

# Prevention of Labral and Rotator Cuff Injuries in the Overhead Athlete

Nathan W. Skelley and Matthew V. Smith

## The Biomechanics of Throwing

There are five main phases of the throwing motion: windup, cocking, acceleration, deceleration, and follow-through (Fig. 2.1) [1–3]. The phases of the throwing motion generate and transmit energy to the arm to create velocity [1]. Windup and follow-through compose the majority of the throwing time, but the entire process can take less than 2 s. As a result, throwing athletes require efficient and well-coordinated motion of the upper and lower extremities.

## The Kinetic Chain

The kinetic chain is defined as the coordinated sequence of body movements that generate force to perform a particular action. In the throwing athlete, the kinetic chain starts when force is generated from the ground and is transmitted to the legs, the hips, torso, and the shoulder. Finally, the arm acts as the delivery mechanism of that energy

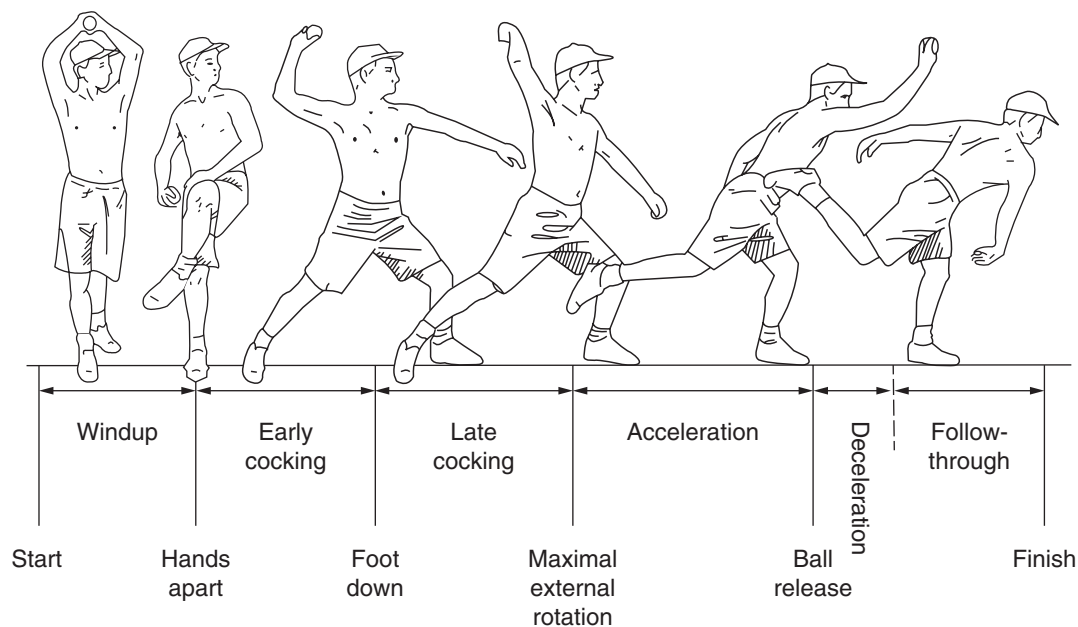
[1, 4]. Coordinated movements transmit energy in a manner greater than what the individual joints could develop on their own. Using more body segments within the kinetic chain can create a greater maximum velocity to the overhead throw [5]. The goal of the athlete's kinetic chain is to develop the optimal force while applying minimal joint loads during movement [6]. When deviations in ideal body mechanics occur, individual joint loads may change with distal segments overcompensating. As a result, the athlete is prone to overuse and to injury. For example, approximately 50 % of patients with superior labral anterior posterior (SLAP) tears have signs of core weakness and deficits in hip flexibility and hip abductor and extensor strength [7–9]. The clinician should evaluate the entire kinetic chain when evaluating the at risk painful shoulder.

Evaluating the entire kinetic chain in a complex movement, like throwing, is challenging [10]. Studying individual parts of the kinetic chain in isolation, however, can provide greater understanding when put in the context of the entire kinetic chain. Coordination of the entire kinetic chain is critical to proper positioning of the arm during throwing. Sufficient power of the lower torso is essential to generate ball velocity [11]. Core and lower extremity weakness creates an unstable platform for the thrower. In fact, weakness in the gluteal region, torso, and scapular region has been postulated to contribute to injury in throwing athletes.

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**Fig.2.1** This image demonstrates the five main phases of throwing: wind up, cocking, acceleration, deceleration, and follow-through. The stride phase is part of the lower body kinetic chain [Reprinted from DiGiovine NM, Jobe

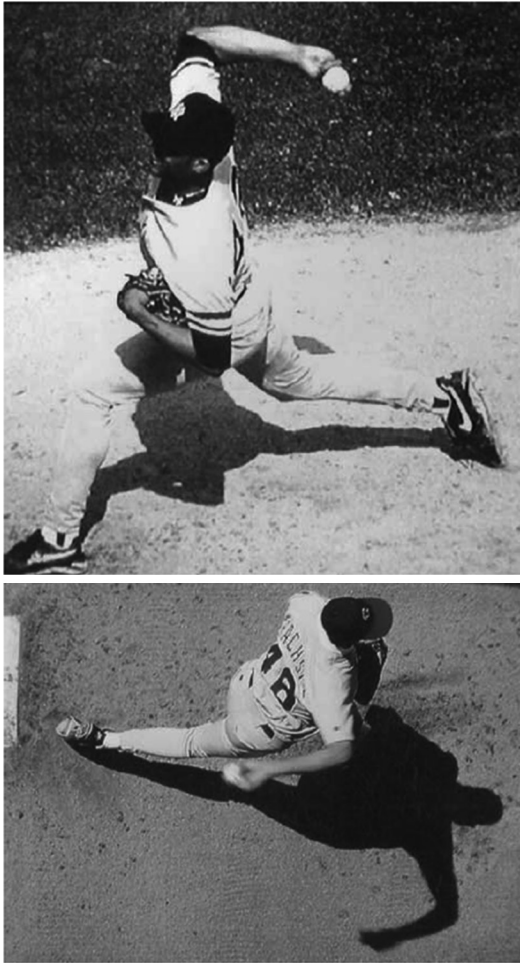
FW, Pink M, Perry J. An electromyographic analysis of the upper extremity in pitching. *Journal of Shoulder and Elbow Surgery*. 1992 1(1): 15–25, with permission from Elsevier]

Other areas to be inspected include lead leg internal hip rotation, lead leg quad tightness, and ankle range of motion. All “weak links” in the chain will lead to “downstream difficulties.” The clinician must evaluate the *entire* kinetic chain to determine weak points that place the overhead throwing athlete at risk. Prompt care to address these abnormalities through muscle training, stretching, and improved throwing mechanics is necessary to prevent kinetic chain abnormalities that could result in shoulder injury [12].

## Shoulder Mechanics

The elite overhead athlete can produce shoulder internal rotation velocity of 7000° per second. This is the fastest recorded motion by a human [1, 2]. This maximum velocity is achieved when the athlete externally rotates the arm to the maximal point of external rotation or the “set point.” Seasoned athletes can obtain >130° of hyper-external rotation during the late cocking phase of throwing (Fig. 2.2). To achieve this amount of

external rotation, adaptive changes in the glenohumeral mechanics are necessary. In abduction and external rotation, the inferior humeral articular surface rotates putting the anteroinferior shoulder capsule on tension. During the follow-through phase, the distraction force on the shoulder approaches 750 N or about 80 % of the pitcher’s body weight [1, 4, 13]. As a result, the posterior capsular tissue hypertrophies and tightens to adapt to these high distraction forces in order to help decelerate the arm. Over time, tightness in the posterior capsule shifts the center of rotation more posterosuperior on the glenoid so that the greater tuberosity does not impinge on the posterior glenoid (Fig. 2.3). The altered center of rotation relieves tension off the anteroinferior capsule resulting in a functional “pseudolaxity” of the anterior shoulder [12]. Pseudolaxity in the anterior shoulder can also be a result of a disruption in the labral ring surrounding the glenoid. If the labrum becomes detached posteriorly, the humerus can displace to this detached area due to the loss of labral restraint. This results in pseudolaxity on the opposite (anterior) side of the detachment.



**Fig. 2.2** This image demonstrates a baseball pitcher at the point of maximal external rotation or “set point” during a pitch. Notice the position of the legs and torso as elements of the kinetic chain [Reprinted from Burkhardt SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part I: pathoanatomy and biomechanics. *Arthroscopy: The Journal of Arthroscopic & Related Surgery*. 2003; 19(4): 404–420, with permission from Elsevier]

The cocking phase of the throwing motion is separated into an early stage and late stage. During the early stage, the deltoid muscle is activated and begins to place the arm and hand in the throwing position. During late cocking, the supraspinatus, infraspinatus, and teres minor are all activated to place the arm in abduction and external rotation. At this time the lower body begins shifting forward. This allows energy from

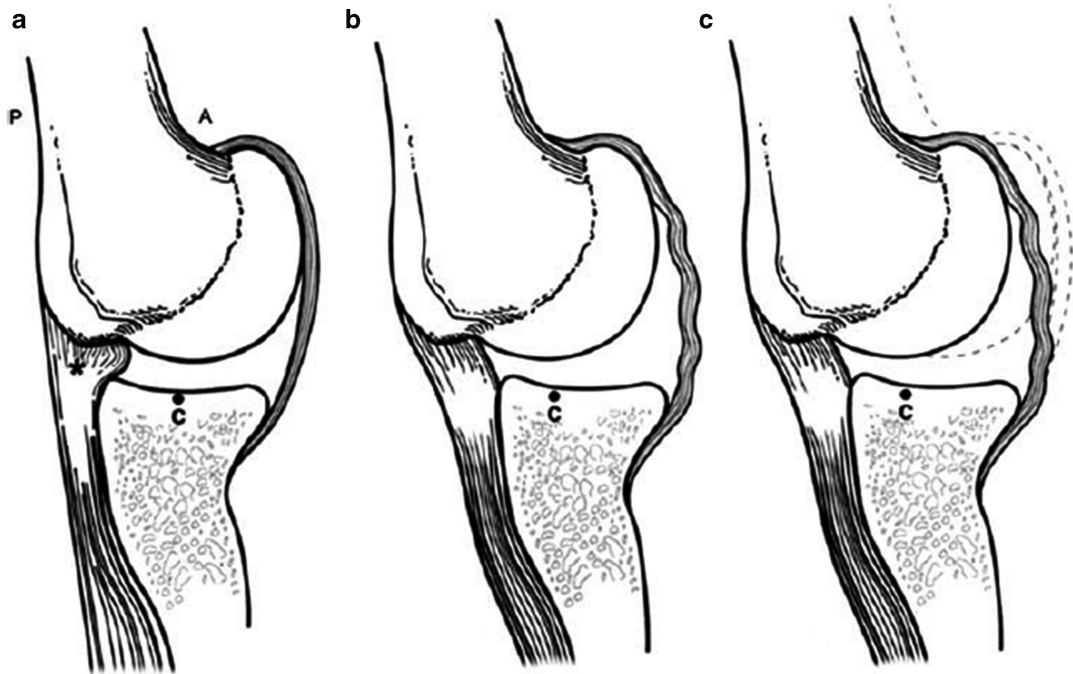
the ground to be transferred through the athlete’s kinetic chain resulting in a greater end force at the throwing hand [1, 2]. At the end of the late cocking phase, the shoulder reaches the “set point” [10, 12].

During the early acceleration phase, the triceps, pectoralis major, latissimus dorsi, and serratus anterior fire to maximize the energy in the kinetic chain. The deceleration phase has a high torque point as all muscles eccentrically contract to slow down the arm motion [1]. At this point, the center of gravity has shifted over the forwardly planted foot channeling the energy of the kinetic chain in the lower body. Finally, in the follow-through phase, the body rebalances forward motion, while the muscles return to a resting state.

### Pathomechanics of Labral and Rotator Cuff Injury

Throwers with shoulder injury commonly describe the feeling of a “dead arm.” A “dead arm” is any pathologic shoulder condition in which the thrower is unable to throw with preinjury velocity and control because of a combination of pain and subjective unease in the shoulder [7, 12]. The throwing arm is prone to injury because it requires greater abduction and external rotation to perform athletic activities compared to a non-throwing arm. Several authors have hypothesized that this greater range of motion is a result of “micro-trauma” or “micro-instability” to the anterior capsule [14]. Halbrecht studied the biomechanics of the shoulder and determined that anterior instability is not part of the pathology in the dead arm [15]. Other studies have similarly demonstrated that labral lesions are more commonly associated with dead arm syndrome instead of micro-trauma or micro-instability [12, 16].

Andrews is credited with first describing superior labral injuries. Snyder et al. further characterized SLAP injuries [17, 18]. Type II SLAP tears are defined as superior labral and biceps anchor detachment from the supraglenoid tubercle. Type II SLAP tears are common in throwing



**Fig. 2.3** (a) Normal orientation of the glenohumeral joint leads to rotator cuff impingement. (b) With tightening of the posterior inferior glenohumeral ligament, the humeral head moves posterior-superior and results in loss of the anterior capsule tension and a decreased cam impingement of the greater tuberosity on the posterior glenoid. This results in an anterior pseudolaxity. (c)

Demonstrates the superimposed humeral head positions [Reprinted from Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part I: pathoanatomy and biomechanics. *Arthroscopy: The Journal of Arthroscopic & Related Surgery*. 2003; 19(4): 404–420, with permission from Elsevier]

athletes, particularly tears involving the postero-superior region (Fig. 2.4) [19]. Type II SLAP repairs comprise approximately 10 % of all shoulder procedures and are the second most common shoulder arthroscopic surgery [20, 21]. The pathomechanics of labral injury are a complex interplay between activity demands and anatomy [12]. O'Brien described the inferior glenohumeral ligament (IGHL) as a two cable system [22]. Usually, the anterior and posterior cables support the humeral head like a sling while in abduction; however, if the posterior cable becomes contracted, from hypertrophy related to repetitive throwing, it can shorten and push the humeral head superiorly [23]. The hypertrophied tight posteroinferior capsule is the initial insult that shifts the humeral head allowing for hyper-external rotation, perceived pseudolaxity,



**Fig. 2.4** Arthroscopic image demonstrating labral detachment from the glenoid. This disruption can allow for humeral displacement resulting in pseudolaxity on the opposite region of the glenoid

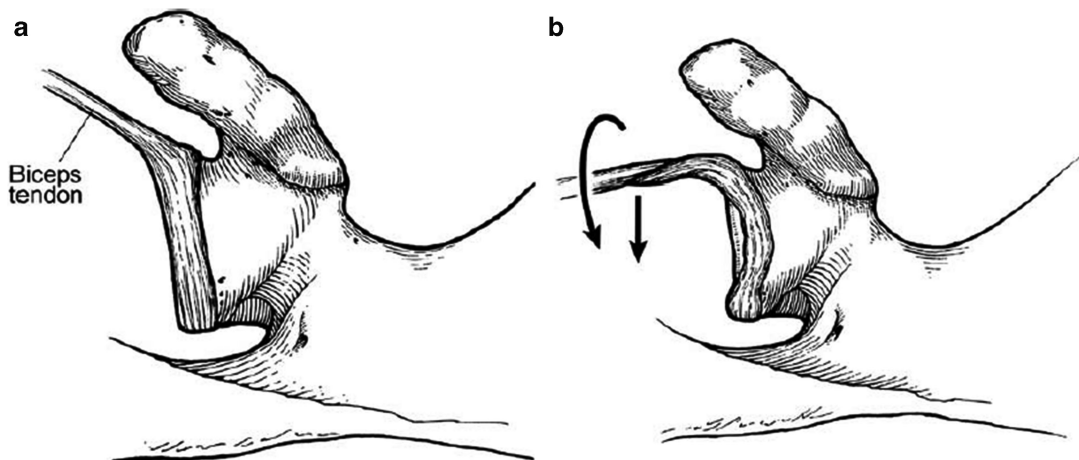
and greater ease obtaining the set point. This improved throwing mechanism is not without consequences. During the throwing motion, hyper-external rotation leads to abnormal impingement and abrasion damage to the rotator cuff [16]. The hyper-external rotation changes the vector force of the biceps tendon to a more vertical and posterior direction during abduction and external rotation [7, 12]. This vector and subsequent biceps muscle contraction create a torsional force across the posterior-superior labrum (Fig. 2.5). Abnormal twisting through the biceps origin on the glenoid leads to torsional overload and shear force injury to the labrum and rotator cuff fibers [16]. The labrum is eventually detached from its anchor as a result of this posterior-superior shift and hyper-external rotation: a “peel-back” phenomenon results.

Although Andrews et al. proposed that superior labral injuries are the result of longitudinal pull on the biceps anchor during the deceleration of the arm, others have proposed that hyper-twisting may be the mechanism causing labral injury [8, 12]. Kuhn performed a biomechanical study that supported the hyper-twisting in the acceleration phase as the mechanism recreating a labral injury [24]. This implies the biceps and superior labrum complex is “peeled off” instead

of “pulled” from bone in the deceleration phase [12].

Approximately, one third of all patients with SLAP tears also have rotator cuff tears [20]. Given this association, Walch and Jobe described internal impingement as abduction and external rotation inducing a pinched posterosuperior rotator cuff between the glenoid labrum and greater tuberosity of the humerus [14, 25]. Impingement in this area may also explain the partial articular-sided rotator cuff tears commonly seen in throwing athletes. Morgan et al. reviewed arthroscopic exams and found that rotator cuff tears were also found in 31 % of throwers being treated for SLAP lesions [19]. Of these tears, 38 % were full thickness tears located in the midportion of the rotator crescent, and 62 % were partial-thickness cuff tears in labral lesion-specific anatomic locations. The superior subluxation of the humerus combined with repetitive torsional loading from hyper-external rotation has been postulated as the cause for location-specific partial-thickness cuff tears [12]. The combination of labral pathology and rotator cuff tears is a complicated multifactorial process that ultimately results in loss of shoulder function and/or athletic performance.

Burkhart et al. consolidated these factors in the development of the dead arm which have



**Fig. 2.5** The position of maximal external rotation results in a vector change for the biceps tendon. During overhead movement, the altered vector creates a peel-back mechanism as the biceps pulls on the labral complex [Reprinted from Burkhart SS, Morgan CD, Kibler

WB. The disabled throwing shoulder: spectrum of pathology part I: pathoanatomy and biomechanics. *Arthroscopy: The Journal of Arthroscopic & Related Surgery*. 2003; 19(4): 404–420, with permission from Elsevier]



been supported in the literature: (1) the tight posteroinferior capsule leading to glenohumeral internal rotation deficit and a shift in the glenohumeral rotation point; (2) peel-back forces causing the SLAP injury; (3) hyper-external rotation of the humerus related to a reduction in the humeral cam effect on the anterior capsule and clearance of the greater tuberosity over the glenoid rim through a larger arc of external rotation; and (4) scapular protraction. The ultimate culprit in this series of injuries is the tight posteroinferior capsule [7, 12, 16]. Shoulder strengthening may be the best preventative measure for compensating for damaging forces in overhead activities and therefore prevent the development of shoulder pathology [16].

### Glenohumeral Internal Rotation Deficient

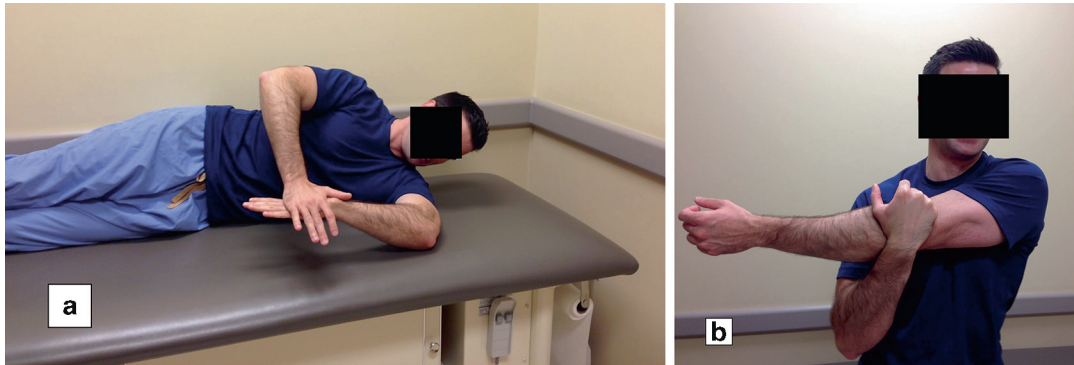
Scapular positioning and glenohumeral rotation are key components of shoulder function. Glenohumeral internal rotation is especially

important to the overhead athlete as a source of force generation. The total rotational range of motion (TROM) of the shoulder is a combination of glenohumeral internal rotation and glenohumeral external rotation with the arm abducted [6]. Maintaining the shoulder total arc of motion is important to protect the shoulder during throwing. Asymmetry of as little as  $5^\circ$  of TROM between shoulders is associated with increased shoulder injury risk [26]. Although studies have demonstrated a high risk of injury with a glenohumeral internal rotational deficit of  $11^\circ$  compared to the contralateral arm [26], most clinicians consider a glenohumeral internal rotation of  $18^\circ$  or greater to be diagnostic of significant glenohumeral internal rotation deficit (GIRD) (Fig. 2.6) [16, 27].

Conditioned throwers develop limitations in internal rotation from posteroinferior capsular contracture. This contracture can lead to GIRD. Prophylactic posteroinferior capsular stretching can minimize GIRD and therefore secondary pain and intra-articular symptoms [6, 12]. The sleeper stretch and cross-body stretch used



**Fig. 2.6** This patient has internal rotation deficit between shoulders. (a) Dominant arm external rotation and internal rotation at  $90^\circ$  of abduction. (b) Nondominant arm external rotation and internal rotation at  $90^\circ$  of abduction



**Fig. 2.7** The sleeper stretch is one of the most effective methods of stretching the posterior inferior capsule. **(a)** The athlete applies a passive internal rotation force to the

abducted shoulder while lying on the side. **(b)** The cross-body stretch is also utilized to stretch the posterior capsule and shoulder musculature

over a 2-week period can frequently improve ROM to 20° or less (Fig. 2.7) [12]. The sleeper stretch is performed in a lateral decubitus position with the back against a wall to stabilize scapular motion. The shoulder is flexed to 90° and passive internal rotation is exerted by the opposite arm to stretch the posterior shoulder. The cross-body stretch is performed in a standing position by placing the non-stretched arm on the distal humerus just proximal to the elbow and passively pulling the arm across the chest. In rare cases of persistent posterior capsular tightness after a prolonged stretching program, arthroscopic release of the posteroinferior capsule followed by a stretching program can improve motion.

## Scapular Dyskinesia

Scapular dyskinesia is any alteration in the normal position or motion of the scapula. The dyskinesia can be related to inflexibility, weakness, muscle activation imbalance, or a combination of these variables. The SICK scapula syndrome is a general term used to describe these presenting findings in an athlete with shoulder pain. SICK scapula syndrome is defined as scapular malposition, inferior medial border prominence, coracoid pain and malposition, and dyskinesia of scapular movement.

The thrower with SICK scapular syndrome frequently has an insidious onset of anterior shoulder pain and eventually notes a “dropped”

scapula. The dropped orientation comes from anterior tilting and protraction of the scapula which also enables the pectoralis minor to contact. The coracoid is often tender to palpation and correlates with the presenting anterior shoulder pain [12]. The key finding is asymmetric scapular malposition, usually lower positioning in the dominant throwing shoulder. The associated pain should not be confused with anterior shoulder instability or a SLAP tear. Overhead athletes with existing or impending labral or rotator cuff injuries commonly note resting pain, particularly anteriorly over the coracoid. They also report pain during the late cocking and/or early acceleration phases of throwing.

Burkhart et al. have described three types of angular deformities that can be statically measured to quantify the level of scapular dyskinesia [6]. The type I is inferomedial scapular border prominence associated with pectoralis major and minor inflexibility and trapezius and serratus anterior weakness. The type II pattern demonstrates medial winging related to trapezius and rhomboid weakness (Fig. 2.8). Both conditions protract the scapular and decrease cocking ability of the shoulder. Posterosuperior labral lesions are associated with these two types of scapular dyskinesia. Type III scapular dyskinesia is associated with impingement symptoms and with rotator cuff pathology rather than labral lesions. In type III scapular dyskinesia, the superomedial border of the scapula becomes more prominent.



**Fig. 2.8** This patient is lowering her arms from a forwardly elevated position. She has a left side type I SICK scapula with inferomedial scapular prominence from tight pectoral muscles and weak triceps and serratus anterior muscles

To appreciate the etiology of the SICK scapula, one must appreciate that the scapula is a flat bone gliding and pivoting about the ellipsoid surface of the thoracic cavity. When the scapula protracts, tilts anteriorly, and moves into abduction, it is essentially riding up and over the thorax [28]. This maneuver tilts the coracoid anteroinferiorly and moves it lateral to the midline. This heightened protraction potentiates contracture of the short head of the biceps and pectoralis minor. As the muscles tighten, they increase the static malposition and anterior tilting of the scapula. Additionally, a tight posterior shoulder capsule can exacerbate scapular malposition as the scapula is pulled anteriorly during the follow-through phase of the throwing motion.

Patients with SICK syndrome typically have difficulty performing complete forward flexion given the scapular protraction. The examiner can perform the scapular retraction test to evaluate for scapular dyskinesis. In this test, the examiner manually repositions the scapula in a retracted position allowing full forward flexion without pain. This maneuver is diagnostic for SICK scapula syndrome [6]. Treatment of throwing athletes with SICK scapular syndrome and scapular dyskinesis starts with a period of active rest and ces-

sation from overhead activities. Focused anterior shoulder girdle stretching, with an emphasis on stretching the pectoralis minor, combined with posterior capsular stretching is started immediately. An isometric strengthening program is started for the posterior scapular muscles initially. As scapular control improves, a progressive strengthening program with closed chain isotonic exercises is initiated and lastly open chain isotonic exercises. The goal is to restore scapular positioning and decrease pain with activities. This nonoperative treatment is typically successful after 2–3 weeks [6, 12]. The throwing athlete should be encouraged to continue these strengthening and stretching exercises to prevent recurrence of pain.

The “shoulder at risk” is an asymptomatic shoulder that demonstrates signs of GIRD, malpositioning of the scapula (SICK), or both conditions. The kinetic chain must be inspected with vigilance as something as seemingly trivial as an ankle sprain may translate to increased demand (and injury) in distal segments.

Early recognition of these conditions is crucial to prevent the natural course of pathology and avoid surgery.

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