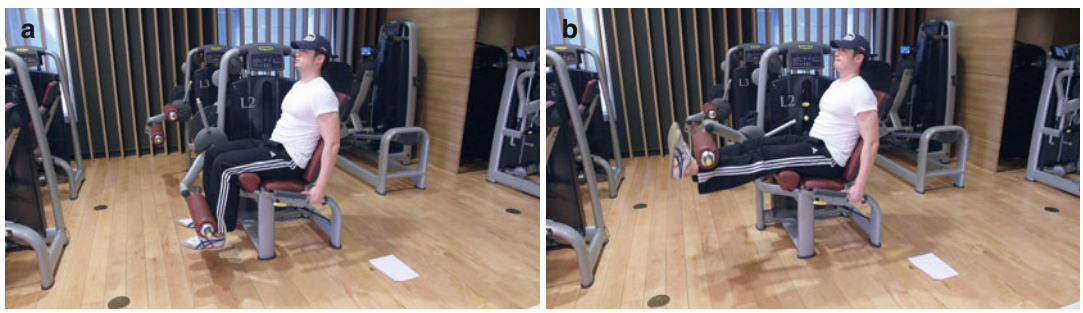

Fig. 32.87 Leg extension

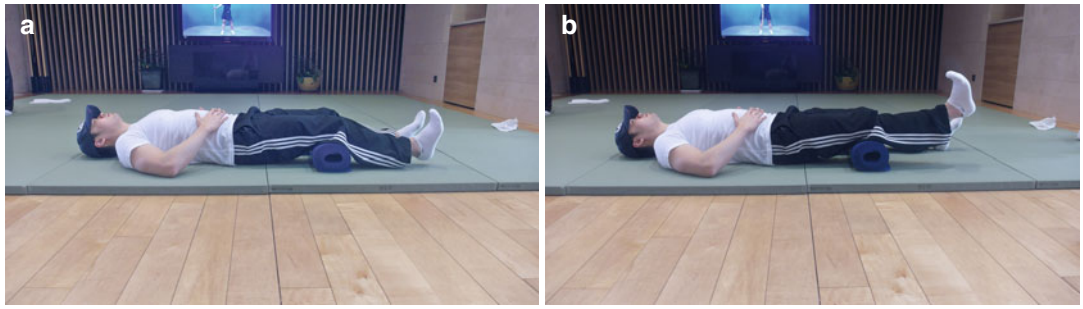

Fig. 32.88 Quadriceps over fulcrum

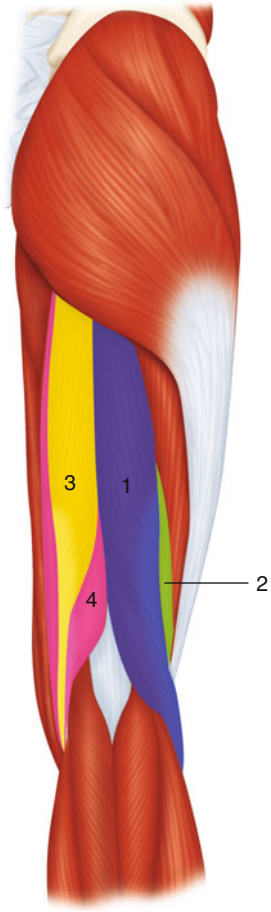

Fig. 32.89 Hamstring muscle

- Inferomedial impression on the posterior part of the ischial tuberosity
- Lower part of the sacrotuberous ligament

Short head:

- Lateral lip of the linea aspera of the femur between the adductor magnus and vastus lateralis
- Lateral intermuscular septum

Insertion:

- Lateral aspect of the head of the fibula
- Small tendinous slip, which attaches to lateral condyle of the tibia
- Divides into two portions
- Embraces fibular collateral ligament of the knee joint

Semitendinosus

Origin:

- Inferomedial impression of the ischial tuberosity
- Aponeurosis that connects the adjacent surfaces of two muscles for an extent of about 7.5 cm from their origin

Insertion:

- Proximal part of medial surface of the body of the tibia, nearly as far anterior as its anterior crest

Semimembranosus

Origin:

- Upper and lateral impressions on ischial tuberosity adjacent to the origin of the biceps femoris and semitendinosus

Insertion:

- Horizontal groove on postmedial aspect of the medial condyle of the tibia

  - Posterior aspect of the lateral condyle of the femur, forming part of the oblique popliteal ligament of the knee
  - Posterior horn of the medial meniscus and posterior capsule
  - Anterior and deep head: flare of the tibial condyle
  - Direct head: tubercle of the posterior aspect of the medial condyle of the tibia
  - Distal head: fibrous expansion of the popliteus
- Fibrous expansion

32.20.2 Function

Semitendinosus and semimembranosus extend the hip when the trunk is fixed; they also flex the knee and medially (inwardly) rotate the lower leg when the knee is bent.

The long head of the biceps femoris extends the hip as when beginning to walk; both short and long heads flex the knee and laterally (outwardly) rotate the lower leg when the knee is bent.

32.20.3 Rehabilitation Exercise

32.20.3.1 Straight Leg Deadlift

1. Hold a barbell using an overhand grip. Stand on your shoulder width apart (or narrower) and keep your body straight. The knees should be slightly bent (Fig. 32.90a).
2. While breathing in, lower the barbell by bending at your hips while keeping your back straight (Fig. 32.90b).
3. While breathing out, return to initial position by extending your hips.

32.20.3.2 Band Hamstring Curl

1. Hold a band with both your hands and place the band on your foot.
2. Kneel down with both arms on the floor (Fig. 32.91a).
3. As you breathe out, curl your leg up (Fig. 32.91b). Stay still for a few seconds, then slowly return to initial position.

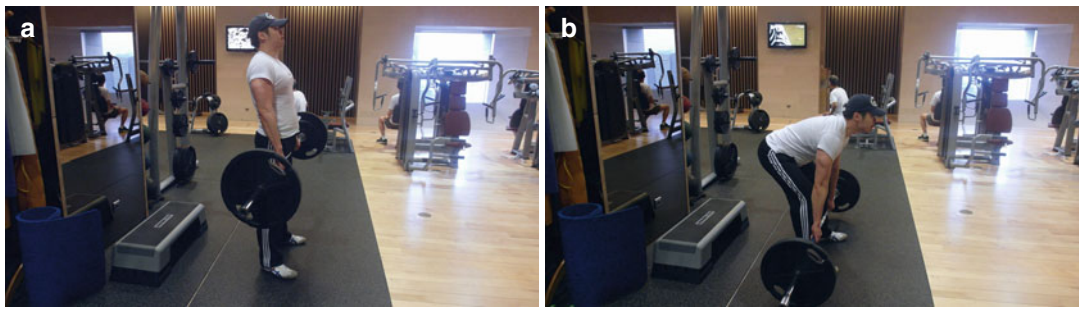

Fig. 32.90 Straight leg deadlift

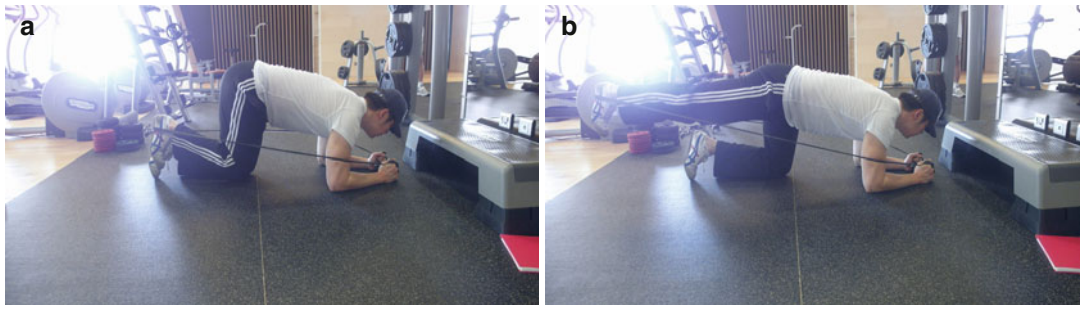

Fig. 32.91 Band hamstring curl

32.21 Hip and Trunk

32.21.1 Rehabilitation Exercise

32.21.1.1 Step

1. Stand just in front of the step, with your feet about shoulder width apart. You can place your arms down by your sides or on your hips (Fig. 32.92a).
2. Step up onto the step with one leg in a steady and controlled motion (Fig. 32.92b), pulling yourself up onto the step (Fig. 32.92c). Pause for a while with both feet up on the step.
3. Step down off the step with your opposite foot and then follow with the other foot, returning to the starting position.
   Repeat this action, alternating the leading leg each time.

32.21.1.2 Squat

1. This exercise starts from a standing position (Fig. 32.93a). Weights (barbells) can be used.
2. Move your hips back and bend your knees (Fig. 32.93b). Depth of your movement will decide the load of the exercise.

**This exercise strengthens the gluteus maximus, quadriceps, and hamstring muscles.*

Primary muscle – quadriceps
Assisting muscle – gluteus maximus
Stabilizing muscle – hamstring

32.21.1.3 Hip Extension (Using Band)

1. Tie or secure one end of the band to the lower portion of a post and attach the other to one ankle (Fig. 32.94a).
2. Facing the attachment point of the band, make balance and stabilize yourself.

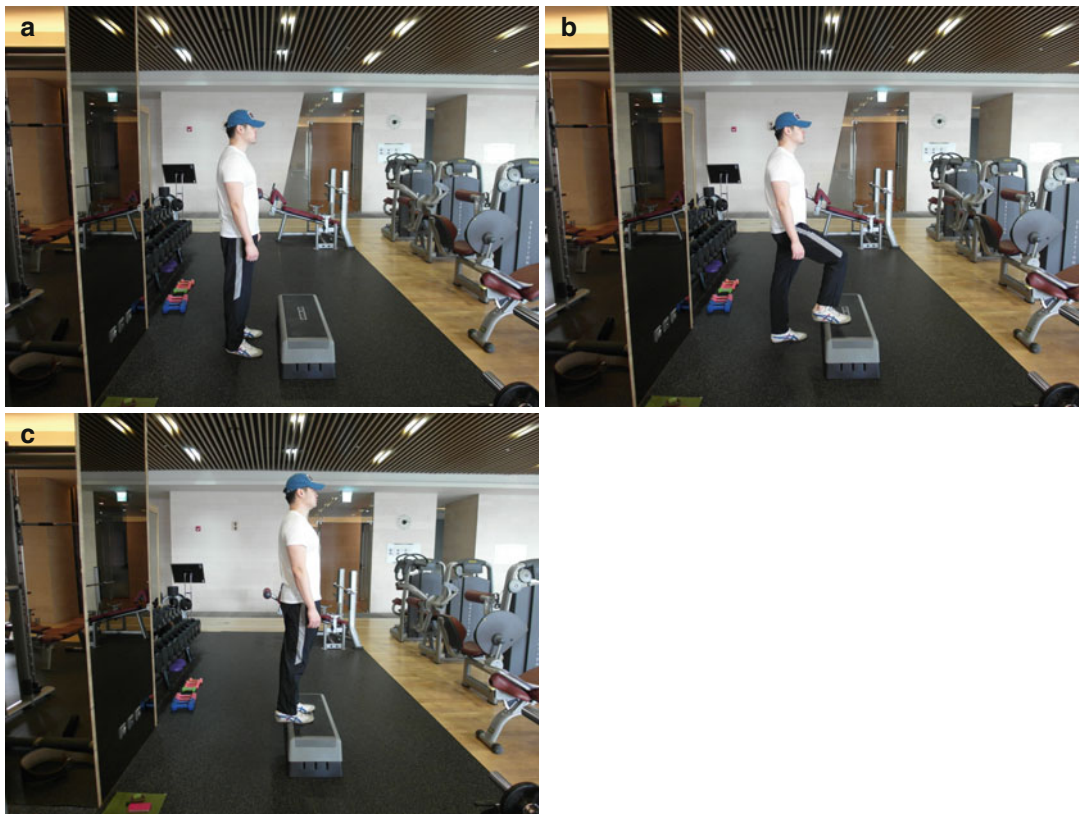

Fig. 32.92 Step

3. Keeping your head and your chest straight up, move the resisted leg back as far as you can while keeping the knee straight (Fig. 32.94b).
4. Return the leg slowly to the starting position.

32.21.1.4 Trunk Extension

1. Start by lying face down on a floor with your body and legs straight and your arms forward (Fig. 32.95a).
2. Slowly raise your chest, shoulders, and arms up toward the roof as high as you can, keeping your toes in contact with the floor at all times (Fig. 32.95b).
3. Then, slowly lower your chest, shoulders, and arms down to the floor, but do not rest on the floor.
4. Make sure to keep some muscle tension throughout your back during the entire exercise.

Fig. 32.93 Squat

Fig. 32.94 Hip extension (using band)

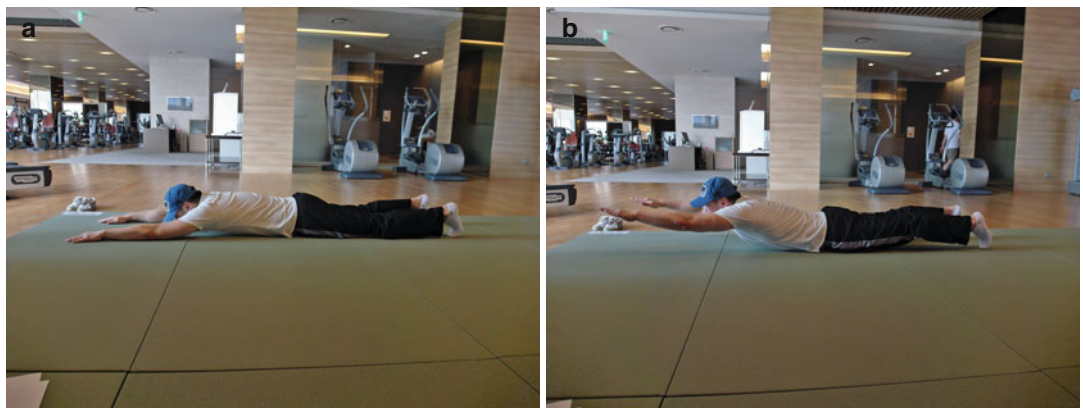

Fig. 32.95 Trunk extension

32.22 Cosmetic Upper Arm Exercise

32.22.1 Rehabilitation Exercise

32.22.1.1 Triceps Extension

1. Stand on your shoulder width apart with a dumbbell held by one hand. Slowly lift it over your head till your arm is fully extended (Fig. 32.96a).
2. Make your upper arms close to your head. While breathing in, lower the dumbbell behind your head until your forearms touch your biceps. The upper arms should remain still and only the elbows should move (Fig. 32.96b).
3. While breathing out, return back to the starting position by using the triceps to raise the dumbbell.

32.22.1.2 Lateral Stretching

1. Stand on your shoulder width apart with your knees slightly bent.
2. Place your hand on your same side hip to support the spine (Fig. 32.97a).
3. Raise your opposite arm and place your hand behind your head. Keep it there as you incline your upper body sideways (Fig. 32.97b).
4. Make sure to keep your weight evenly distributed between both legs.

32.23 Cosmetic Exercise

32.23.1 Foot Break

1. Sit on the floor with your knees extended. Place both hands on the floor beside your body.

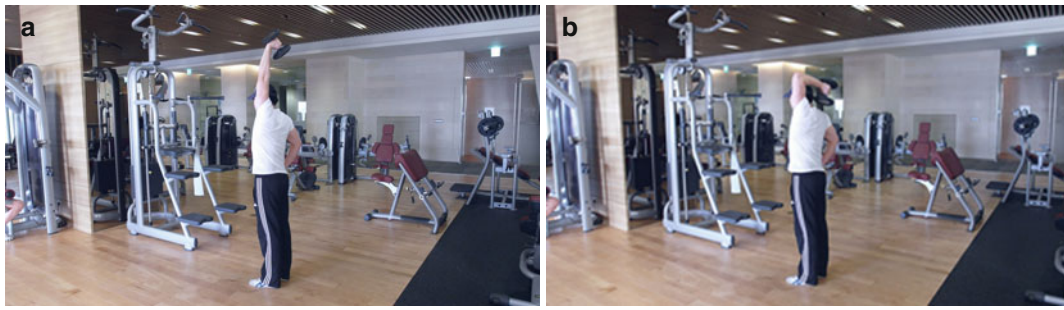

Fig. 32.96 Triceps extension

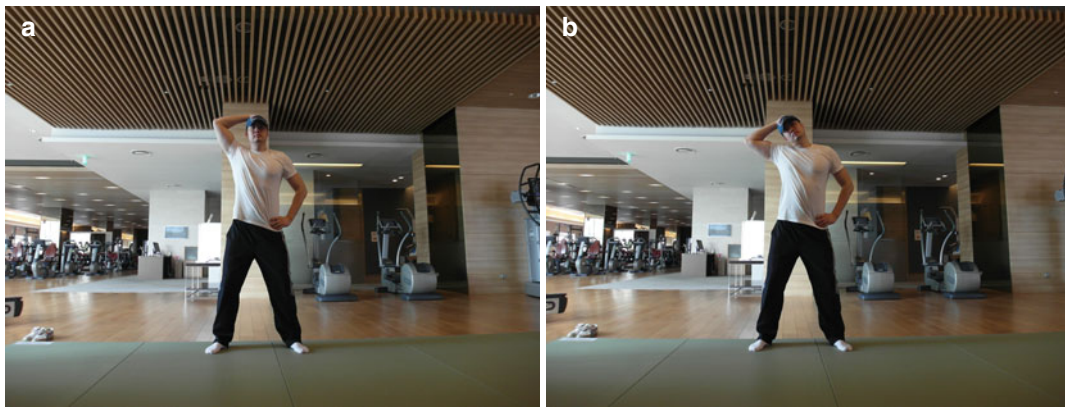

Fig. 32.97 Lateral stretching

2. Push your toes forward as if your stepping on a brake of a car (Fig. 32.98b).
3. Then pull your toes toward your body for 2 s (Fig. 32.98a).
   - Repeat by contracting and relaxing your calf muscles to ease muscles on your calf.

   This exercise is effective for stretching and relaxing your calf muscles.

32.23.2 Swing Chair

1. Stand on your shoulder width apart with knees slightly bent.
2. Fix your torso, lift up your heels, and make balance with your weight on your toes (Fig. 32.99a).
3. Then lift your toes to make balance with your heels (Fig. 32.99b).
   - This exercise is for making smooth leg lines by contracting and relaxing your calf muscles.

   Placing your buttocks behind your feet will make balancing much more easier.

32.23.3 Sky Bicycle

1. Lie down on a floor with both legs pointing the ceiling. Place your hands on your buttock or your waist with upper arms on the floor to make balance.
2. Rotate and step on the air like riding a bicycle.
   - It can be easily done anywhere, which is an advantage of this exercise.

32.23.4 Standing Bending

1. Stand with twice the shoulder width apart and spread your arms straight on your shoulder level. Knees should be fully extended for maximal stretching (Fig. 32.101a).
2. While breathing in and out, slowly bend your torso 90° (Fig. 32.101b).
3. Exhale and hold your heel with both hands (Fig. 32.101c). Then, bend your elbows, relax your shoulders, and extend your upper body with holding your breath.

Fig. 32.98 Foot break

Fig. 32.99 Swing chair

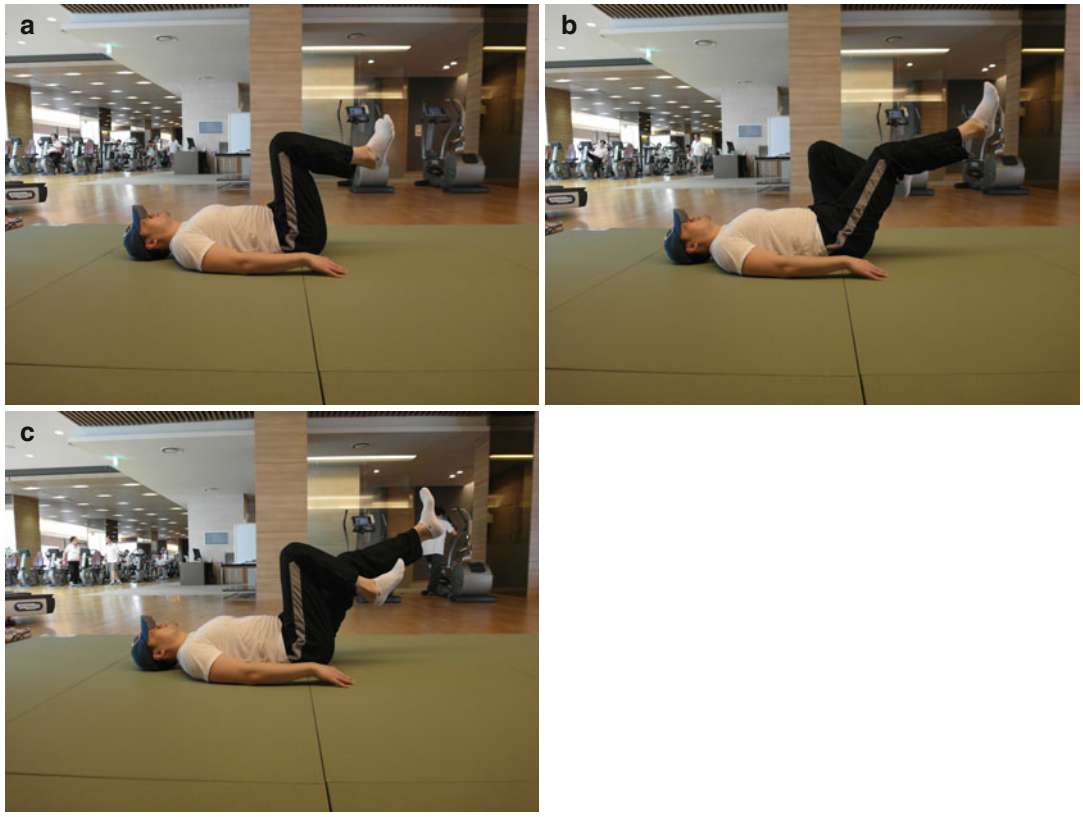

Fig. 32.100 Sky bicycle

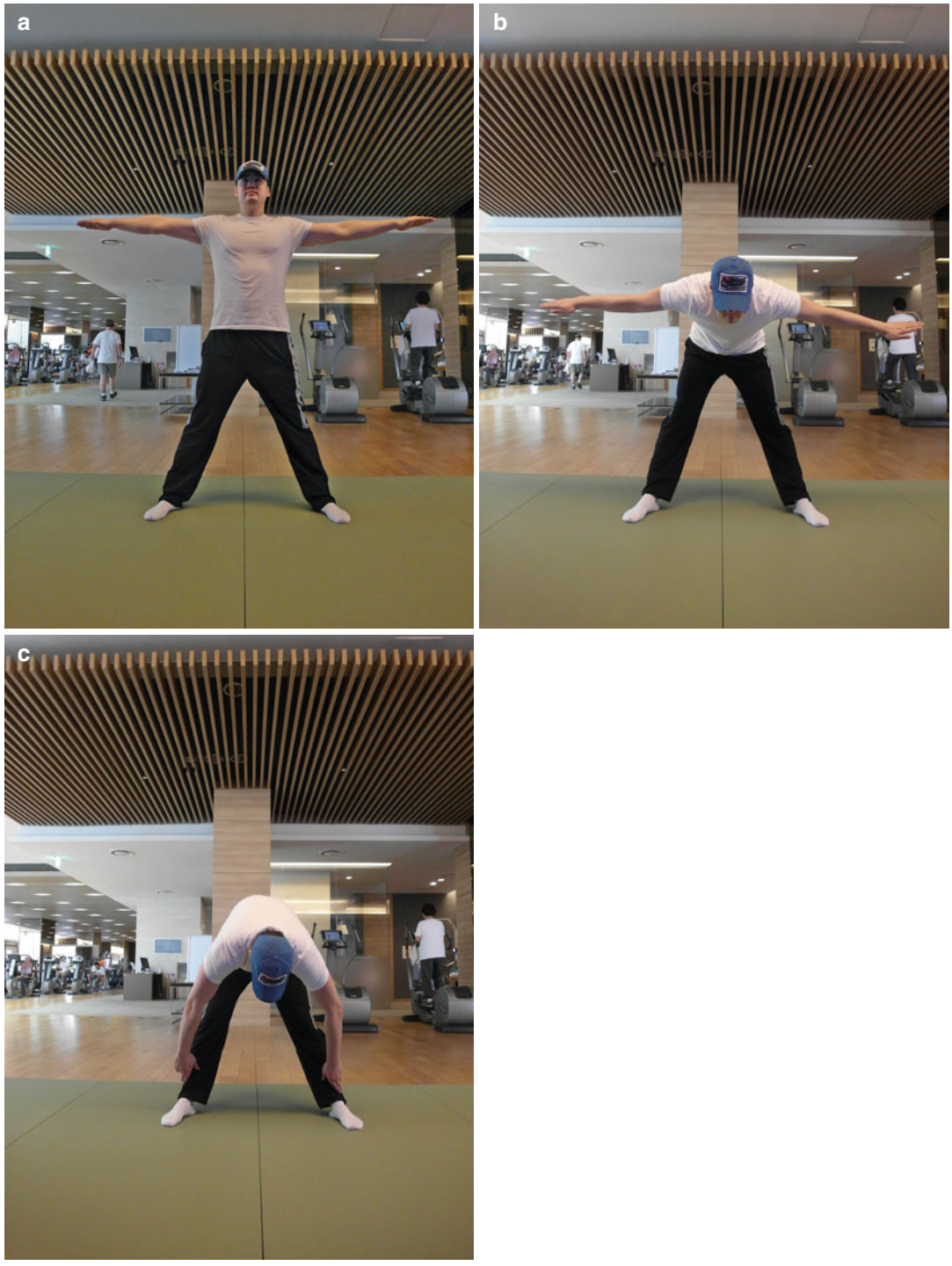

Fig. 32.101 Standing bending

Rehabilitation: Part III. Throwing Athletes

33

Jin-Young PARK and Jae-Hyung Lee

33.1 Physical Characteristics for Overhead Athletes

Overhead athletes are the players with unique and complicated movements. Thus, the repetitive motions involved with the throwing of the ball cause injuries in the shoulder joints and expose the adjacent tissues to extreme conditions. Also, overhead arm motions create excessive stress during the last phase of the pitching motion. During this time, the angular velocity (rotation angle per second) of the ball during the pitch almost reaches 7,000°/s (approximately 19.5 rotation in 1 s) and the act of rotation exerts a force equivalent to the body mass of the pitcher to the anterior shoulder joint and 1.5 times the body mass in stress. These issues occur in other sports such as football, softball, and tennis.

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It is called sports medicine which involves preventing damage and curing injuries of people associated with sports. Shoulder joint damage occurs from various causes, including muscle fatigue, muscle weakness, unbalanced power, decreased range of motion, weakness in flexibility of soft tissue, change in pitching mechanism, and degenerated static stability.

33.1.1 Physical Examination

Pitchers who consistently utilize overhead arm motions have unique different body characteristics, such as limitations in the movement of shoulder joints, scapula positions, laxity of joints, strength of muscles, and the development of proprioception. It should be noted that these unique characteristics should be considered as adaptive mechanisms that improve further performance.

33.1.2 Discomfort for Athletes

Pitchers who sustained injury from overhead arm motions generally do not feel the pain in a relaxed state; however, while pitching, the pain returns, causing hesitations and unease during the motion. Studies have shown that this pain, in most cases, is limited to overhead arm motions only, meaning

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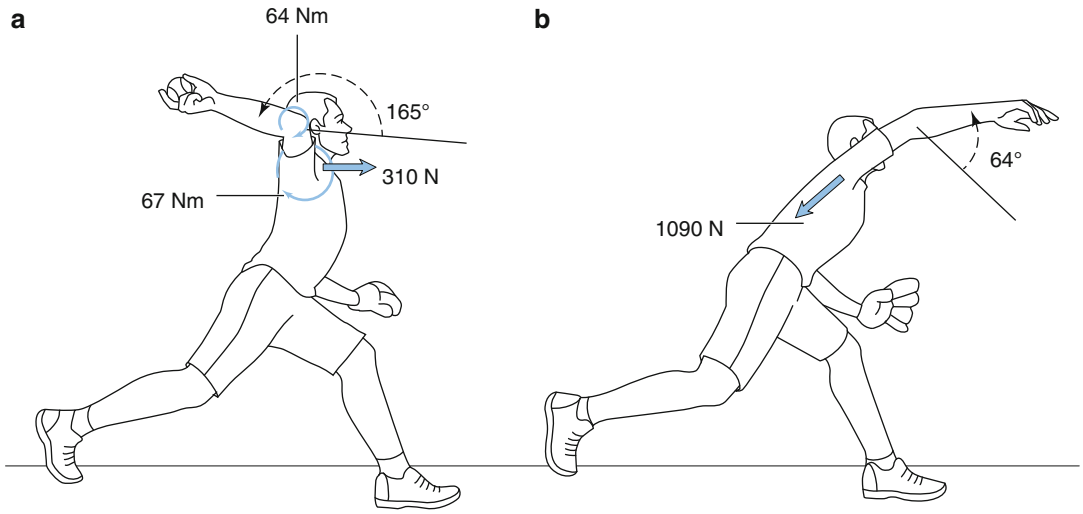

Fig. 33.1 The two critical instances of potential injury during the throwing motion. (**a**) The moment of full arm cocking when the shoulder reaches maximal external rotation. During this moment, 67 N m of internal rotation torque and 310 N of anterior force are applied to the shoulder. (**b**) The moment of ball release as the shoulder begins to decelerate. Forces at this moment include 1,090 N of compressive force at the shoulder joint to prevent subluxation

that other actions does not cause discomfort. Typically, such an injury occurs during exercise and practice, especially from repeated pitching motions (Fig. 33.1). The majority of professional players suffer from these chronic injuries, with symptoms appearing gradually over time.

As the initial symptoms are not visible to the eye, players often are unaware of these injuries and are able to pitch unhindered. But as the symptoms progress, the pitchers often express vague discomfort in the shoulder region while pitching and also has stiff shoulders that often feels like they are being "pinched." They also feel that it becomes harder to warm up and have weaker joints, and with continued movements in this condition, the pitching speed eventually decreases and the pain becomes worse. Chronic pain can be seen to affect pitching ability negatively, and resulting from this, affected pitchers who attempt to use techniques from before the injury exhibit abnormal pitching motions. As a result, it overexerts the structure within the joints and damages the body severely.

Previous experiences, number of balls thrown in recent plays, and number of recent innings, they all play a part in identifying the fatigability of the player.

As the symptoms develop, patients can locate the source of the pain, but as the symptoms appear with normal pitching pain, it becomes difficult to differentiate the two through basic medical examinations. The action in which the chronic injury appears most frequently is the overhead arm motion, specifically the late cocking and the ball release stages. These positions are the positions that create the most stress for the shoulder joints.

33.1.3 Range of Motion

The most easily observed symptom resulting from the overhead arm motion is the limited motion of the shoulder joint. Most of the pitchers benefit from increased arm external rotations; however, at the same time, they suffer from decreased arm internal rotations. These characteristics are not exclusive to baseball pitchers, but also extend to other sports, such as tennis. This is due to the following reasons: First, the bone that is involved with the pitching of the ball becomes deformed from attempting to pull back the arm for long periods of time, which in turn limits the shoulder movement. Second, the anterior portion of the shoulder joint becomes loose while the

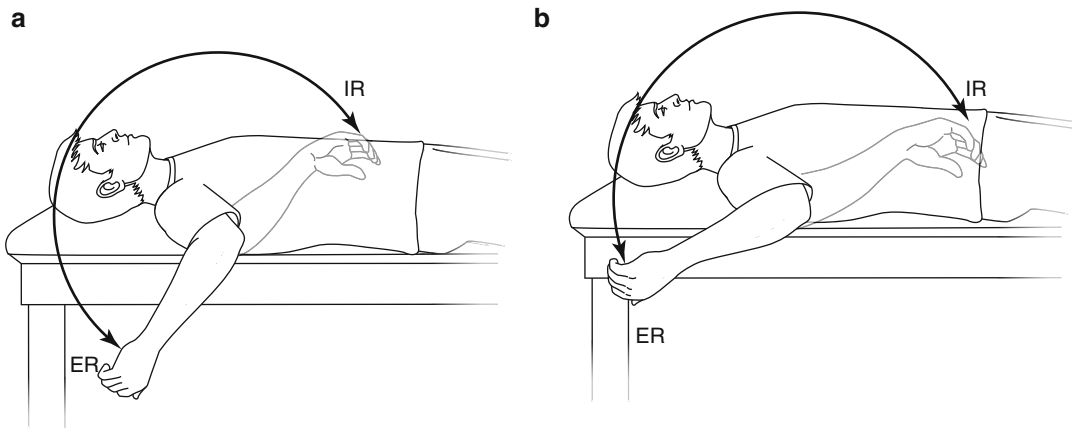

Fig. 33.2 The total motion concept. The dominant shoulder (**a**) of overhead-throwing athletes exhibits a greater external rotation (ER) and lesser internal rotation (IR) compared to the nondominant shoulder (**b**). However, the total motion (external and internal rotation) is equal bilaterally

back part contracts; by doing so this limits the shoulder joint movements (Fig. 33.2).

However, research has shown that when the injured shoulder joint is compared with the other, uninjured shoulder, there is no difference in the total degrees of movement. As such, it can be observed that the contracted joint capsule does not have any effect in the case of the decrease in the internal rotation. Third, a theory exists that due to the unusual contraction of the posterior shoulder joint muscle, minor muscle damage occurs, while at the same time, range of motion seems to decrease. Finally, a theory exists that the range of motion for young baseball players is different to normal population due to adaptation of the physeal plate. The theories mentioned suggest that many other factors may be involved and the damaged range of motion in the shoulder joints can be repaired, and athletes should make a constant attempt to adjust their motion to a normal range.

33.1.4 Scapular Position

Scapula problems should be checked by sports players who often utilize their arm in a throwing or similar motion repetitively, such as pitchers or even archers. This can be done by standing in front and backward at the mirror to check the placement of the scapula and inspecting for twisted or asymmetrical placements. If this method is difficult to distinguish problems, weights of 1 or 2 kg can be used in each hand with vertical movements to confirm proper or improper scapular movements.

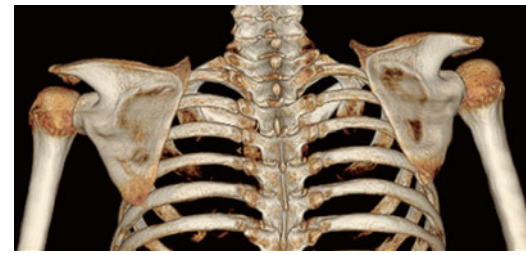

Fig. 33.3 Abnormal scapular position of right shoulder shown in 3D-CT scan

It has also been discovered that many players have suffered from asymmetric shoulder placement, causing lateral scapular displacement and the inferior border of the scapula to extrude away from the body while moving. This phenomenon occurs due to the lack of rehabilitation of the muscles which holds the scapula, and if movement is continued in this condition, it will cause pain in the anterior region of the shoulder and also in the cartilage inside the shoulder as it gets damaged. The photo below shows the 3D CT scan of a national archery player's scapula (Fig. 33.3). This player pulls the bowstring using his right arm, causing the right scapula to be twisted. If the player continues to play the sport in this way, there is a possibility that it will lead to permanent damage, leading to decreased athletic performances.

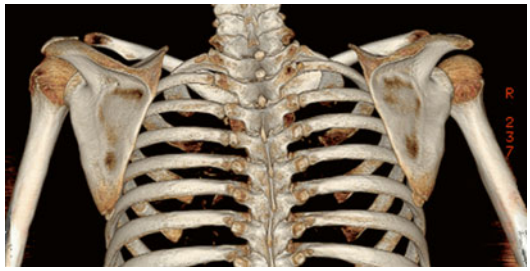

Fig. 33.4 Correction of scapular position of right shoulder after rehabilitation

At this point, many players take months off to rehabilitate in order to strengthen the muscles and readjust the rising scapula to look like the photo below (Fig. 33.4). However, if no action is taken against this problem, there have been recorded cases of problems developing that cannot be helped with rehabilitation.

33.1.5 Muscular Strength

Professional baseball pitchers who exercise every season for durations of up to 8 months have shown that there is a significant loss in muscle strength. This loss of strength persists even through shoulder joint conditioning programs designed to minimize muscular strength loss. The external rotation power of the arm reduces around 16 % during the mid-season and almost 21 % toward the end of the season. These issues do not make a huge difference to the shoulder muscles, but because of this, the fatigability of the muscle may increase. If these symptoms are continued, eventually the stability of the joint will decrease and there is a possibility that it could potentially lead to the development of subacromial impingement syndrome.

33.1.6 Proprioception

Overhead athletes have more relaxed joints and will be exposed to a wide variety of issues, leading to the shoulder joint to rely on proprioception. The proprioception ability will reduce when the player gets tired; however, it will return after a short break of 10 min.

33.2 Principles for Prevention of Injuries and Treatment Programs of Overhead Athletes

A general principle for the prevention of injury and curing of patients has been developed. Thus, it is important to have accurate basic knowledge on prevention and treatment program and also to practice clinically. The general injury prevention and treatment programs are as follows:

(i) Maintain the range of motion.
(ii) Maintain the shoulder muscle strength and endurance.
(iii) Strengthen the neuromuscular control functions.
(iv) Maintain the core and lower body muscles.
(v) Practice and exercise during off-seasons.
(vi) During the season, maintain muscle mass and muscle control.

33.2.1 Maintenance of ROM

The first principle includes maintaining proper pitching movement of the shoulder joint. Players who use overhead swinging motions generally have increased external rotation capacities, which allow for further movement to the back of the body, but suffer from limited movement in internal rotations, which limits the movement toward the front of the body. Even so, both arms still maintain the same range of motion. In other words, even if the injured arm has limited and increased movements in swinging back and forth, it will add up to an angle of 170, which when compared to the opposite arm which has a normal range of motion will be the same, as the other arm will also have a range of 170°. As this is just the body's way of adapting to the continued use of the shoulder joint, it should not be a point of concern. Thus, after the season is over, the range of motion would be different from the initial

range, so rehabilitation should be considered to equalize the range of motion, taking care to not excessively stretch the body to increase the range of motion.

If injuries are sustained or with incomplete rehabilitation, it is important to completely restore the range of motion. Rehabilitation times and methods vary among different types of injuries, but the most important thing to note is that attempting to return to using the same pitching motion without fully restoring the range of motion is very dangerous; therefore, trainers or doctors must prevent patients from throwing balls until they are fully recovered.

33.2.2 Maintaining Strength of the Shoulder Musculature

Repetitive throwing motion can cause damage to the shoulder joints, ligaments, and muscles, as it continuously applies pressure in those areas. Therefore, it is important to strengthen the muscle of the shoulder joint (which keeps the scapula in place within the body), elbow, and wrist. Perhaps you have seen athletes off the game using Thera-Bands or dumbbells to exercise while watching the game; this is one method to exercise during off days to improve the player's playing ability.

Each player can check for the muscles that they need to exercise, but all players should not exclude the shoulder joint muscles, shoulder blade muscles, and the lower trapezius. These muscles are not the muscles to improve ball throwing but serve as protection to the athlete's body while in throwing motion and also provide basic endurance.

Intrinsic muscles and extrinsic muscles originate from the scapula which makes the harmony during throwing motion. It is essential to keep up the harmony between the scapular and the humeral head to generate the high speed in throwing motion. And also the position of the scapula affects the glenoid labrum while pitching.

33.2.3 Emphasis on Neuromuscular Control

If you pitch with a lot of force, the joints and ligaments will get loose and will not move in a stable way and will be unstable during the pitching motion. When the joints move in an unstable fashion, damages can occur to the cartilage within the joint or tendons from the muscles. In order to prevent this, players must exert proper control over the muscle movements. Muscle control is an important factor in overhead motion injuries and treatment programs but is also one of the most important components in rehabilitation. With great control over the muscles, it is possible to prevent the forward movement of the shoulder during high-speed pitching and can prevent injuries of the shoulder muscles conflicting with nearby bones; therefore, muscle control is very important to overhead athletes who primarily utilize overhead arm motions.

Muscle control can be trained by rhythmic stabilization, reactive neuromuscular control drills, closed chain exercises, and plyometrics. If rehabilitation is done correctly, it is possible to develop balanced shoulder muscles, which prevents injuries during excessive throwing motion.

33.2.4 Core Muscle and Lower Body Training

The lower body makes up more than 50 % of the power while pitching. Core muscle exercises and lower body training not only allow the arm to move smoothly around the body, but also allow the shoulder to be in a stable state during pitching and are essential for the elbow and hand to pitch naturally. If the lower body muscle is weak and has a lack of endurance, or the muscle control is not great, the athletes cannot pitch in a normal fashion. As such the making of a good player is dependent on the strength of the lower body.

Core muscle training is an important factor in maintaining the exercise chain. It is also important for nonathletes on a weight-losing diet program, as it increases metabolism. But obviously, a good pitch will not happen if the lower body's

strength is not delivered into the arm. Suppose that core muscle is not strong enough. The strength from the legs will be stopped from the waist which means the strength should be reproduced beyond the upper body. Then, it is obvious that the speed or weight feeling during pitching will decrease.

Occasionally players who pitch while ignoring these facts will attempt to draw strength from the arm to maintain the speed of the ball, and unbalanced strength will be generated from the upper extremity. If this motion is continued, it could damage the arm and may result in deformation in pitching forms.

In conclusion, the leg and the body play an important role in the stability of pitching motions. Lack of body flexibility, the weakness of nondominant hip muscles, and decreased strength of hip abductors and spinal muscles can lead to destroyed exercise chain. This results in a spinal lordosis which leads to an abnormal posture (late forward movement of the shoulder joint compared to the lower extremities) at the acceleration of the throwing phase, and this is called "slow arm." "Slow arm" can arise from excessive external rotation and abduction of the shoulder joint, which could possibly damage the glenoid labrum.

33.2.5 Off-Season Preparation

Also, the off-seasons are very good for the resting of the body, rehabilitation, and cure in preparation for the next season. Although they have a break in the beginning of the playoff season, they need to strengthen the muscles of the entire body step by step and work hard to regulate and maintain it. Although there are some public appearances during the break, it is still important to keep up the endurance and strengthen muscles sufficiently so that the player will not feel tired or weak during the season.

At the end of the season, even if the players do not play, physical rehabilitation should be continued. It is useful to maintain recreational activities such as swimming, golf, cycling, and jogging. This period before the next season can be seen as valuable time to rehabilitate the injuries previously sustained.

33.2.6 In-Season Maintenance

As much as off-season, it is also important to maintain muscle control, muscle strength, and endurance during the season. Repetitive exercises for such a long season can lead to decrease in performances of the player.

It is essential to take on a whole body muscle strengthening and regulating program and to look carefully at the shoulder joints and muscles. Shoulder or elbow joint fatigue or muscle weakness can lead to injuries as it makes the joints unstable; therefore, the muscle maintenance program during the season is an essential component.

33.2.7 Balanced Periscapular Muscle Exercise

The rehabilitation of the periscapular muscle is the most neglected aspect in overhead athletes. Many shoulder pain occurs from abnormal scapular movements and lack of rehabilitation. Scapular dyskinesis can also be divided into three subtypes:

Type 1 is anterior tilting of the scapula, resulting in protrusion of the inferomedial border of the scapula, and makes scapular retraction difficult during the cocking phase. It is related to the lack of flexibility in pectoralis major, pectoralis minor, and serratus anterior muscles (Fig. 33.5).

From the 3D CT scans, as shown on the left when compared to a normal person (right), the scapula is separated from the thoracic cage (Fig. 33.6).

Type 2 is protrusion of the medial border of the scapula due to an increase in internal rotation of the shoulder joint. It happens mostly from weakness in the upper, middle, and lower trapezius and rhomboid muscles. From the pictures below, the second type of scapular dyskinesis is visible on the right side (Fig. 33.7).

With the use of 3D CT scans, it is visible on the right side where the scapula is rotating anteri-

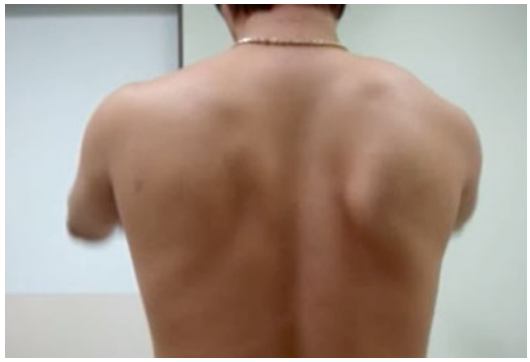

Fig. 33.5 Anterior tilt of right scapular (SD type I)

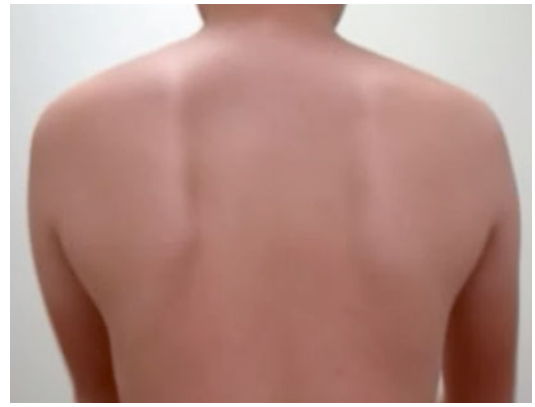

Fig. 33.7 Internal rotation of right scapular (SD type II)

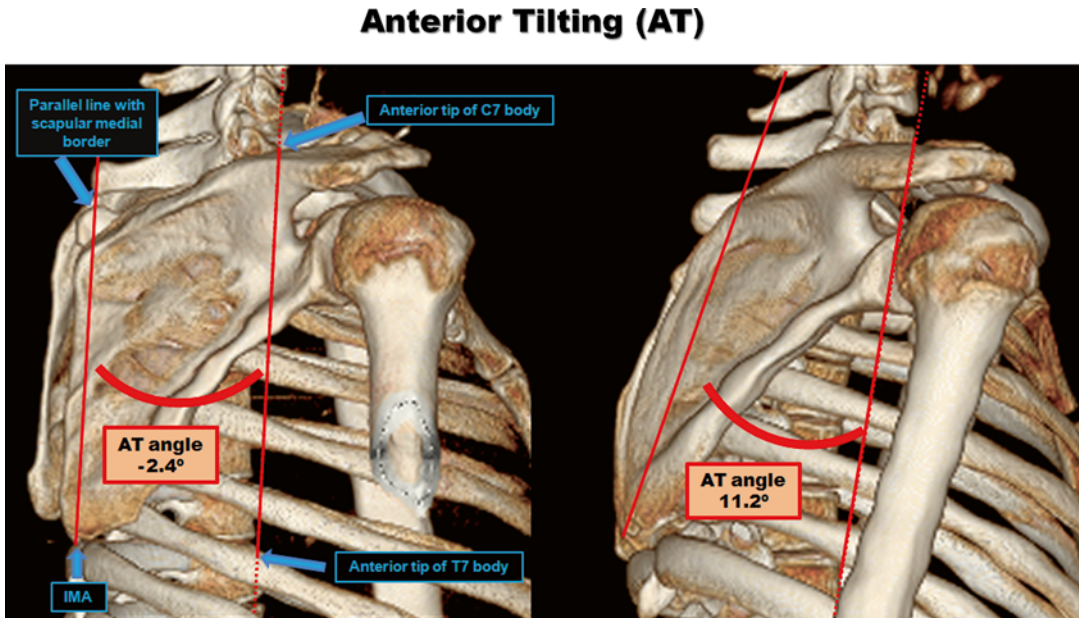

Fig. 33.6 Left picture: normal scapular position in 3D-CT scan. Right picture: anterior tilt of scapular in 3D-CT scan (SD type I)

orly to the thoracic cage compared to the normal left side of the scapula (Fig. 33.8).

Type 3 is popping out (protrusion) of the superomedial angle of the scapula due to superior translation of the scapula. This phenomenon can easily be observed from your own body as you move your arm vertically while holding some weights. It is also known that the serratus anterior has been weakened in many players with type 3 scapular dyskinesis (Fig. 33.9).

Also with the use of 3D CT scans, it can be seen that on the left side, the scapula appears to move to the upper side, which is very different to a normal person's scapula (Fig. 33.10).

For players who suffer from symptoms of scapular dyskinesis, it is possible for the scapula to return back to its original place after rehabilitation of 3–4 months. As the scapula returns to normal position, players will experience disappearing pain from the anterior part of the shoulder.

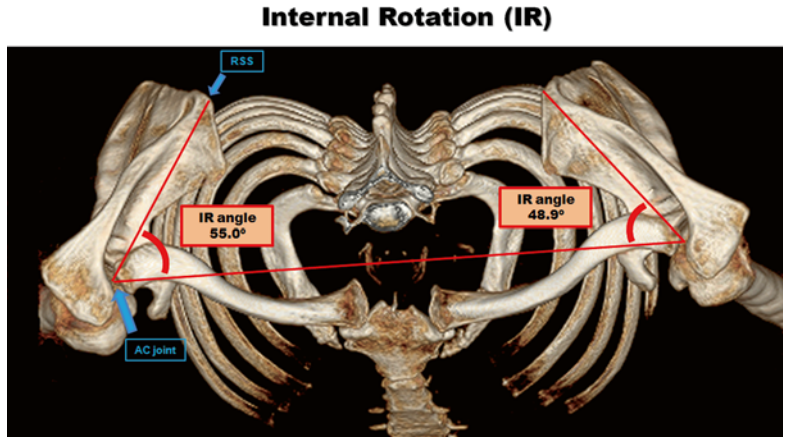

Fig. 33.8 Internal rotation of right scapular in 3D-CT scan (SD type II)

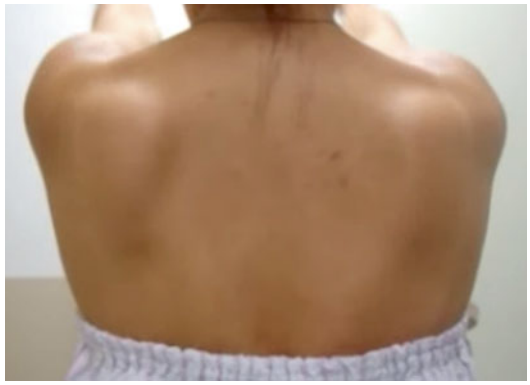

Fig. 33.9 Superior translation of left scapular (SD type III)

33.3 Rehabilitation Progression

Rehabilitation not only treats pain and inflammations for throwing athletes, but retains and mounts up both muscle strength and endurance. And it also helps with recovering proprioception, range of motion, and neuromuscular control. The progression of sports medicine rendered the development of rehabilitation methods, which allow faster recuperation of players. This enabled players to maintain their optimum state over a long duration.

33.3.1 Acute Phase

It is considered as an acute phase once afflicted with injury or on the day after the surgery. Initially, players do not participate on the competition. The acute phase duration depends on injury heaviness and healing capacity over impaired tissue.

ROM is decreased just after injury or surgery. Prompt stretching is required for recovering of ROM, within doctor's permissible amount. Gradual movement over incipiency rehabilitation program is required, and intensity should be increased steadily by enlarging ROM. One of the banned factors in this period includes rehabilitating by the player himself, and assistance from trainers or physiotherapists is essential. For initial stretching, a mild passive-active exercise (exercise with player's own strength added by trainer's strength) should be performed.

33.3.1.1 Exercises for Building Up ROM

Flexibility exercise for posterior shoulder muscles (flexibility exercise) should be performed initially. Posterior shoulder injury develops due to repetitive and extreme eccentric contraction during pitching motion and brings result in diminished internal rotation (Fig. 33.11). In this case, cross-body horizontal adduction stretch and sleeper stretch can be performed as stretching in diminished internal rotation (Fig. 33.12).

Excessively performed sleeper's stretch should be evaded as causing extreme stretch. Repetitive relaxed stretch draws better consequence rather than tensed stretch. So, softly

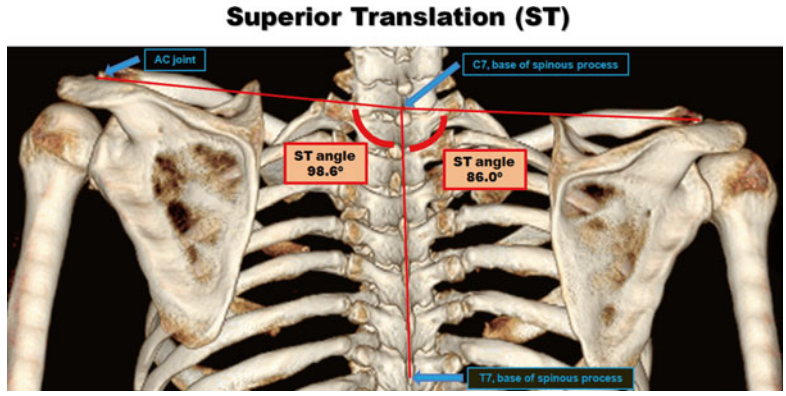

Fig. 33.10 Superior translation of left scapular in 3D-CT scan (SD type III)

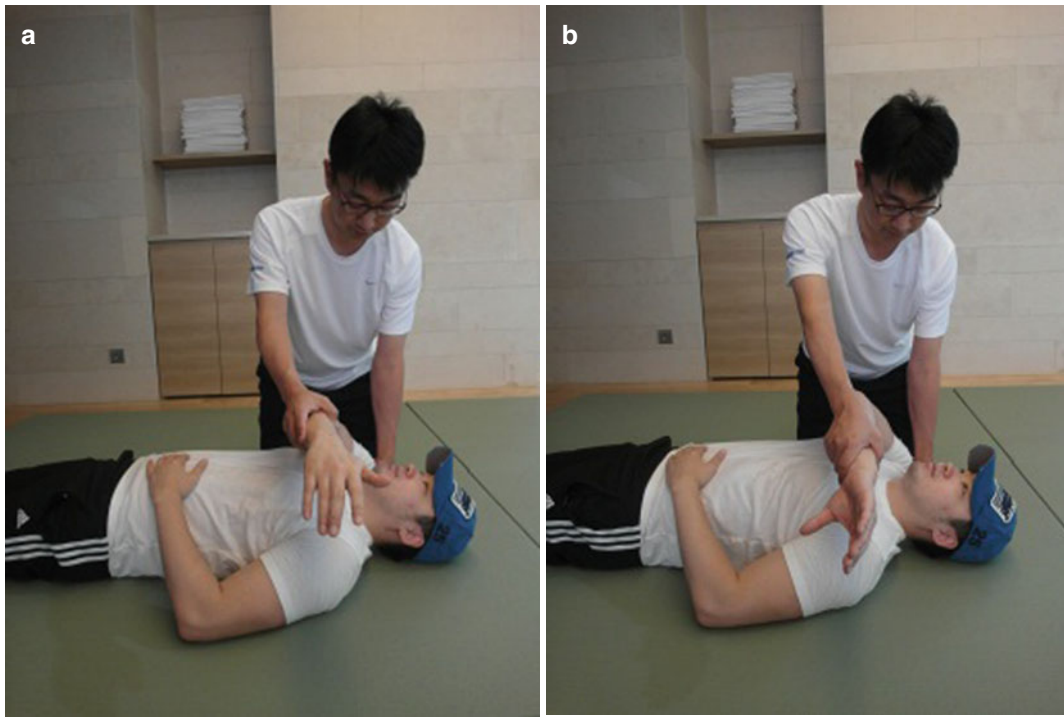

Fig. 33.11 Cross-body horizontal adduction stretch/the clinician may also perform the stretch with the shoulder in internal rotation

ingeminating multiple times is highly commended. If posterior capsule is stretched and posterior subluxation occurs, then this exercise should not be performed.

Alternative isometric exercise leads to simultaneous anterior and posterior rotator cuff contraction. Rhythmical stabilization exercise is performed while lying down with the arm positioned at 90° horizontal abduction and 90° external rotation (Fig. 33.13).

ROM for forward flexion of the shoulder should be enlarged similarly as above. Initially single-handed arm raising should be precluded. With the assistance of trainers or physiothera-

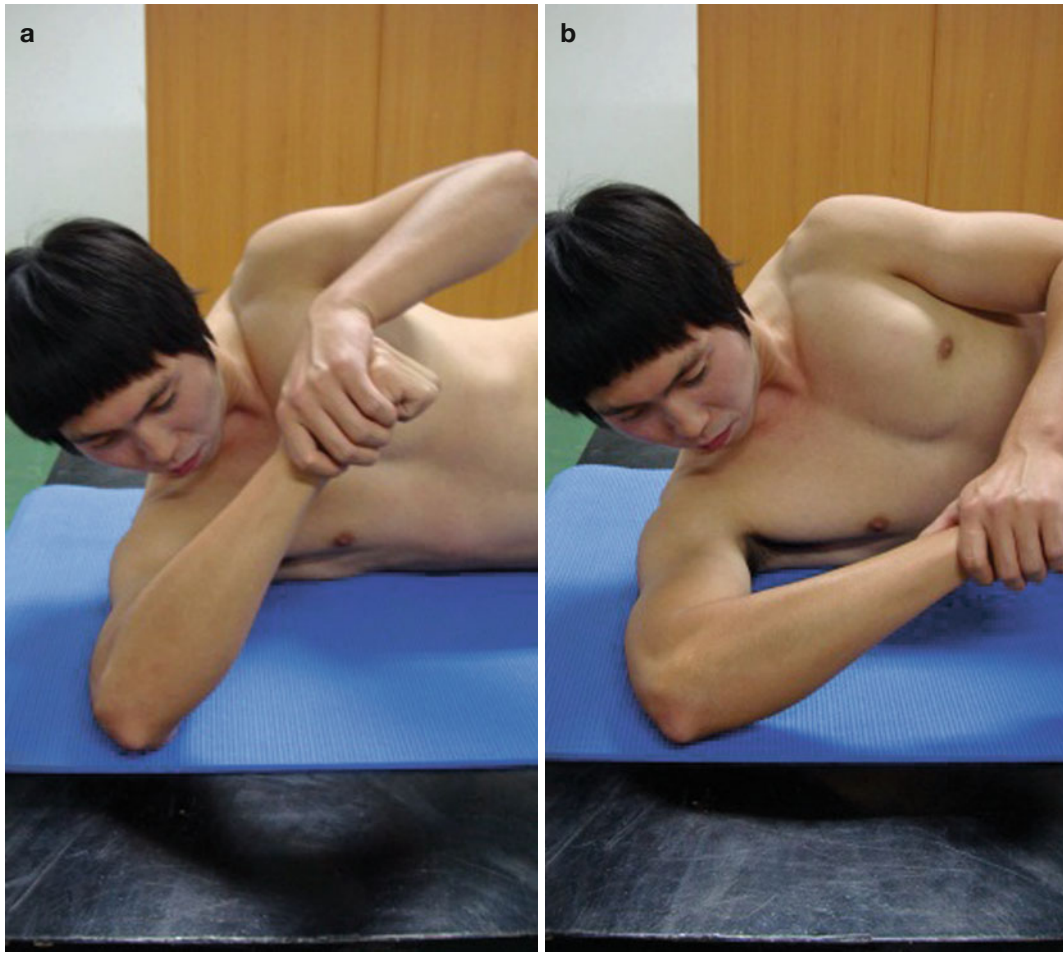

Fig. 33.12 Sleepers stretch to gain internal rotation

pists, forward flexion up to 100° should be performed (Figs. 33.14 and 33.15). Exceeding the range stands a chance of pain. Certain resistance should be applied while raising the arm in order to strengthen the shoulder muscles (Fig. 33.16).

When movement of joint scopes certain level and tissues appear to have healed, weight-bearing exercise is to be conducted. Weight is applied on both shoulders from the trainer in lying down position with arms and legs comfortable on the floor (Fig. 33.17). If they gradually waggle one's body back and forth, the shoulder muscle is contracted and this trains joints to stabilize. This exercise is conducted by the palm placed on the floor and pressing weights further on the shoulder.

Subsequent phase progression can be done by the hands on a gym ball to bear weight on arms and maintain balance.

Initial exercise without a trainer or physiotherapist ought to be conducted after proper completion of muscle strengthening and muscle balance.

With player's eyes closed, the therapists should passively flex, externally rotate, and internally rotate player's arms, and if movements are capable as they desired, yield to initial position. Then, active workout should be performed till demanded ROM is achieved and yield to initial position again. Physiotherapists have a duty of executing the appropriate ROM rehabilitation to perform initial basic position, working out posi-

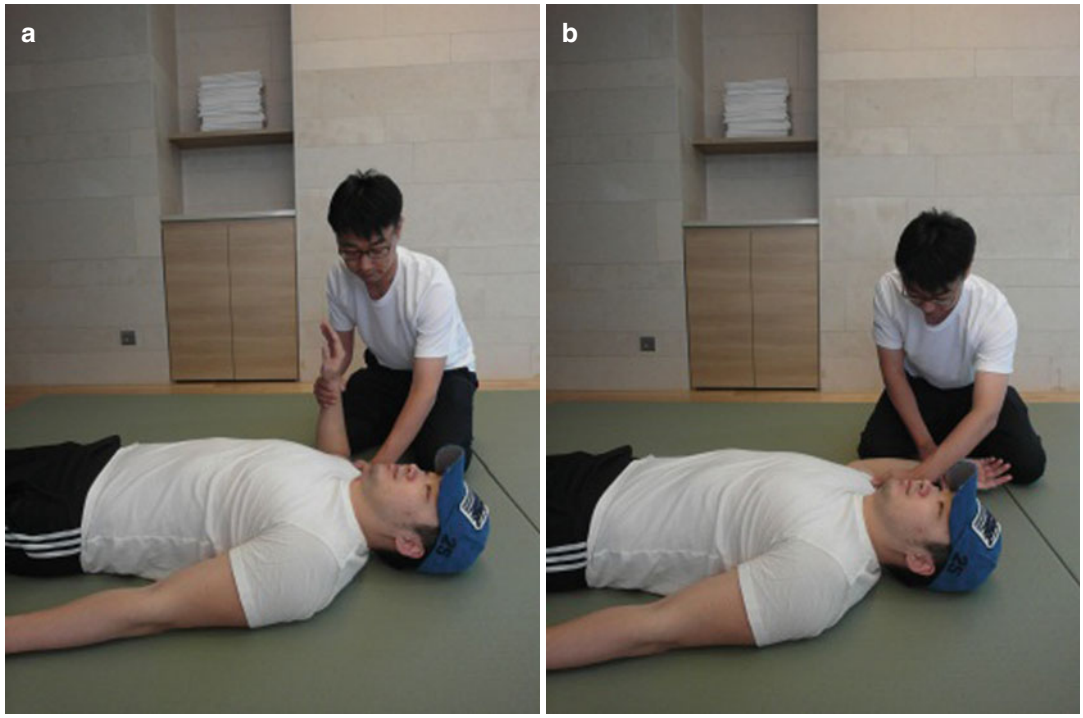

Fig. 33.13 Rhythmic stabilization drills for internal and external rotation with the arm at 90° of abduction and neutral rotation and 90° of external rotation

Fig. 33.14 Rhythmic stabilization drills for flexion and extension with the arm elevated to 100° of flexion in the scapular plane

Fig. 33.15 Rhythmic stabilization drills for the throwing shoulder while weight bearing in the quadruped position

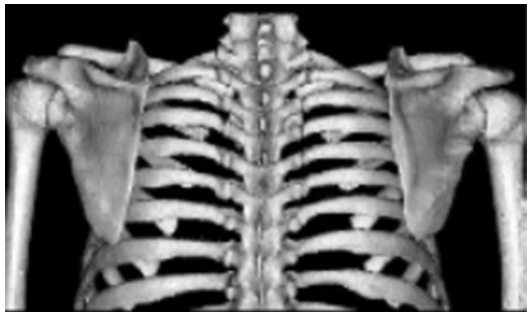

Fig. 33.16 Adequate scapular position obtained from 3D-CT scan

Fig. 33.17 Weight bearing exercise on the shoulder assisted by a trainer

tion, and recuperating to initial position again for players.

Ice, high-voltage stimulation, iontophoresis, ultrasonic wave, and taking NSAIDs may perhaps require for adjusting pain and inflammation. Iontophoresis is helpful, particularly to eliminate pains and inflammations.

Periscapular Strengthening Exercise

Scapular positions from the back or the exercising figures are ought to be checked via the trainer or physiotherapist while players either relax or exercise. If the player's shoulder turns out to be round shaped or has forward head or straight neck, then generally this indicates that scapular retractor or the extensor muscle of the neck is contracted which obstructs the flexibility of the shoulder. The worst case is protraction or anterior tilting of the scapula. If the scapula is inclined to anterior side, shoulder ROM will decrease.

In the scapular position which has been described above, it can be caused by shortening of pectoralis minor, upper trapezius, and levator scapulae and also can be caused with weakening of low trapezius, serratus anterior, and deep neck flexor muscle groups. It is noted that such sorts of problem drive arms to get easily fatigued with subsequent pain while pitching, and in severe cases, pressing the shoulder would result in arterial occlusion or neurovascular symptoms like cyanosis of the skin.

Shortening of the pectoralis minor, pain over coracoid process and scapular dyskinesis are common symptoms in pitchers, and complain with pain over coracoid process and scapular dyskinesis. Also players suffer from other several anterior shoulder pains. To release the pectoralis minor shortening, place towels between your scapula while lying down. Push both shoulders equally from the top, so that both shoulders reach the floor and gradually release the shortening of pectoralis minor.

Muscle strengthening exercise may possibly be conducted at a level of submaximal effort on

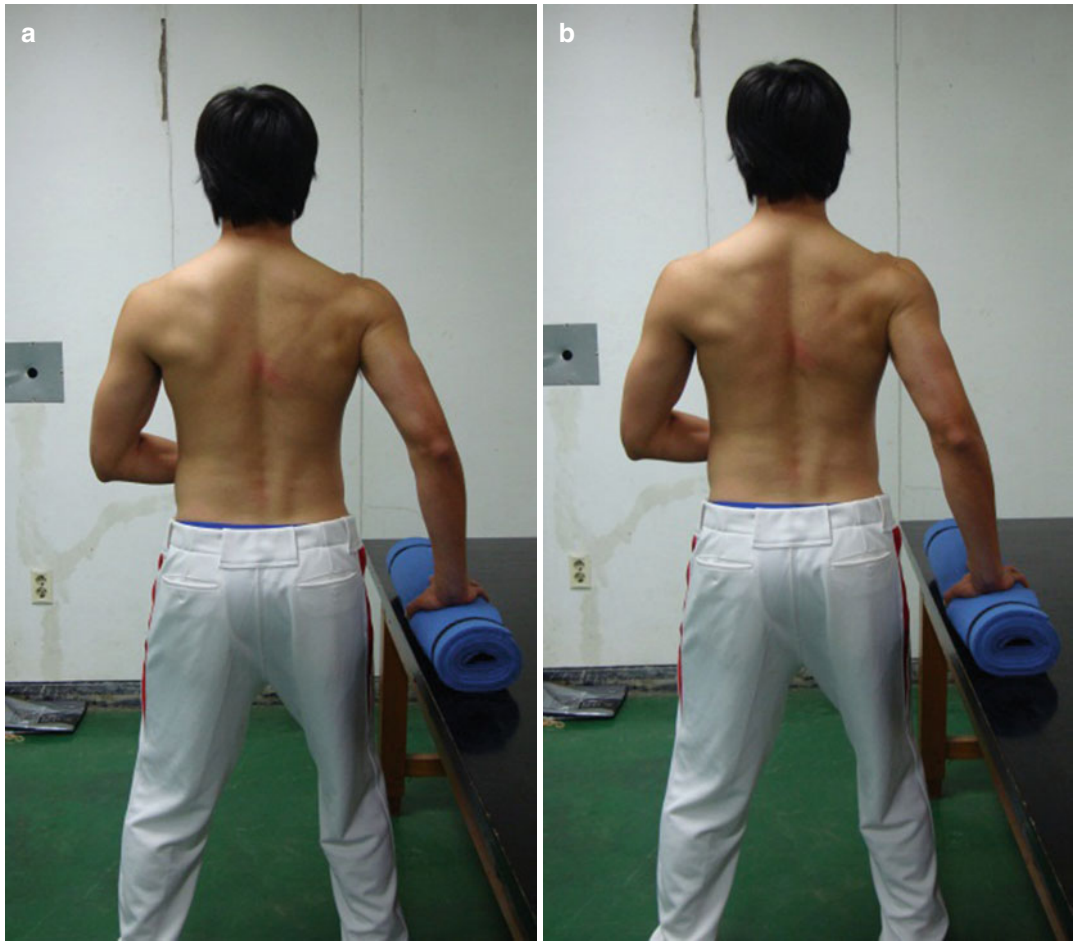

Fig. 33.18 Scapular retraction and protraction exercise on the table

the shoulder and scapula. Workout should begin with pain-free isometric exercise. Isometric exercise should be conducted from small ROM angle to large ROM angle.

Scapular Retraction and Protraction Exercise on the Table

Like the pictures above (Fig. 33.18), extend the elbow by fixing the hand on the table around the height of the lower waist. Then repeat axial loading to exercise scapular retraction and protraction.

Scapular Clock Exercise

Place the hand on the wall as shown in the picture and then move your scapula from 12 to 6 o'clock and 6 to 12 o'clock to obtain up and down motion of the scapula (exercise for upward and downward movement of the scapula).

From 9 to 3 o'clock and from 3 to 9 o'clock, repeat the front and the rear exercise continuously (exercise for scapular protraction and retraction) (Figs. 33.19 and 33.20).

If the shoulder joint pain is unnoticeable and exercises become familiar, then increase the

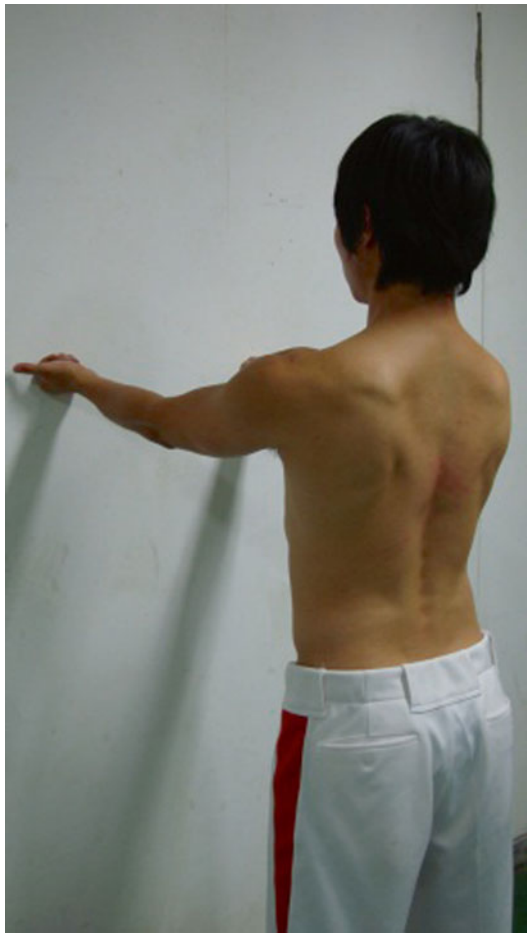

Fig. 33.19 Scapular clock exercise (external rotation)

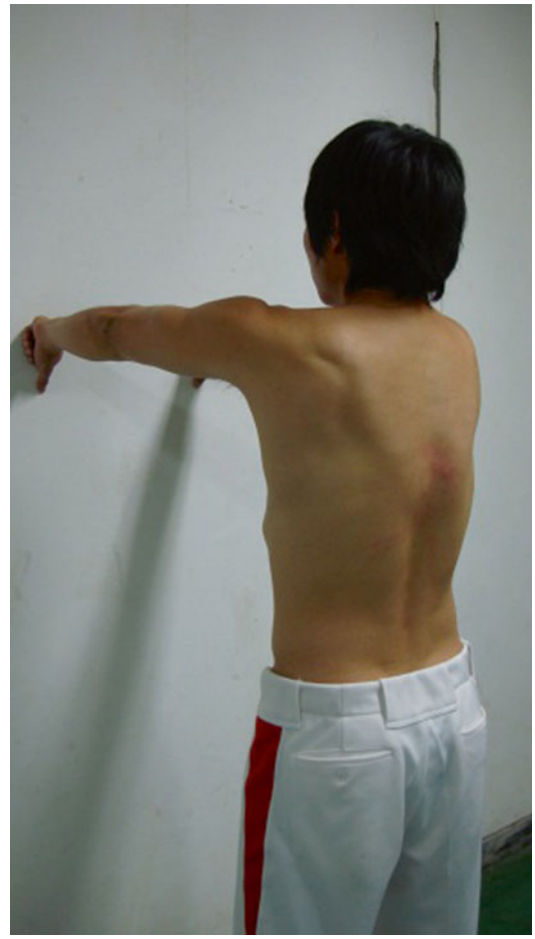

Fig. 33.20 Scapular clock exercise (internal rotation)

amount of protraction and retraction for enough movement of the scapula (Fig. 33.21).

When protraction/retraction exercise and upward/downward exercise of the scapula is well executed, then rotation exercise should be started. Retract the protracted scapula, and downward movement is made to strengthen muscles for retraction and downward movement of the scapula.

Basic closed chain exercise is easily executed in acute phase since the pain will not be presented during exercise (Fig. 33.22). When initially conducted, the hand position should not be higher than the shoulder height. In case of the hand or elbow going up further than the shoulder height, subacromial impinge or intra-articular glenohumeral impinge can be generated. As pain subsides, as ROM grows, or shoulder stabilization is established, active control exercise can be increased with placing a ball between the wall and the hand.

Place fairly same size as a bowling ball on the wall with arm abducted 90° and rotate wrist from side to side (Fig. 33.23).

When over the shoulder activity is able to be executed due to reduction of pain and joint stiffness, then wall wiping exercise can be conducted.

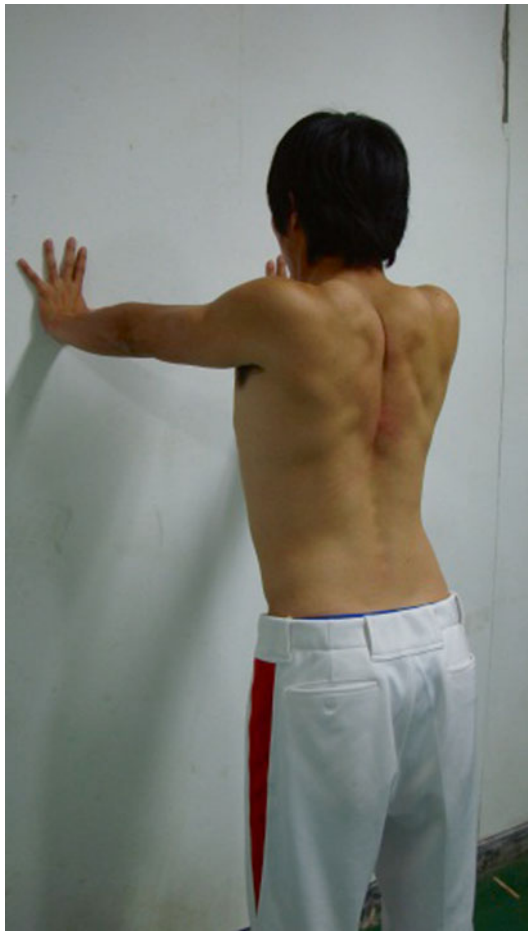

Fig. 33.21 Scapular clock exercise (neutral)

Wall Wiping Exercise (Fig. 33.24)
Contract the scapula and place the elbow bended in 90°, position the towel on the shoulder level at the wall, and move the towel in upper diagonal direction as shown in the picture.

If there is no pain for stroking the arm and if scapular and shoulder muscle arises, apply some weight to increase the endurance.

Push-up Exercise in Sitting Position
(Fig. 33.25)
In a sitting position, straighten the legs and do sit ups to raise and lower the body.

At this period, lower body and core muscle workout can be conducted without excessive forces loaded to the arm. First of all, to increase the flexibility, continue to flex, and extend the lower extremity and add rotational force of the body for core muscle flexibility.

Exercise for core and lower extremity should be performed in sequences listed below:

1. Core extension/scapular contraction
2. Body rotation/scapular contraction
3. Stand with one leg/diagonal direction body rotation

Hip Extension and Body Rotation (Fig. 33.26)
Extend and stretch your body and puff up and down the stairs repeatedly.

Rotate the body as seen in the photo, bend and stretch the hip continuously by one foot only or with two feet.

33.3.2 Maintenance Phase

At the point when the players achieve near to normal passive ROM with sufficient muscle strength and proper muscle balance, then maintenance phase begins. Lower body, core muscle's power, and stability are important factors to convey the power from the lower body to the arm.

Lower body strengthening and core muscle stabilization are trained at maintenance phase. In association to maintenance phase, proprioception and sense of movement should be recovered. And when athletes are using their arm, the muscles around the joint should be well developed to generate stabilization. Especially, at the end range of motion, the joint can be in an appropriate location and disrupt stability, so it is important to rehabilitate not to make joint slip out of normal position or make an abnormal scapular movement at the back.

Generally during the season, maintenance phase exercise starts within few days for injured players. Strengthening static stability which helps normal joint movement, maintaining and generating the individual's particular neuromuscular control ability, and improving the strength for returning to competition with basic stamina can be described as a goal of maintenance phase.

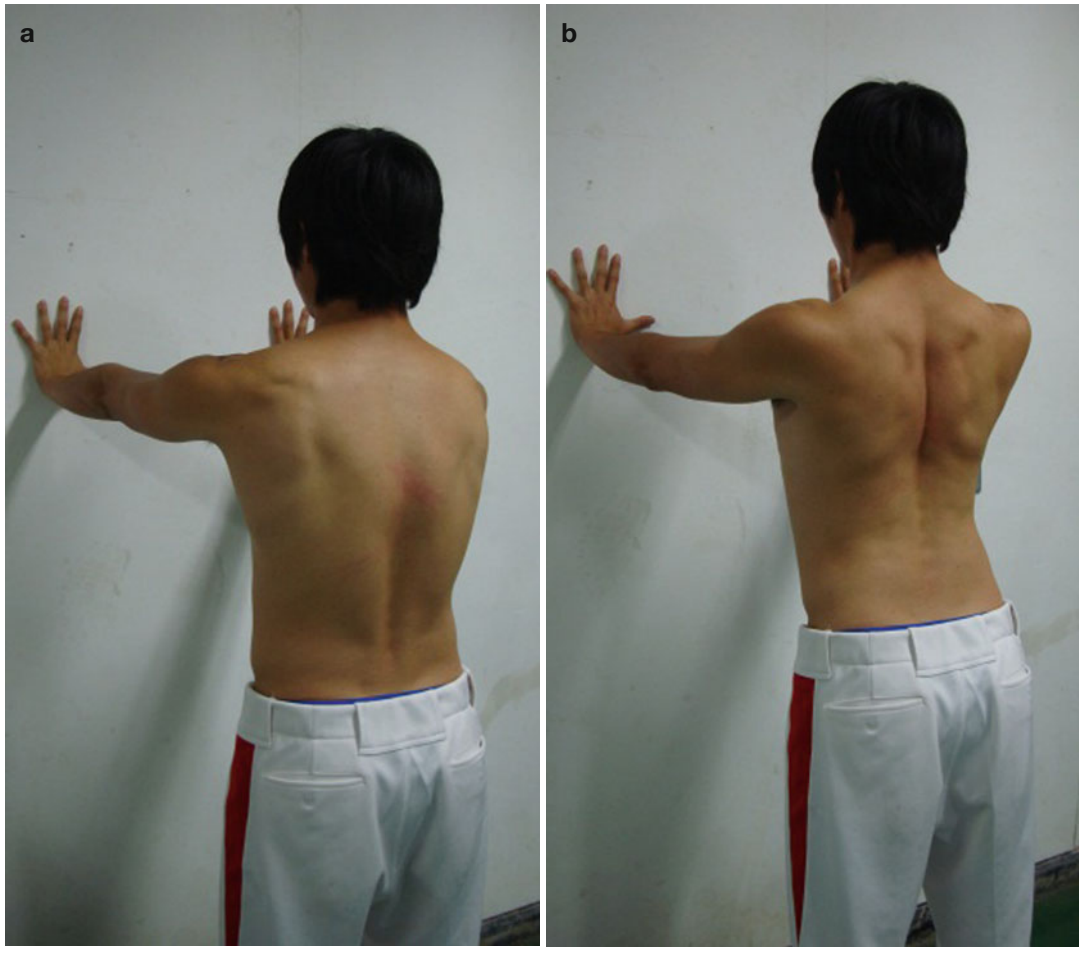

Fig. 33.22 Scapular retraction/protraction exercise against the wall

Fig. 33.23 Active control exercise for scapular

Fig. 33.24 Wall wiping exercise

Fig. 33.25 Push-up exercise in sitting position

Fig. 33.26 Hip extension and body rotation

At this period, rehabilitation program emphasizes the muscle's balance as well as recovery, and isotonic strengthening is performed aggressively. Furthermore selectively trained muscle improves muscle balance and symmetry.

Exercises to reinforce shoulder external rotation, scapular protraction, retraction, and infe-

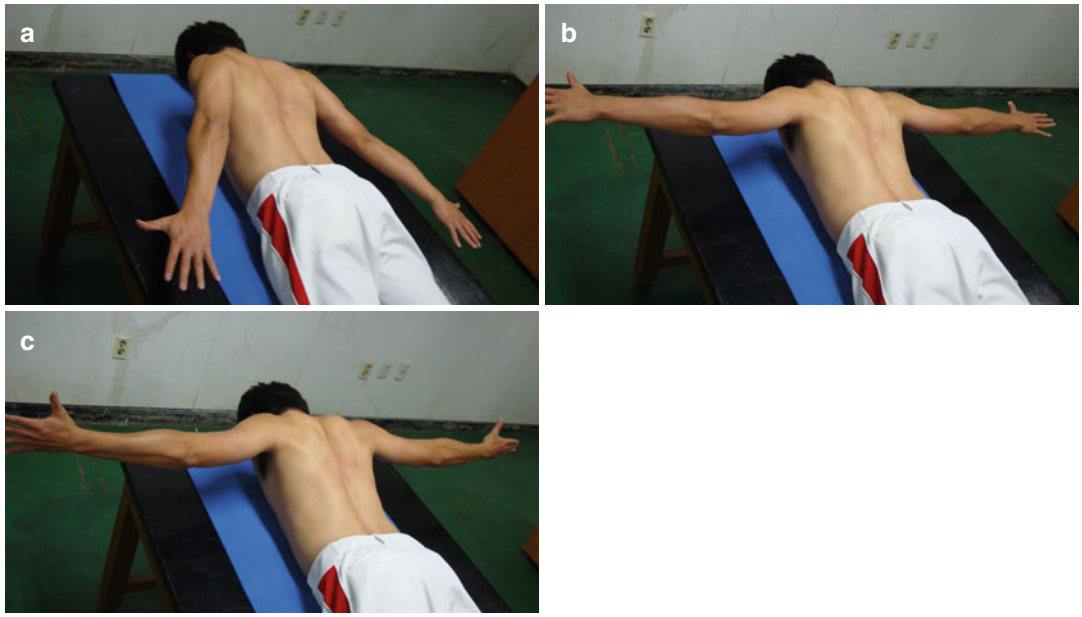

Fig. 33.27 Shoulder stabilization exercise I: start with gentle retraction scapular (**a**) and abduct arm 90 degree (**b**), then slowly externally rotate (**c**) to squeeze shoulder blades

rior translation are basic exercises for overhead-throwing athletes. The players tend to show weakened external rotation, but external rotation in a lying down in one side or rowing position is possible. Both exercise methods derive posterior rotator cuff muscle activities (Figs. 33.27 and 33.28).

Rehabilitations in the acute phase progress to make stability at the end range of motion by moving joints with eyes closed. At the intermediate phase, after passive motion with the help of therapists, resistance should be applied. And for certain exercises, the therapist may control the amount of resistance to make the player contract muscles for proper rehabilitation.

When it comes to a period of recovering muscle strength and neuromuscular control, externally rotate injured arm on the desk and progress to gently lifting up injured arm and rotating the arm externally around 45°. And if pain has subsided, then from 90° abduction position, internal and external rotation should be performed with tubes (elastic bands).

It is important to strengthen periscapular muscles and increase neuromuscular control ability to make stable movement for shoulder joints. Therefore, exercises such as rowing to improve muscle strength or isotonic exercise should be performed additionally.

Insert an additional exercise in the intermediate phase from the closed kinetic chain exercise, if supplementary balance maintenance exercise is needed.

From moving weights on an exercise ball, progress to push-ups on a ball or a table. Both shoulder and body stabilization is proceeded with rehabilitation therapist.

Place the back to the wall and check if both scapulas are touching the wall to find out if they are in the right position (Fig. 33.29).

Stabilization training using a wall is started with grasping a small ball (Fig. 33.30). For additional axial compression exercises, tables, slide pads, wrapping a towel on the hand, or any irregular surfaced objects can be used.

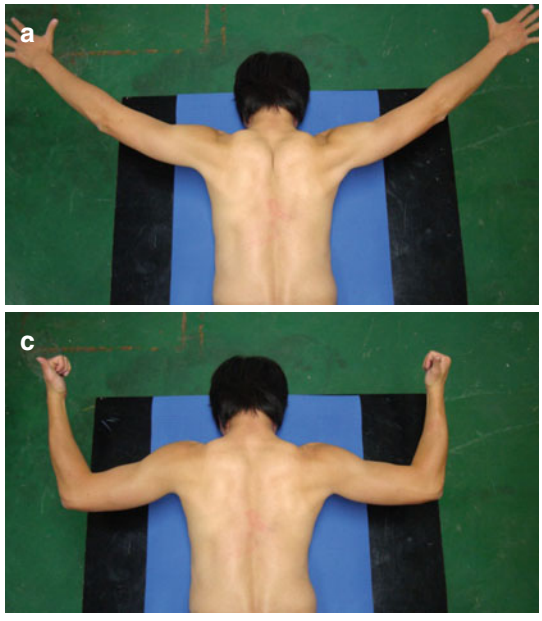

Fig. 33.28 Shoulder stabilization exercise II: start with gentle retraction of scapular with arm abducted more than 100 degree (**a**), externally rotate (**b**) to squeeze shoulder blades and flex both elbows (**c**) concentrating on the scapular to squeeze further more

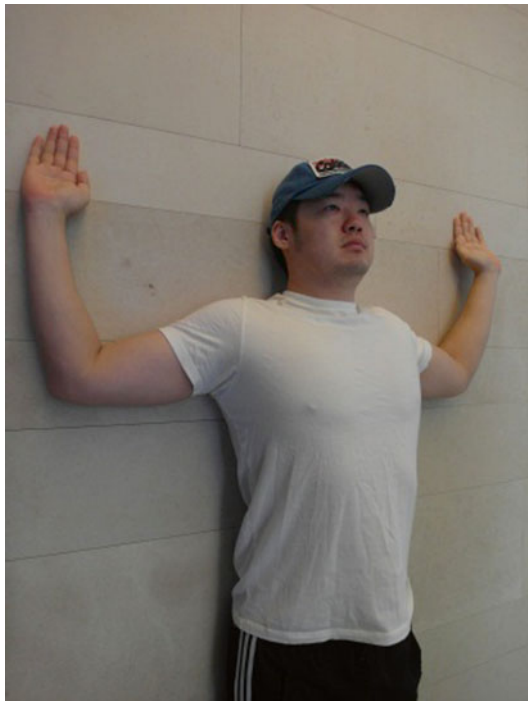

Fig. 33.29 Arm-extension wall slides to facilitate proper scapular retraction and posterior tilting

Fig. 33.30 Rhythmic stabilization drills in the 90° abducted and 90° external rotation position on an unstable surface in the closed kinetic position against the wall

33.3.2.1 Shoulder Dumps Exercise

Rotate the arm on the axis of throwing side; while retracting the scapula, raise and lower the dumbbells.

The stabilization of the scapula is vital for proper pitching motions; therefore, conduct strengthening exercises for muscles around the scapula (Fig. 33.31).

Fig. 33.31 Shoulder dumps exericise

Fig. 33.32 Low-row exercise using band I

33.3.2.2 Low-Row Exercise Using Band

With the elbows extended, repeat pulling Thera-Bands to your back. This exercise helps strengthen the serratus anterior (Fig. 33.32).

As shown in the figure below, stand with holding the band at 90 degree of arm abduction. Then flex the elbow and extend the shoulder to repeat retraction of the scapula (Fig. 33.33).

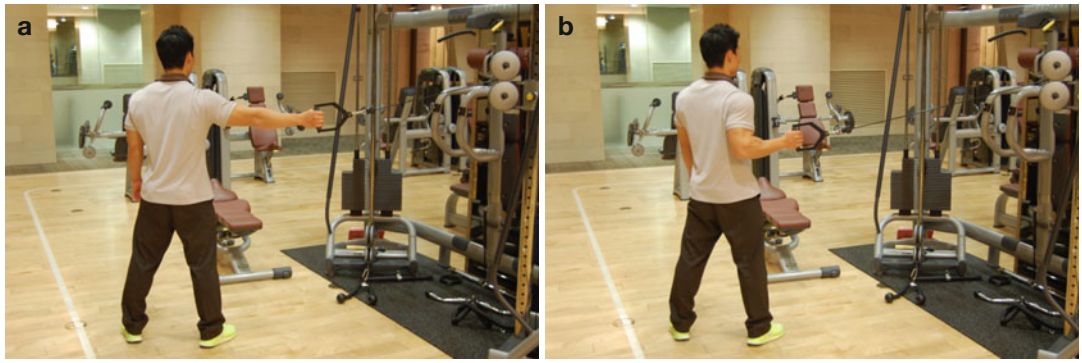

Fig. 33.33 Low-row exercise using band II

Fig. 33.34 Body and scapular exercise using band

33.3.2.3 Body and Scapular Exercise Using Band

As shown in the picture above, from forward-flexed position of the shoulder, extend ipsilaterally the body and hip. Then abduct and externally rotate the shoulder to repeat scapular retraction (Fig. 33.34).

33.3.3 Advanced Phase

The third-level rehabilitation program is getting prepared to return to actual sports activity. To attain this level, pain and tenderness should not exist, and ROM, symmetric capsular mobility, manual muscle strength testing (more than 4/5 of normal range), upper extremity and scapulathoracic joint endurance, and enough static stability should be based. In order to progress in this level, it is required to move the arm in a comfortable state and the posterior muscle ought to remain flexible.

At 90° abducted position, internal rotation and external rotation exercise using tubes proceeds to eccentric and high-speed contraction exercise. In the beginning, conduct with placing the arm on desk and later slightly execute the arm rotation in a small degree without any assistance. If the pain is not present and strength is enough, rotate the arm until 90°. In that way, exercise can be progressed without any discomfort.

Aggressive strengthening exercise of the upper body can be started on the individual patient's demand. General exercises contain limited isotonic weight machine bench press, seated row, and latissimus dorsi pull-down. In bench press and seated row exercises, players should refrain from stretching the arm behind to minimize the stress of the shoulder capsule. Latissimus pull-down is performed at the front of the player's head, and to minimize upper extremity traction, full extension is avoided.

To distribute the strength of the upper extremity, the plyometrics should be performed in this

Fig. 33.35 Rhythmic stabilization drills during exercise tubing at 90° of abduction and 90° of external rotation and during wall dribbles

period. Chest pass, overhead throwing, and alternating side to side throw by both hands is conducted with using a 3–5 lb medicine ball.

After ten to fourteen days of both-handed training, one-handed training drill is recommended. One-handed plyometrics include baseball style throw, deceleration flips, and stationary and semicircle wall dribble which uses a 2 lb ball with 90° abduction/90° external rotation position. Then, wall dribble is proceeded in 90/90 position. These exercises are helpful for endurance of the upper extremity on overhead-throwing sports (Fig. 33.35).

Training for dynamic stability and neuromuscular control should be responsive, functional and reproductive for unique position of athletes. In external rotation exercise when the player's arm rotation is in 0° by using a tube, concentric and eccentric passive resistance may be applied. Rhythmic stability is executed at the end range of motion in 90/90 position to challenge stability against resistance from the therapist or tubing (Fig. 33.35).

Rhythmic stability can be applied to end range of motion with 90/90 wall dribble exercise. These sorts of exercise methods are executed to obtain dynamic stability of the shoulder.

As fatigue rises during throwing motion, the risk of shoulder or elbow injury increases and muscle endurance exercise is emphasized. Endurance exercise is repeated with light weight such as wall dribbling, ball flip, wall arm circles, upper body cycle, or isotonic exercise (Fig. 33.36).

Murray investigated fatigues in the whole body while pitching a ball by analyzing exercise movements. When muscle gets tired, external rotation decreases, followed by reduction of the ball speed and increase of knee flexion along with decrease in shoulder adduction torque happens. With muscle fatigue, proprioception is affected. With fatigue rotator cuff, humeral head will be superiorly translated when the pitcher raises the arm.

In little league baseball players, the fatigue of the muscle is a major factor for shoulder injuries. Therefore, endurance training is the furthermost vital portion in overhead-throwing athletes.

33.3.4 Return-to-Activity Phase

With minimum pain or tenderness, complete recovery of ROM, balanced movement of joint capsule, proper proprioception, and dynamic stability and as all rehabilitation programs are finished, the player can get back to activity phase. Return to throwing starts with long-toss program, designed to lengthen the distance of pitching and to increase the pitching counts. Players start to pitch from the distance of 30–45 ft and increase up to 60, 90, and 120 ft. After long-toss program, the pitchers begin mound pitching program, and positional players get trained in their own position with longer-distance toss program. Pitching on mound includes increase of pitching counts, growth of effort intensity, and pitching different types of balls. Generally

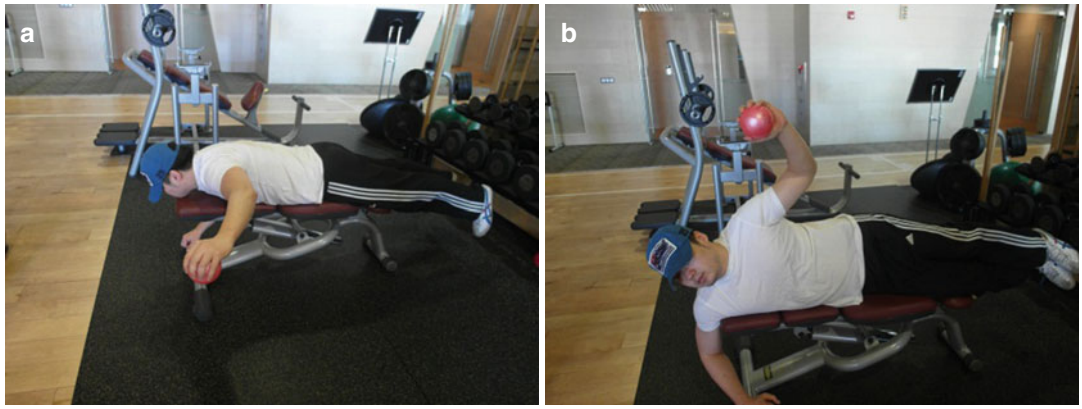

Fig. 33.36 Ball flips for endurance of the external rotators and scapular retractors

players pitch three times per week and have a day off in every other day. After that, it is repeated two to three times before moving on to the next level.

During interval throwing program, the rotator cuff and periscapular muscle should be exercised with low intensity. Every muscle strengthening exercise, plyometrics, and neuromuscular control training should be performed three times a week and conducted in same day as ISP (interval sports program). Players must warm up and stretch once before ISP and twice after the program. These methods contribute to adequate warm-up exercise, ROM maintenance for essential joint and secure flexibility of the upper extremity.

The day without throwing is used to exercise the lower extremity, cardiovascular system, body core stabilization, ROM, posterior cuff muscles, and periscapular muscles. This cycle is repeated for a week, and the seventh day is for rest. On rest day, athletes conduct light range of motion exercise and strengthening exercises.

33.4 Common Disorder in Throwing Athletes

33.4.1 Internal Posterosuperior Glenoid Impingement

Posterosuperior glenoid impinge (internal impinge) is the most common condition to overhead-throwing athletes, and it occurs due to excessive laxity of the anterior shoulder joint. Rehabilitation program's primary goals are to stabilize dynamic stability, control humeral head anterior translation, and restore the flexibility of posterior rotator cuff muscles. It is needed to approach cautiously to stretching of anterior and inferior shoulder structures since it causes anterior translation. Also it is important to confirm the position of the scapula. If anterior tilting of the scapula increases, the posterosuperior labrum and the posterior part of the supraspinatus would make contact and present with internal impingement. On this condition, special concern is required to the middle trapezius, lower trapezius, and serratus anterior, and eccentric muscle strengthening should be conducted.

Doctors or coaches must frequently observe the player's pitching mechanism after beginning of ITP. In players with internal impingement, the arm usually rises late since it follows behind the scapula (lag, excessive horizontal abduction). This excessive hyperangulation leads to excessive laxity of the anterior capsule and internal impingement of the posterior rotator cuff. Treatment of choice for internal impingement is nonoperative treatment.

33.4.2 Subacromial Impingement

Primary impingement syndrome is relatively rare in young overhead-throwing athletes. But with excessive hyperlaxity and decrease of glenohumeral dynamic stability, subacromial impingement syndrome can occur.

Nonoperative treatment is similar to internal impingement which emphasizes periscapular strengthening.

The patients presented with impingement syndrome appear to have a less posterior tilting compare to the patients without impingement. Rehabilitation program should contain pectoralis minor stretching, posterior inferior trapezius strengthening to establish scapular posterior tilting, and posture training to reduce anterior translation of the humeral head. Excessive protraction of the scapula will increase anterior tilt and reduce acromial-humeral space. And if rehabilitation is done to posteriorly retract the scapula, the space will increase.

Impingement syndrome can be treated with or without subacromial injection. Injection relieves pain and inflammation, which helps to conduct rehabilitation program after a break of certain periods.

33.4.3 Overuse Syndrome Tendinitis

Especially in the beginning of the season, players are not in an optimal condition. In this period players present with symptom of overuse tendinitis on rotator cuff or long head of biceps.

Players often appeal bicipital pain which is also called groove pain. During overhead pitching motion, the long head of the biceps is moderately active. The long head of biceps tendinitis is generally the secondary condition. Mostly, the first problem is instability, SLAP, or other issues. Rehabilitation for this problem focuses on dynamic stability through muscle training.

The long head of biceps is the muscle that reacts first when the capsule gets stimulated. The long head of biceps becomes more active with excessive laxity or inflammation in the glenohumeral capsule. Nonoperative rehabilitation treatment includes controlling the pitch counts, establishment of dynamic stability, and reduction of inflammation for the long head of biceps.

33.4.4 Posterior Rotator Cuff Tendinitis

To treat successfully on rotator cuff tendinitis, distinguishing internal impingement is an essential factor. In a subjective view, posterior rotator cuff tendinitis makes pain at the posterior shoulder at the ball release phase or the deceleration phase. The patient who's suffering internal impingement has pain during the late cocking or the early acceleration phase. During throwing motion, excessive force is loaded at the anterior side, and posterior cuff muscles contract to prevent the anterior subluxation. Players often experience weakness of the infraspinatus, lower trapezius, and middle trapezius and tightness of external rotators.

For rehabilitation, the eccentric muscle of the external rotator and lower trapezius should be strengthened. In the deceleration phase, the teres minor shows 84 % of maximal voluntary contraction, and the lower trapezius shows 78 % on electromyogram (EMG), so it should be focused when conducting muscle strengthening program.

33.4.5 Acquired Microinstability

In throwing motion, the anterior capsule attains vast tension stress in late cocking and early acceleration phase. This stress causes progressive stretching of capsule collagen and leads to anterior capsule laxity. Some authors argue that repetitive anterior capsule tension brings laxity of the anterior capsule and makes internal impingement worse. Even with avoiding excessive stretching, professional baseball players are known to have more than 5° of external rotation compared to the preseason.

As external rotation increases, the anterior band of the glenohumeral ligament complex will extend followed by increased anterior and inferior translation of the shoulder. Anterior translation can cause impingement between the inner part of the rotator cuff and the posterosuperior glenoid rim.

Many capsular plication and thermal capsular shrinkage have been developed to reduce joint laxity without giving too much tension. Rehabilitation for this type of surgery should steadily recover ROM, muscle strength, and neuromuscular control. Just after surgery, passive motion is permitted but aggressive stretching should be avoided. Excessive ER, elevation, or extension is also restricted. In 6 weeks, 75°

external rotation and in 8 weeks 90° ER and 90° abduction should be obtained. Normally between 6 and 8 weeks, the obtained flexion should be 170–180°. For overhead-throwing athletes, especially the pitchers, 115° of external rotation must be gained.

Gradual ROM exercise should be conducted, but full ROM should not be obtained before 12 weeks. Players should not stretch themselves to reach 115–120 ER. Their normal movement should be earned by functional activities based on rehabilitation programs such as plyometrics. For overhead-throwing athletes who are having rehabilitation after surgery, having difficulties in gaining full ER is a common phenomenon.

33.4.6 SLAP Lesion

SLAP lesion is a detachment of glenoid labrum-biceps complex from the glenoid rim. These injuries can be seen in a various injury mechanism such as falling accident, traction, car accident, and other sports. Overhead-throwing athletes are commonly observed with type II SLAP lesion. Also detached biceps tendon from the glenoid and posterosuperior labrum with peel back phenomenon can be found.

Type II and type IV SLAP lesion with labral instability or underlying instability is often unsuccessful to conservative treatment. After the surgery, at the beginning of rehabilitation, the important factor is to control the strength which is delivered to the restored labral tissue. When planning the rehabilitation program, it is required to consider the size or position of the lesion and the number of stitches. After surgery of type II SLAP lesion, return to competition takes about 9–12 months.

33.4.7 Triceps Tendonitis

Triceps tendonitis occurs due to the inflammation and swelling of the triceps tendon which is placed in the backside of the elbow. It usually happens with repeated stimulus from elbow extension motion. The treatments are as follows: maintain the range of motion, increase the flexibility, and recover the muscle strength.

Conclusion

Various lesions can develop to overhead-throwing athletes due to the strength generated while pitching. Treatment should be fully understood for each disease. Rehabilitation should be conducted steadily and orderly. ROM, flexibility, rotator cuff and periscapular strengthening, posture, and dynamic stability should be the main issue of preventing injury and the rehabilitation program. The program should be fitted to individuals, and the period of season, player's ability, and the injury type must be considered.

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