The Disabled Throwing Shoulder: Spectrum of Pathology—10-Year Update


Abstract: In the 10 years since the current concept series entitled “The Disabled Throwing Shoulder: Spectrum of Pathology” was conceived and written, many studies have been reported that add much more information to the understanding of the disabled throwing shoulder (DTS). The editors of Arthroscopy and the authors of the original series believed that an update to the original series would be beneficial to provide an organized overview of current knowledge that could update the thought process regarding this problem, provide better assessment and treatment guidelines, and guide further research. A dedicated meeting, including current published researchers and experienced clinicians in this subject, was organized by the Shoulder Center of Kentucky. The meeting was organized around 5 areas of the DTS that were highlighted in the original series and appear to be key in creating the DTS spectrum and to understanding and treating the DTS: (1) the role of the kinetic chain; (2) the role and clinical evaluation of the scapula; (3) the role of deficits in glenohumeral rotation, glenohumeral internal rotation deficit, and total range-of-motion deficit in the causation of labral injury and DTS; (4) the role of superior labral (SLAP) injuries and rotator cuff injuries; and (5) the composition and progression of rehabilitation protocols for functional restoration of the DTS. The meeting consisted of presentations within each area, followed by discussions, and resulted in summaries regarding what is known in each area, what is not known but thought to be important, and strategies to implement and enlarge the knowledge base.

The series of articles entitled “The Disabled Throwing Shoulder: Spectrum of Pathology”1-3 presented a systematic, holistic overview of the known mechanics of the overhead throwing motion and a proposed relation of the mechanics to the dysfunction associated with injuries to the shoulder. Through its emphasis on the kinetic chain contributions to force generation for the motion, possible sequencing of the pathophysiologic “cascade of injury,” treatment of all of the components of the intra-articular injuries, and the role of rehabilitation...
in correcting all the deficits, this series suggested a common starting point for discussion of these injuries and for future research.

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**Kinetic Chain, Scapula, and GH Rotation Deficits**

The DTS is most frequently the result of a process that involves deficits in body segments that are proximal to the shoulder and affect the shoulder joint anatomy.

**Role of Kinetic Chain**

The evidence that the overhead throwing motion is developed and regulated through a sequential coordinated and task-specific kinetic chain of force development and a kinematic chain of sequential body positions and motions is clearly established. This allows a unifying concept to understand the mechanics of the overhead throwing motion. The term “kinetic chain” is used collectively to describe the results of both of the chains. The kinetic chain has several functions: (1) using integrated programs of muscle activation to temporarily link multiple body segments into 1 functional segment (e.g., the back leg in the cocking stance and push off and the arm in long-axis rotation before ball release or ball impact) to decrease the degrees of freedom in the entire motion; (2) providing a stable proximal base for distal arm mobility; (3) maximizing force development in the large muscles of the core and transferring it to the hand; (4) producing interactive moments at distal joints that develop more force and energy than the joint itself could develop and decrease the magnitude of the applied loads at the distal joint; and (5) producing torques that decrease deceleration forces.

The clinical implications of the use of the kinetic chain for performance have been reported in several studies. At ball release, only 4 body motions—trunk rotation, shoulder internal rotation, elbow extension, and wrist flexion—contribute positively to ball velocity. However, all of the positive contributions to elbow extension and wrist flexion are by interactive moments produced by proximal sequences of motion and position. In tennis, maximal speed of the racquet at ball impact is correlated most highly with the velocity of the back hip going from its lowest position in cocking to its highest position in follow through. The difference in velocity of a baseball throw from slow to fast is most correlated with the speed of trunk rotation. The maximal velocity of a ball throw is correlated to the extent of body segments used, with the slowest being arm use alone and the fastest being use of the legs and trunk. The sequential use of 5 key body positions and motions is associated with maximal velocities and minimal joint loads. The fastest tennis serves are associated with a muscle activation pattern that uses the back leg muscles pushing up and through ball impact. Finally, the muscle pattern in baseball throwing involves sequential activation, both in onset timing and peak activation, of the contralateral hip and oblique and abdominal muscles, proceeding to the posterior trunk muscles and then to the scapula and shoulder muscles, ending in the proximal arm muscles.

Clinical implications for injury risk have also been reported. A mathematical model showed that a 20% reduction in trunk kinetic energy development resulted in a requirement of 33% more velocity or 70% more mass in the distal segments to maintain the same energy at ball impact. A tennis study showed that not adequately flexing the knees in the cocking phase while serving resulted in a 17% increase in shoulder load and a 23% increase in elbow valgus load if resultant ball velocity was maintained at speeds comparable to those in players flexing the knees. Decreased hip range of
motion (ROM) is associated with shoulder injury and poor throwing mechanics. Scapular dyskinesis is associated with rotator cuff disease, impingement, and internal impingement.

Methods of evaluation of kinetic chain function have been devised. Evaluation of kinetic chain function as a whole is difficult, because of the multi-segment, dynamic activity. Breaking the motion down into key positions and motions that can be analyzed by video or direct observation has been helpful. The baseball pitching motion can be evaluated by analyzing a set of 5 progressive positions and motions (Table 1). The tennis serve motion can be evaluated by analyzing a set of 6 “nodes,” or positions and motions that are correlated with optimal biomechanics (Table 1). These key positions have been correlated with optimal force development and minimal applied loads and can be considered the most efficient methods of coordinating kinetic chain activation. There may be multiple individual variations in other parts of the kinetic chain, but these are the most basic and the ones required to be present in all motions. Clinical evaluation of flexibility and strength in the proximal kinetic chain segments can be helpful in determining deficits. Good screening examinations for hip/leg capability are the single-leg stability series, 1-leg stance, and 1-leg half squat. Direct measurement of knee, hip, and back flexibility; strength; and rotation can be performed by standard physical therapy measurements. A specific inclusive profile for evaluating anatomic areas most highly associated with shoulder injury, the High Performance Profile, has been developed by the United States Tennis Association (http://www.usta.com/sportsmedicine).

In summary, the kinetic chain is the mechanism by which the body most effectively meets the demands of the overhead throwing motion. The body works as a unit in performance and may fail in multiple places in injury.

A comprehensive evaluation program is necessary to evaluate the thrower, both in terms of injury potential and understanding all alterations in injury. This will identify the components of the “non-shoulder” deficits that are contributing to the shoulder injury and whose restoration has been shown to maximize shoulder rehabilitation in addition to the shoulder deficits.

However, there are limitations in the knowledge regarding the kinetic chain. Although the kinetic chain alterations are well documented in association with injury and as negative contributors to optimal mechanics, it is unclear when these occur in the injury sequence and by what mechanisms they affect the DTS; moreover, the natural history of how they occur is unclear. It is not known whether they are exclusively a cause of pathology or whether they are an effect of other pathologies. It is also not known whether early identification and correction of the alterations have significant impact on improved performance or decreased injury incidence. Long-term longitudinal studies are needed to supply this information.

Further studies should be performed to completely characterize the kinetic chains of all overhead throwing activities. Easily implemented and reliable clinical evaluation methods for kinetic chain capability must be established, data on the cause-effect relation to the DTS should be developed, and specific prevention studies should be organized.

### Table 1. Key Positions and Motions for Baseball Pitch and Tennis Serve

<table>
<thead>
<tr>
<th>Task</th>
<th>Node</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseball pitch</td>
<td>Weight on back leg with trunk straight Hip and trunk synchrony Elbow at or above 90° of abduction with scapular retraction and hand on top of ball Front foot directly toward home plate Long-axis rotation: shoulder internal rotation and forearm pronation</td>
</tr>
<tr>
<td>Tennis serve</td>
<td>Use of back foot to push off Knee flexion &gt;10° Back hip counter-rotation away from net and downward tilt Trunk rotation away from net, with hip/trunk separation angle of 30° Arm cocking in scapular plane Long-axis rotation</td>
</tr>
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advanced rehabilitation can be achieved by prone exercises\textsuperscript{46} and kinetic chain diagonal exercises.\textsuperscript{47}

To help establish the presence or absence of scapular dyskinesis in DTS patients and to thereby guide rehabilitation efforts, an effective clinical observation method has been developed.\textsuperscript{48–51} This method, recommended after a consensus meeting,\textsuperscript{50} has clinical utility similar to other tests of shoulder pathology and has been correlated with biomechanically determined scapular positions and motions in symptomatic patients.\textsuperscript{51}

The testing protocol starts with evaluation of inflexibilities (pectoralis minor\textsuperscript{52} and humeral rotation deficit\textsuperscript{53}). The scapular resting position and dynamic motion can be evaluated by observing the scapula as the arms move into forward flexion and descent.\textsuperscript{50} Medial border prominence is characterized as “yes,” seen, dyskinesis present, or “no,” not seen, dyskinesis not present. Finally, the effect of dyskinesis on symptoms can be estimated by corrective maneuvers. The scapular assistance test creates posterior tilt and can decrease external impingement symptoms.\textsuperscript{54,55} The scapular retraction test\textsuperscript{54} or scapular reposition test\textsuperscript{56} creates external rotation and posterior tilt and can increase demonstrated rotator cuff strength and decrease internal impingement symptoms in labral injury.\textsuperscript{56,57}

A rough measurement of pectoralis minor tightness may be obtained by having the patient stand against the wall and measuring the distance from the wall to the anterior acromial tip. This can be performed with a “double-square” device with the patient standing with his or her back against the wall.\textsuperscript{58} A bilateral measurement is taken (in inches or centimeters) to determine whether there is a notable difference between the involved and noninvolved shoulder, with a side-to-side asymmetry greater than 3 cm considered abnormal. A more direct measurement can be obtained by measuring the distance from the coracoid process to the fourth rib at the rib/sternum articulation.\textsuperscript{52}

In summary, the scapula plays key roles in shoulder function and the DTS. A clinically effective method of evaluation has been developed, and specific effective rehabilitation protocols exist.

However, the precise correlation between scapular position and injury is not known. It is unclear whether scapular position is a cause or an effect, where in the causation cascade it exerts its effects, and how much alteration of the kinematics creates the pathologically related dyskinesis. It is also not known whether identification or correction of dyskinesis is an effective injury prevention activity.

The most important future development would be to move the evaluation process from a subjective evaluation that can reliably establish the presence or absence of dyskinesis to an objective method that would also allow measurement and better determination of the effects of treatment. Current investigations using a combination of single planar measurements may provide an adequate system. In addition, further information should be developed regarding the exact cause and effect between scapular dyskinesis and the DTS.

**GH Rotation Deficits**

GH rotation has been identified as a key component in normal and abnormal throwing mechanics. Alterations in the magnitude of GH rotation are the most consistently found deficits associated with shoulder injury. Glenohumeral internal rotation (GIR) has received the most attention, because it has been identified as key to normal force development\textsuperscript{4,6}; when altered beyond certain levels, it changes GH kinematics\textsuperscript{1,59,60} and it is implicated in shoulder\textsuperscript{30,61} and elbow injury.\textsuperscript{62} GIR is usually found to be asymmetrically decreased on the dominant side of throwers and servers. Glenohumeral external rotation (GER) has not been associated with injury but is a key component of thecocking position coupled with shoulder horizontal abduction and scapular retraction. It is usually found to be asymmetrically increased on the dominant side. The total rotational range of motion (TROM) (GER plus GER) is a useful concept to better understand the adaptations in rotation that occur and gives early evidence of potentially deleterious alterations in rotation. Most studies show that TROM is symmetric in throwers and servers but should not exceed 186° as an absolute number.

Alterations in GIR and TROM become clinically significant regarding injury risk when their magnitude becomes associated with shoulder injury. The consensus of the meeting is that the terms “GIRD” and “TROMD” should be associated with only those rotation values at the level of magnitude associated with injury and injury risk. However, there is lack of consensus about what the specific threshold values should be. Traditional definitions suggested a side-to-side asymmetry in GIR greater than 20°. However, studies have shown that a GIRD of as little as 11°\textsuperscript{63} and 18°\textsuperscript{61} is associated with shoulder injury. In addition, a prospective study showed that a GIRD of 18° was related to a 1.9 times increased risk of injury.\textsuperscript{61} These values would also take into account the alteration in GIR due to osseous adaptation. However, because TROM should be symmetric, relatively small changes in symmetry of this number are likely significant. A 5° asymmetry in TROM has been shown to be predictive of increased injury risk.\textsuperscript{61} The consensus of the meeting attendees is that GIRD will be defined as side-to-side asymmetry greater than 18° and TROMD will be defined as side-to-side asymmetry greater than 5°. Although GIRD and TROMD may be considered predictive for shoulder injury, they are not causative by themselves. They alter the normal GH kinematics and place increased load on adjacent joint structures, and they may be considered components of the shoulder at risk \textsuperscript{59,60}

Since alterations in GIR were first reported\textsuperscript{64} multiple studies have been conducted further describing it. GIR
has been shown to decrease with years of throwing exposure and acutely after a throwing exposure. GIR and GIRD have also been shown to be different in the amount of change over 1 season between starters and relievers. These data show that GIR, TROM, GIRD, and TROMD are dose or exposure dependent.

Multiple studies have focused on possible bony, capsuloligamentous, and muscular factors for the observed and verified findings. Bony changes have been well defined. Differential rotational strain has been shown to alter the proximal humeral morphology, producing a slowing of the normal antetorsion process in the maturing bone, resulting in greater retrotorsion (Chuck Thigpen, data presented at summit). This change in torsion is reflected in a corresponding loss of GIR (C.T., data presented at summit). The 7 studies that have examined this difference show an average of an 11° asymmetric decrease in GIR with a corresponding increase in GER. It has been suggested that this is a positive adaptation, with the increased GER allowing better ability to achieve the cocking position. More recent studies, presented at the dedicated meeting, suggest that the bony changes that create retrotorsion may increase the posterior capsular and muscular strain as the arm moves into deceleration and create more soft-tissue tension alteration (C.T., data presented at summit). This would decrease the margin of safety in total internal rotation so that further losses in GIR from muscular or soft-tissue causes may be significant. This may explain the variability in the amount of GIR loss that has been associated with labral injuries. From a measurement standpoint, the bony changes alter the magnitude of degrees of GIR and GER, so a 10° to 12° side-to-side asymmetry in GIR should not be considered pathologic, as long as the TROM is symmetric.

Capsuloligamentous changes that have been assumed to result from scar tissue related to chronic tensile overload have been well documented to alter GH kinematics. Experimental capsular plication has been shown to cause the humeral head to displace anteriorly/superiorly in forward flexion and posteriorly/superiorly in cocking. Thickened posterior capsules are commonly seen on imaging of patients with superior labral injuries and clinical examination and therapeutic techniques directed at detecting and correcting capsular components of joint mobility have been demonstrated. These changes do not account for the high percentage of patients without thickened capsules on imaging, for the acute changes in the magnitude of GIR after a throwing exposure, or for the frequently rapid alteration in GIR measurements with stretching programs. These more dynamic changes point to a muscular component in causation.

The muscular component is related to acute and chronic responses to strain, which then affect joint ROM. An acute response to repetitive tensile strain in eccentric muscle contractions, actual “sarcomere popping,” with separation of the sarcomere ends, has been described. This muscle damage releases intramuscular calcium, which mediates fibril contraction, resulting in muscle shortening. This process peaks between 4 and 18 hours after throwing and is modifiable with gentle stretching at 12 hours. One chronic muscle response to muscular strain is represented by thixotropy, an increased muscle stiffness that is mediated by exposure history of the muscle. Repetitive exposure to strain can result in increased stiffness within the muscles that can affect joint motion and is not related to neurologic changes. The thixotropic response is modifiable by stretching within certain ranges and velocities. Another muscle response is related to the architectural design of the rotator cuff muscles. The pennation angle of these muscles makes them most efficient at producing force through a short mid ROM in abduction and internal rotation but also means that they are poorly adapted to withstand large changes in length. Experimental data suggest that increases in strain rate or strain length of these muscles beyond their normal physiological limits will induce muscular responses that increase internal stiffness and decrease ROM. Data presented at the dedicated meeting showed that an exercise program that increased posterior shoulder muscle eccentric endurance and increased resistance to length change was associated with no loss of GIR through 1 baseball season (S.M., data presented at summit).

Measurement accuracy of GH rotation is required for evaluating these adaptations, because repeated measurements are required, and changes of 5° to 10° may be meaningful. Most clinical studies now rely on the same protocol, with the arm at 90° of abduction in the plane of the scapula and using a bubble goniometer or digital inclinometer. Each athlete should be placed in a supine position on a flat, level surface. A second examiner should be positioned above the athlete to properly stabilize the scapula during testing by applying a posteriorly directed force to the coracoid to ensure that scapular movement does not occur. The humerus is supported on the surface with the elbow placed at 90° and the arm on a bolster in the plane of the scapula. The following landmarks should be identified before placement of the goniometer: the fulcrum set at the olecranon process of the elbow, the stationary arm perpendicular to the table as documented by the bubble on the goniometer, and the moving arm in line with the styloid process of the ulna. The athlete’s humerus is then passively moved into internal rotation. Rotation is taken to “tightness,” a point where no additional GH motion occurs unless the scapula moves or the examiner applies extra rotational stress. For external rotation, similar methods are...
used. TROM is then calculated as GIR plus GER. The procedures are repeated bilaterally to obtain measurements from both the throwing and non-throwing shoulder. This method has been shown to be highly reproducible, with a test-retest reliability of 0.9670 and sensitivity to detection of meaningful change of 3°.

The major gap in the knowledge regarding GIRD and TROMD relates to the exact soft-tissue mechanisms that create the motion loss and how much the bony adaptations affect the “safe zone” of altered GIR. More precise knowledge regarding the pathophysiology could guide rehabilitation protocols to alter the progression of the deficit, provide the best conditioning to prevent the deficit, and provide information about when GIRD is contributing to the “shoulder at risk” of injury.1 More information should also be developed correlating correction of GIRD and TROMD with change in injury incidence.

Specific intervention strategies to modify the changes in GIR and TROM, as well as to correct GIRD and TROMD, should be better developed. Exercises need to include decreasing capsular stiffness and increasing capsular mobility, decreasing muscular stiffness, and increasing muscle eccentric strength and endurance. Another issue is the timing of these interventions: before the throwing/serving episode, immediately after the episode, between episodes, or a combination of all.

Longitudinal studies tracking GIR, GER, TROM, GIRD, and TROMD over the course of 1 or 2 seasons are needed to completely understand the effect of athletic exposure on shoulder motion. “Microcycles” of change over a single athletic exposure or several athletic exposures can be performed multiple times within a season to determine whether patterns of change and recovery could give clues to impending injury. Histologic evaluation of capsular tissue could quantify the location and composition of the capsular component. In addition, the effect of a dedicated program of identifying and modifying GIRD and TROMD on injury incidence and performance should be completed. Emphasis should be placed on understanding the best balance of true stretching, tissue mobilization, and eccentric strengthening and endurance exercises in the modification program. These investigations should be performed for all overhead sports in which GIR and TROMD have been identified.

Labral and Rotator Cuff Injuries: How Much Treatment?

Superior labral and rotator cuff injuries are the most common GH pathoanatomic problems associated with dysfunction in the DTS. However, how these alterations in anatomy contribute to the symptoms is not well understood, with consequent uncertainty about evaluating and treating the observed findings. Information has been developed regarding the known roles of the labrum in normal shoulder function and how injuries of the labrum and rotator cuff can be most effectively evaluated. In addition, it appears that in higher-level throwers, some of the observed pathology may be adaptive and necessary for function.

The approach to treatment of throwers with shoulder pain has improved because the mechanisms behind the development of pathology are better known. However, many athletes are unable to return to throwing after surgery, despite near-anatomic restitution of their presenting pathology.

Review of the mechanics behind pitching can provide insight into how pathology seen in the thrower’s shoulder develops, the approach to observed pathologies, and the reported rates of success. From this perspective, some of the pathology seen in the thrower’s shoulder may be adaptive, in that the presence of the pathology may be required for an athlete to throw at elite levels, and anatomic restoration is not only not required but may be detrimental.

Pathomechanics of Throwing

Throwing is an extreme skill that requires the GH joint to achieve incredible positions and experience exceptionally high angular velocities and forces.14,92 To throw at high velocities, it has been recognized that a thrower must achieve certain nodes, or body and arm positions, in a specific progression to optimize this skill.19,92 Achieving these nodes optimizes proper pitching mechanics and may reduce injury. In addition, because it has been shown that the velocity of the pitched ball correlates with the amount of external rotation of the abducted arm in the late cocking phase of throwing, these nodes likely optimize the body’s ability to maximize external rotation.93-95 The forces and position of the shoulder at maximal external rotation are impressive,14 with the arm in a position of 165° ± 11°. This position has been called a “critical moment” in throwing and is thought to be a potential source of the development of pathology in the thrower’s shoulder.14,96,97 Numerous studies have shown this position to be responsible for high strain in the superior labrum98 and the development of SLAP lesions in cadaveric shoulder models.99,100 With regard to the rotator cuff, this position of maximal GH joint external rotation is thought to produce articular surface partial-thickness rotator cuff tears through compressive loads of internal impingement101,102 or, alternatively, through external rotation tensile overload of the capsular portion of the rotator cuff.96,103 The repeated torsion in external rotation during throwing also leads to the remodeling of the thrower’s humerus in external rotation torsion.71,72,75,76,104 Bony remodeling also occurs on the scapular side of the dominant arm of the thrower with increased glenoid retroversion.71,74 Finally, physical examination tests of asymptomatic professional baseball players show asymmetry in inferior laxity as
Labral Roles and Injuries in Throwers

Andrews107 was the first author to report lesions of the superior glenoid labrum in throwing athletes, and Snyder et al.108 reported this lesion in other populations and developed the widely adopted and sometimes modified classification system for SLAP tears. This system is observational and is based on arthroscopic visualization and criteria. As experience with this lesion increased, its importance as a key factor in the DTS was identified1,39,80,109,110 and surgical techniques to treat this lesion were developed. This lesion is also being diagnosed and treated more frequently in non-throwing populations. Current American Board of Orthopaedic Surgery data and New York State data indicate a large increase in the number of SLAP surgery repairs in the last 10 years, from 4 per 100,000 population to 22 per 100,000 population11; moreover, such repairs represent 10% of all shoulder procedures and have become the second most common shoulder arthroscopic procedure.112

There is increasing evidence that SLAP lesions are being poorly diagnosed, overdiagnosed, or misdiagnosed, with resulting poor outcomes for diagnosis and treatment. The studies providing this evidence report on the imprecision of physical examination tests,113 diagnostic imaging,114 and arthroscopic evaluation115 as sole determinants of the diagnosis and show variable results from surgical repair.116-118 Basic to correcting these outcomes is a better understanding of when the anatomic alteration that is called a SLAP injury is contributing to shoulder dysfunction (a clinically significant SLAP injury) and how it should be treated to restore the role of the labrum in shoulder function.

Labral roles in shoulder function have been traditionally identified as an attachment site for the biceps, a bumper to deepen the GH socket and to improve stability by minimizing GH translation, and an aid in increasing capsular tension.119 However, labral roles may actually be more complex. The actual role of the superior labrum as a mechanical bumper is controversial. The amount of increased mechanical GH translation after superior labral resection was only 10%, meaning that the superior labrum likely has functional roles other than just a mechanical stability role.120

Recent biomechanical studies have highlighted 3 other important functions for the labrum: (1) as a deformable structure with high compliance interposed between 2 surfaces to more evenly distribute contact pressures between the surfaces, increase boundary lubrication, and maximize concavity/compression characteristics, much like a washer between 2 surfaces; (2) as a pressure sensor maximizing proprioceptive feedback; and (3) as an attachment site for muscles and ligaments, to optimize their tension.119,121 Experimental labral release resulted in significant changes in capsular tension.119

Another cutting study showed increased GH translation that was restored to normal by posterior superior labral repair (Anthony Romeo, data presented at summit).

The intact labrum would result in optimal GH kinematics in dynamic shoulder motion, resulting in smooth GH motion in rotation, stable ball-and-socket kinematics, and maximal force transfer from the engine of the core and legs through a stable linkage at the shoulder to the delivery mechanism, the hand. The labrum should be seen as a key component for functional GH stability.

Many labral “injuries” diagnosed on imaging are not clinically significant, in that they are not contributory to the symptoms or dysfunction in the DTS. The clinically significant SLAP injury is one in which the anatomic alteration in the labrum results in elements of the clinical history of the dysfunction that can be attributed to the loss of labral roles, and the injury can be highlighted by specific physical examination tests that are clinically useful for detection of the injured labrum. It is a positive diagnosis, not a catch-all term in the presence of shoulder pain of unknown etiology.

The history findings suggestive of loss of labral roles include pain on external rotation/cocking, indicating increased posterior-superior translation1,120,121; weakness in clinical or functional arm strength, indicating pain and/or increased translation; symptoms of internal derangement (clicking, popping, catching, sliding), indicating loss of the bumper effect or washer effect, or decreased capsular tension; and a feeling of a “dead arm,” indicating loss of proprioceptive feedback, decreased capsular tension, and increased translation. These are not exclusively seen in a labral injury but point toward the loss of labral roles.

The literature, as well as the consensus of the meeting attendees, suggests that there is not a specific clinical test that conclusively shows a clinically torn labrum or distinguishes the clinically significant SLAP tear. However, a comprehensive examination will show local and distant physiological and biomechanical deficits that are commonly associated with SLAP injury, need to be addressed in treatment, and have been found to decrease the DTS dysfunction. In addition, there are labral examination tests that can provide intra-articular clues to the presence of a labral injury.

Kinetic chain deficits are discovered on examination in a majority of patients with SLAP injuries. Deficits in hip abductor or extensor strength, deficits in hip rotation flexibility, or core strength weakness have been identified in 50% of SLAP injuries.3,31,80 They can be identified by the methods described in the section on the kinetic chain.31,32,122 Scapular dyskinesis is frequently seen in patients with labral injuries, as has been described in the section on scapular dyskinesis.1,30,42
Of the labral examination tests, the modified dynamic labral shear test has been shown to be of high clinical utility in the evaluation of labral injuries when the test is performed in the manner described. It is performed by abducting the arm and flexing the elbow to 90°. The arm is then abducted in the scapular plane to above 120° and externally rotated to tightness. A shear load is applied to the joint by maintaining external rotation and horizontal abduction and lowering the arm from 120° to 60° of abduction. A positive test is indicated by reproduction of the pain and/or a painful click or catch in the joint line along the posterior joint line between 120° and 90° of abduction. The test has a sensitivity of 0.72, specificity of 0.98, positive predictive value of 0.97, and positive likelihood ratio of 31.6. Other labral examination tests advocated include the O’Brien active compression test, the relocation test with pain as the indicator, and an anterior levering maneuver to place a posterior load and shear (Richard Hawkins, data presented at summit).

GIRD and TROMD are seen in virtually all patients with SLAP injury and have been found to be predictive of future injury in asymptomatic patients. Precise, reproducible measurement is possible with specific protocols that stabilize the scapula and measure GH motion with either a goniometer or inclinometer. Scapular forward posture has been implicated as a negative factor for success of nonoperative treatment in SLAP injury (S.M., data presented at summit). This posture results from weaker posterior scapular muscles and tight pectoralis minor muscles. Clinical assessment by observation and palpation of the tight anterior structures and measurement by the double-square method can be performed and have been shown to be clinically reliable.

In addition, intra-articular examination tests can provide clues to loss of labral roles. A positive painful arc-of-motion test in the Hawkins-type motion, with no relief by scapular posterior tilt in the scapula assistance test, indicates increased translation. A positive O’Brien maneuver indicates loss of the washer effect, increased biceps tension, and increased translation. The modified dynamic labral shear test has been shown to have high clinical utility because it specifically replicates a peel-back phenomenon. A positive test indicates loss of biceps stability, loss of the washer effect, and increased translation.

In summary, the physical examination can provide findings that help construct a comprehensive picture of the anatomic and physiological deficits that exist and may cause or exacerbate the SLAP injury and that should be the focus of a comprehensive rehabilitation program.

**Imaging**

The labral injury can be confirmed by magnetic resonance imaging (MRI), MRI arthrography, or computed tomography (CT) arthrogram but should not be defined by imaging. Specific criteria have been developed to distinguish a labral alteration, but MRI is best viewed as a static estimation of labral status with inconsistent relation to the dynamic roles. A percentage of patients will show “labral tears” without symptoms relating to loss of the labral roles.

MRI studies in asymptomatic throwers have shown that cuff disease (including surprisingly high levels of partial-thickness rotator cuff tears) and effusions—findings that would normally be considered pathologic—are often seen in an asymptomatic thrower’s shoulder and may not be a source of symptoms. The aphorism “treat the patient, not the X-ray” is particularly relevant in the thrower with DTS.

The clinically significant SLAP injury as defined by the history, physical examination, and/or imaging that differentiates it from other sources of shoulder pain needs to be treated, but surgical treatment is rarely the first option.

**Nonoperative Treatment of Labral Tears**

Though often recommended and performed, rehabilitation has not been widely studied, nor has its outcomes. There is only 1 case series in the literature describing the results of nonoperative treatment of SLAP lesions. Edwards et al. collected data using a survey with validated outcome measures in 50 of 305 patients with labral tears (response rate, 16.4%). In this group 39 patients had symptomatic SLAP lesions diagnosed by a positive O’Brien test, pain at the bicipital groove, and MRI with greater than 1 year of follow-up. All patients were treated nonoperatively with nonsteroidal anti-inflammatory drugs and a physical therapy protocol focused on scapular stabilization and posterior capsule stretching. This protocol was considered successful in 19 (49%). In this group 18 patients were active in sports before treatment and 15 performed overhead athletics. Of the 15 overhead athletes, 10 (67%) were able to return to overhead athletics after nonoperative treatment.

Data presented at the dedicated meeting summarized the findings for a Major League Baseball organization (David Lintner, data presented at summit). These players all had SLAP lesions and underwent physical therapy focusing on addressing the posterior capsule contracture (GIRD), as well as scapular rehabilitation. Surgery was offered if the thrower had persistent pain despite improvements in GH motion and scapular dyskinesis. Nonoperative treatment at the Minor League level had failed in many athletes, and they repeated the physical therapy program if they presented to the head team physician with GIRD and scapular dyskinesis. Over a 10-year period, 115 shoulder-related events required evaluation by the head team physician,
Surgical Treatment of SLAP Lesions

The results of current methods of surgical treatment for superior labral tears in the thrower’s shoulder are summarized in Table 3 (available at www.arthroscopyjournal.org). If return to play at the same level or a higher level is used as the outcome measure of interest, only 5 of 22 series reported success rates at or above 85%. As shown, there is a great degree of variation, with successful outcomes ranging from 22% to 94%. This likely relates to the low levels of evidence of these series, because there is no controlling for acuity of the SLAP lesion, location of the SLAP lesion, presence of additional pathology, indications for surgery, surgical technique, postoperative rehabilitation protocols, and willingness of the athlete to return, all of which may affect the outcome. These poor results point to the need for a more detailed understanding of what intra-articular pathology needs to be treated and how it should be treated.

If arthroscopy is recommended, the arthroscopic evaluation of the suspected labral injury must be specific to understand and treat the labral injury properly. The arthroscopic findings most frequently associated with a clinically significant labral injury include (1) a type II or higher lesion, denoting loss of attachment from the glenoid; (2) a peel-back phenomenon, indicating labral detachment, increased compliance, loss of washer effect, and loss of bumper effect; (3) glenoid articular cartilage damage or chondromalacia, indicating increased translation; (4) loss of capsular tension, indicated by a drive-through sign, or loss of tension in the posterior band of the inferior glenohumeral ligament; (5) increased posterior labral thickness, indicating increased translation and shear with compression on the labrum; and/or (6) excessive posterior inferior capsular thickness and scar, indicating end-stage capsular damage that helps create GIRD. Care must be taken to differentiate labral detachment from anatomic variants such as sublabral foramina, a Buford complex attachment of the middle GH ligament, or a meniscoid-like labral attachment that does not peel back.

On the basis of these principles, arthroscopic treatment guidelines for labral injury include (1) evaluation of the peel-back phenomenon, labral injury and mobility, glenoid surface, and capsular tension by direct visualization; (2) preparation of the glenoid to maximize bone-to-labrum healing; (3) multiple anchor placement to secure at least 2-point fixation of the labrum on the posterior-superior glenoid (10:30 and 11:30 clock-face positions in a right shoulder) (a double-loaded single anchor is still only 1-point fixation); (4) placement of enough posterior-superior anchors to eliminate the peel-back phenomenon; (5) evaluation of biceps mobility after anchor and suture placement to make sure there is adequate motion of the biceps in shoulder external rotation; (6) rare placement of anchors and sutures in the anterior-superior glenoid (12:00-o’clock position to 2:30 clock-face position in a right shoulder) to reduce the chance of biceps tethering; (7) evaluation of the effect of the labral repair on capsular tension by evaluation of the tautness of the posterior band of the inferior glenohumeral ligament and elimination of the drive-through sign; (8) assessment of total GH rotation to ensure that no external rotation has been lost; and (9) treatment of the associated pathology in the joint.

Surgical Treatment of Rotator Cuff Tears

The surgical treatment of rotator cuff tears in the throwing athlete has received more attention, but again, most studies have low levels of evidence (Table 4, available at www.arthroscopyjournal.org). Three different methods have been used to surgically treat partial-thickness rotator cuff tears: debridement; repair of delaminations in situ, without repair to bone; and repair of the partial tear to bone. In the high-demand population of throwing athletes, by use of return to play at the same level or a better level as a primary outcome, the results of surgical treatment of partial-thickness rotator cuff tears show moderate success. Debridement produces return-to-throw rates of 16% to 85%. Again, in these case series, there is no controlling for the nature of the rotator cuff tear, the concomitant pathology, the indications for surgery, the postoperative management, or the athlete’s willingness to return.
There are few data on repair of delaminations without repair to bone, but the early results seem promising, with return-to-play rates of 89%. When a throwing athlete has a partial tear that is repaired to bone, the return-to-play rates seem less optimal, with only one-third returning to the same level of play. In a similar vein, repair of full-thickness rotator cuff tears to bone in throwing athletes is met with dismal return-to-throw rates (8% for professional pitchers) (Table 5, available at www.arthroscopyjournal.org). These data suggest that the anatomic restoration of the rotator cuff has led to poorer outcomes than a debridement or repair in situ.

Concept of Adaptive Pathology

Throwing requires repetitive high loading of the osseous and soft-tissue structures. To achieve the extremes of external rotation required for throwing with high velocity, these structures undergo adaptive remodeling and possibly failure. The fact that MRI scans of asymptomatic throwers show significant findings that would normally be considered the source of the athlete’s pain suggests that these athletes have developed these anatomic alterations to throw at high levels, just as their humerus has remodeled into greater retroversion. Chronic SLAP lesions in throwers may allow for increases in external rotation required for throwing. Articular-side partial-thickness rotator cuff tears may represent failure of the tissue in external rotation, again allowing for the extremes of external rotation required for high-level throwing. It is conceivable that in some throwers, the anatomic repair of these structures will lead to an inability to achieve the extremes of external rotation required to throw at high velocity and may end their careers. This may explain the relatively poor results obtained with the surgical treatment of the combined labral/rotator cuff pathology. The throwing athlete can be considered on the edge of a cliff in terms of the demands of the throwing motion on the anatomic structures. Treatment of the DTS should put the athlete back on the edge of the cliff and not restore his or her shoulder to “normal” anatomy. This concept leads to a suggested approach to the symptomatic throwing athlete, described in the next section.

Surgery

Surgery is to be used only after extensive and appropriate rehabilitation fails. The rehabilitation must address deficits in internal rotation (GIRD) and total range of motion (TROMD), as well as scapular dyskinesis and kinetic chain deficits. If the athlete improves clinically with regard to GIRD, TROMD, and dyskinesis but still has pain and cannot throw, surgery may be offered in an attempt to salvage his or her career. Surgery should be considered a method to improve the anatomy within the shoulder so that rehabilitation can be successful. When one is performing surgery, a minimalist approach is ideal. In rotator cuff disease, debridement is likely preferable to repair, and if repair is performed, a trans-tendinous repair of the delaminated rotator cuff is likely going to produce better outcomes than repair to bone. With regard to SLAP lesions, repair of the “peel-back” posterior labrum is performed surgically, but it is important to avoid overconstraining the bicep, which serves as an important restraint to external rotation of the abducted arm.

Conclusions

Throwing at high velocity requires extremes of GER of the abducted arm, where extremely high forces are endured. Repeated throwing leads to bony remodeling of the humerus and glenoid. Many elite asymptomatic throwers have findings on MRI that would normally be considered pathologic. On the basis of our understanding of throwing mechanics, these findings, along with the well-reported bony changes that affect the humerus and glenoid, may be adaptive and may allow for the extremes of external rotation that are required for high-velocity throwing. The pathology often treated surgically in the thrower’s shoulder includes partial-thickness rotator cuff tears and SLAP lesions. In some throwers, this pathology can also be considered adaptive, because it allows the thrower to achieve the extremes of motion required to throw at high velocity.

Clinically significant SLAP tears do exist. They represent a relatively small subset of general patients with shoulder pain but are common in intensely competitive overhead athletes as part of the DTS. The optimal integration of clinical history, physical examination, imaging findings, and arthroscopic criteria has not been formulated. Clinical efforts to determine the SLAP injury that is associated with dysfunction should be related to demonstration, by history and physical examination, of loss of labral roles. Treatment guidelines should first emphasize a structured rehabilitation program, because studies indicate that around half of all lesions can be symptomatically resolved through rehabilitation. Surgical guidelines should also emphasize delineation of loss of labral roles and their surgical restoration.

Although the data are limited, nonoperative treatment has been shown to be successful for a number of elite and recreational athletes with SLAP lesions. Rehabilitation should be focused on treating GIRD, TROMD, scapular dyskinesis, and kinetic chain deficits. Surgical results for returning throwers to their former level of play are not as high as we would hope. Throwers who have surgical repair of SLAP lesions with treatment of rotator cuff tears have in past literature shown poor rates of return to play. Throwers with full-thickness rotator cuff tears in the dominant arm who undergo repair to bone also have poor rates of return to play. As a result, surgery should be undertaken if the
thrower continues to have pain despite corrections in GIRD and scapular dyskinesis. A surgical approach should be considered career salvaging, and athletes should be counseled on expectations.

The following principles should be considered when one is performing surgery on the thrower’s shoulder: (1) Surgery should be considered only after extensive and appropriate rehabilitation has failed. (2) The surgical approach should be minimalistic, with the concepts to repair as needed but not to achieve normal anatomic repair of the rotator cuff. (3) Surgery should be considered as a last resort to attempt to salvage a thrower’s career, and throwers must be cognizant of the poor return-to-play rates.

Areas worthy of future study include (1) optimizing surgical techniques for SLAP repair, especially with regard to the location of the anchors, the type of sutures and knots, and postoperative management; (2) identifying the optimal surgical approach to the damaged rotator cuff; (3) decreasing the lengthy convalescence for return after surgery; and (4) identifying the features that distinguish the athlete with adaptive pathology from the athlete who has fallen from the cliff edge and has pathology that limits the ability to throw.

Rehabilitation
Rehabilitation can play a key role in modifying the dysfunction of the DTS and reducing the need for surgery. A clinically applicable rehabilitation and conditioning program to achieve these goals has been outlined (described in the “Recommended Exercise Program for Rehabilitation and Conditioning” section). The program is outlined so that health care providers can use it clinically in the care of their patients with DTS. The primary aim of this rehabilitation approach is to emphasize evaluating and treating the entire body. Often, the source of the problem may not be only at the site of pain; therefore evaluating and treating the entire system are necessary and beneficial for the outcome of the patient. The rehabilitation program addresses 3 primary focus points: kinetic chain, shoulder mobility, and shoulder strengthening. The rationale and evidence, when available, for the interventions are then integrated into the 3-phase exercise program.

1. Kinetic chain
   a. Leg drive and lower extremity strength
   b. Hip mobility
   c. Proximal core stability
   d. Dynamic core strength/power
   e. Scapular stabilization
2. Shoulder mobility
   a. Restoring internal and external rotation deficits to achieve safe total ROM
   b. Addressing joint restrictions in the spine and shoulder complex
   c. Addressing muscular flexibility imbalances in the spine and scapula
3. Arm strength and endurance
   a. Restoring scapular motor control for functional and pain-free motion
   b. Facilitating inhibited or weak scapular musculature
   c. Integrating scapular exercises with functional tasks
   d. Facilitating endurance and eccentric control by advanced shoulder and scapular exercises
   e. Strengthening forearm, wrist, and hand

The DTS usually presents to the physician, physical therapist, or athletic trainer with the primary symptom of pain during throwing. It is well established in young throwing athletes that an increased volume of pitching increases the risk of having shoulder or elbow pain by 2- to 3-fold. Early management would include modification of exposure to throwing, from decreasing throwing to complete restriction if symptoms are severe. The exact duration of this modification or restriction is not known. The consensus is that 2 to 3 weeks would allow acute inflammation to subside and allow for improvement in tissue mobility. It is possible to see measurable strength changes in 3 weeks, primarily because of neuromuscular coordination changes. In practice, athletes frequently will only take a few days off and may have to deal with recurring issues of pain throughout the season. The group consensus recommends setting a window of time that would allow deficits to be addressed and symptoms to resolve. This should be considered a period of active rest and rehabilitation, not complete rest.

Kinetic Chain
During the initial evaluation of the athlete’s kinetic chain, deficits in hip mobility or single-leg stability are likely to be identified. These deficits can be treated during the time of active rest. Nearly 50% of ball velocity comes from leg-forward step and trunk rotation. Addressing deficits found in the trunk and lower extremity is important because the ability to accelerate the elbow and wrist during throwing is due to torque generated more proximally in the trunk. Throwing performance may also benefit by targeting the core musculature, given that increased pelvic velocity during the arm-cocking phase and upper torso rotation during the acceleration phase of throwing is positively related to ball velocity. Limitations of motion of the spine, hip, and the rest of the lower extremity should be addressed. Typical hip motion in various baseball athletes has been studied previously, a summary of these values is presented in Table 6 (available at www.arthroscopyjournal.org) and should be used for re-establishing observed deficits.
Strengthening and stability of the core and lower extremity musculature are critical because of their contributions to the throwing motion. During pitching, the stance or back leg balances the body weight during the wind-up and drives the body forward during the push off. But the lead or front leg requires sufficient leg and hip strength to control the landing force of 72% of body weight. Deficits detected during screening may require further evaluation during rehabilitation to determine whether they arise from lower extremity or core deficits of inflexibility, strength, or lack of balance. Strengthening the core for returning to dynamic movements in throwing is different from prescribing stabilization exercises for spinal disorders. Abdominal muscles activate in an orderly sequence from the ground up through the torso when throwing a ball. Therefore muscles must be stiff enough to stabilize the joints but not too stiff to impede the transfer of energy through the kinetic chain. One exercise does not target all the abdominal muscles; therefore a program that addresses the anterior, lateral, and posterior sides of the torso should be undertaken. Mat exercises emphasizing endurance are good starting points, with consideration given to positions that limit stress to the shoulder. The athlete should be engaged in lower extremity and core exercises such as rotational chop-and-lift activities that simulate the demands of throwing. The use of unstable surfaces, such as stability balls or foam mats, has shown increased core activation and improvement in core strength but has not necessarily shown carryover into functional tasks. Recently, a study in handball players using an unstable closed kinetic chain program that used a sling suspension to train the core reported improvements in throwing velocity of 5%.

The relations established between leg and core motions and kinetic chain force development to improve throwing velocities provide a solid rationale for training the lower extremity and core in specific routines for throwers. The nature of the throwing task, combining stepping and rotating, produces a complex motion that is necessary to simulate in rehabilitation and conditioning for throwing. Specific routines and their effectiveness on increasing mobility, strength, or balance have not been completely developed. Furthermore, the effect of enhancement of these functions on injury prevention is unknown. Dynamic core exercises are recommended to improve performance particularly related to power, but what specific exercises and at what training regimen (number of sets and repetitions) are appropriate for the various levels of throwing athletes are currently unknown.

**Shoulder Mobility**

Individuals with shoulder injury often present with shoulder ROM deficits. Deficits in GIR and TROM can be predictive of future injury. One study identified that patients with resolution of shoulder symptoms displayed greater improvement in glenohumeral horizontal adduction range of motion (GHA) than those with residual symptoms. As such, prevention and treatment of shoulder motion deficits are critical components of an intervention program for throwing athletes.

The goal of the intervention program is restoration of ROM to acceptable values (Table 7, available at www.arthroscopyjournal.org). Deficits in GIR and TROM should be addressed to restore safe ROM values; however, it is important to remember that ROM values are affected by previous throwing activities for at least 24 hours. GIR of the throwing shoulder should be within 18° (range, 13° to 20°) of the nondominant shoulder, TROM in the throwing shoulder should be within 5° of the nondominant shoulder, and TROM should not exceed 186° to avoid an increased risk of injury. The cross-body stretch and sleeper stretch target the posterior shoulder musculature and capsule to effectively improve GIR and GHA. Additional beneficial treatment techniques include joint mobilizations, muscle energy techniques, and soft-tissue mobilization. Assessment and treatment of GHA are becoming more important. A multimodal treatment approach that included posterior shoulder stretching and joint mobilization showed that change in GHA is as important as change in GIR in reduction of symptoms.

Muscular adaptations that can alter scapular kinematics during elevation can occur because of tight anterior structures such as the pectoralis minor. Pectoralis minor tightness has been identified on the dominant side of tennis players and is associated with increased scapular anterior tilt and scapular internal rotation. Pectoralis minor tightness can be effectively treated with corner stretching exercises.

Only 1 study has investigated the efficacy of posterior shoulder stretching in symptomatic patients. Because the exact pathophysiology of shoulder mobility deficits is not known, it is unknown which tissues—musculotendinous, joint capsule, or bony—may be the source of the deficit and therefore should be the target for intervention. The ability to specifically address alterations in each of these tissues will help give insights into the most effective intervention for restoring shoulder mobility. The role of rehabilitation specialists in direct treatment and exercise supervision versus home exercise is not clear because studies have provided mixed results. Further investigation into treatment effectiveness and the level of supervision is needed to maximize effective care for the athlete. Continued investigation into shoulder mobility throughout the season, along with the baseline measures, may help improve the ability to detect overuse injuries that are evolving before the onset of symptoms.

**Arm Strength and Endurance**

The strength and endurance of the athlete’s shoulder musculature are important to allow normal throwing
function. Asymptomatic throwers often present with greater internal rotation strength compared with external rotation strength on the throwing side and typically present with a 3:2 strength ratio when measured isokinetically.182-185 Deficits in shoulder strength are predictive of future injury, particularly in external rotation and supraspinatus activation.186 Athletes presenting with shoulder symptoms are typically found to have strength deficits of the rotator cuff187,188 and scapular musculature,189 which may present with various movement patterns.190 Treatment of these deficits has many common themes and treatment protocols.191 Initial management should focus on scapular musculature control, which facilitates stability to prepare the shoulder musculature for more dynamic and stressful exercises. Repeated emphasis of proper position and movement instructions are important because many of the movement strategies learned are only temporary.192 Establishment of proximal stability such as scapular orientation exercises should precede longer lever-arm activities (i.e., prone horizontal abduction exercises) to establish proximal functional control.33,193 Over-activation of the upper trapezius during elevation, indicating a muscular imbalance around the scapular force couple, has been found in injured shoulders.24,194 Scapular setting exercises have been shown to primarily activate the trapezius musculature193 and can facilitate muscular balance between the scapular force couple of the serratus anterior and upper and lower trapezius.24,39,195 The focus of the scapular exercise program should be on the rhomboid, lower trapezius, and middle trapezius musculature. Facilitation of serratus anterior and lower trapezius musculature can be targeted with isometric adduction and extension exercises with continued focus on proper scapular orientation of posterior tilting and retraction.196

Progression to dynamic exercises that incorporate all shoulder muscles and simulate function is the next stage of rehabilitation. Exercises in this phase are frequently individualized for specific goals, and direct supervision by a rehabilitation specialist is critical to meet the individual’s needs and anticipated functional levels.

Most literature regarding electromyographic activity of shoulder exercises has focused on which exercises activate specific muscles primarily during a particular exercise197-209 or activate them synergistically.210-212 However, studies have also shown how particular exercises affect scapular kinematics to ensure that proper motions are being emphasized46,190,213 and improper motions are de-emphasized.214 Scapular posterior tilting and retraction are facilitated with prone horizontal abduction and prone scapular retraction with the elbows flexed.46

It is well established that strengthening exercises will increase strength, but there is limited information on how these exercises affect sport performance parameters or prevent throwing injuries. Rotator cuff strength and serve velocity have been significantly increased in elite tennis players with 6 weeks of isokinetic shoulder strengthening using concentric and eccentric resistive training approaches.215,216 Recent evidence has shown that incorporating plyometric and eccentric exercises has important benefits in developing power for overhead athletes.217-219 Plyometric exercise training regimens that last at least 10 weeks have resulted in increased throwing velocities of approximately 2% in trained athletes.217,220 In adolescent throwers (aged 11 to 15 years), throwing velocity was significantly increased by 2.2 mph with a 4-week elastic training regimen of 17 exercises performed for 20 to 25 repetitions.221 Throwing velocity can be improved with several intervention approaches. A study comparing 6-week training programs with plyometric exercises, thrower’s 10 isotonic exercises, and pneumatic exercises showed significant improvements in throwing velocity for all 3 programs (range, 1.2% to 2%).222 In a similar group of adolescent baseball players, arm endurance and leg strength significantly increased with a 2-month training program emphasizing endurance training (S.M., data presented at summit). Swanik et al.223 showed that a combination of elastic resistance and isotonic exercises for 6 weeks before competitive swimming season decreased the incidence of reported shoulder pain episodes in 13 athletes in the training group (1.8 ± 2 incidents) compared with a control group (4.6 ± 4.7 incidents).

Several different types of intervention strategy exist in the literature, but none have shown the best outcomes.1,34,80,191,224,225 Better documentation of content and outcomes of intervention approaches in overhead throwing populations could determine which interventions are effective. Randomized controlled trials are difficult to perform in this population but are important to delineate cause and effect of therapeutic interventions and develop insights into the management of this population. Although many therapeutic exercises and protocols have been devised, it is not known which exercises are primary and which exercises are supplementary in recovering function or preventing injuries. Volume of pitching and fatigue increase the risk of development of significant shoulder or elbow injury; therefore it is important that the exercises emphasize fatigue resistance for pitchers.226,227 However, each position has differing demands, so each exercise program may need to focus on endurance (low loads/high repetitions) or on strength and power (low repetitions/high loads). A combination is probably needed, but when and how exercise programs are applied are currently unknown. There is limited, but promising, evidence that other forms of training, such as plyometrics, are beneficial to performance parameters and may have injury prevention effects. Evidence of the effectiveness of eccentric training in managing common
tendinopathies\textsuperscript{,228,229} suggests that the limited studies in the shoulder\textsuperscript{,230,231} require further investigation, because eccentric loads have been implicated as causative factors in maladaptations in the disabled overhead throwing athlete.

**Recommended Exercise Program for Rehabilitation and Conditioning**

In this report we describe an exercise program that is primarily directed toward the nonoperative rehabilitation of athletes with a DTS. It can also be used for postoperative rehabilitation when modified for the healing status of the repair. Finally, these exercises can form a base for conditioning and injury prevention programs. The suggested exercise program is based on literature review and consensus of the clinical experiences among the meeting attendees. Many other specific exercises may also be effective, but they should be used in progressions as outlined. The exercise program is progressive and is divided into 3 phases based on the level of disability and tissue irritability that exist. Movement through the phases is variable and based on achieving functional capabilities rather than adhering to specific time frames. Phase 1, the acute phase, should minimize loads on the injured tissues, so it should focus on scapular and GH muscular activation, particularly in correcting timing of muscular activation to ensure that both the scapular and GH muscles are working synchronously (Figs 1-22, available at [www.arthroscopyjournal.org](http://www.arthroscopyjournal.org)). Phase 2, the recovery phase, should focus on strengthening and restoring core, kinetic chain, and progressive isotonic strengthening (Figs 23-54, available at [www.arthroscopyjournal.org](http://www.arthroscopyjournal.org)). Phase 3, the functional phase, should focus on sport-specific actions and includes endurance and ballistic exercises (Figs 55-80, available at [www.arthroscopyjournal.org](http://www.arthroscopyjournal.org)). Strengthening exercises are oriented toward endurance with an emphasis on higher repetitions and lower resistance. An athlete should demonstrate the ability to perform 3 to 4 sets of 15 to 20 repetitions with correct form before progressing to a greater resistance. Another way to challenge the endurance of the musculature has been introduced and described by Wilk et al.,\textsuperscript{332} called the advanced throwers 10 program. This program challenges the endurance of the athlete with the application of sustained holds and alternating arm motions and can be incorporated in phase 2 or 3. The sequence described below is performed twice without rest between sets for a total of 60 repetitions.

The following exercise example is for a prone horizontal abduction “T” exercise: Set 1 comprises 1 set of 10 bilateral isotonic contractions. Set 2 comprises 1 set of 10 sustained holds with the uninvolved arm in horizontal abduction while the involved arm performs unilateral isotonic contractions. Set 3 comprises 1 set of 10 alternating isotonic contractions between arms with a sustained isometric hold in horizontal abduction from arm to arm.

Daily stretching exercises should be completed either statically or dynamically. Static stretches are to be held for 30 seconds and repeated 4 times. Dynamic stretches are to be performed for 12 repetitions with a minimum hold time of no longer than 2 seconds. All stretching should be performed until a strong pull is experienced but without pain or neurologic symptoms. If these symptoms occur, the patient should move to a less aggressive stretch or see a rehabilitation specialist for further instruction.

**References**


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**Fig 1.** Phase 1: core mobility/stability/balance. Hip flexor tightness: lunge position with erect and extended trunk.

**Fig 2.** Phase 1: core mobility/stability/balance. Hip rotator tightness: quadruped position with hips abducted and knees flexed to 90°.

**Fig 3.** Phase 1: core mobility/stability/balance. Hip adductor tightness: sideways lunge can be performed dynamically or statically.

**Fig 4.** Phase 1: core mobility/stability/balance. Hip rotator tightness: cross-legged, pulling knees to chest.

**Fig 5.** Phase 1: core mobility/stability/balance. Hip extension.
Fig 6. Phase 1: core mobility/stability/balance. Hip abduction.

Fig 7. Phase 1: core mobility/stability/balance. Hip adduction.

Fig 8. Phase 1: core mobility/stability/balance. Hip flexion.

Fig 9. Phase 1: core mobility/stability/balance. Leg strength and balance: step back and up onto a step.
Figure 10. Phase 1: core mobility/stability/balance. Move to "power position" with front leg lifted to 90° of hip flexion (hold for 3 counts).

Figure 11. Phase 1: core mobility/stability/balance. If shoulder is not too painful, place arm in cocked position with palm facing away from target and front elbow bent and pointing toward target; squeeze scapula together (hold for 3 counts).

Figure 12. Phase 1: shoulder mobility and stability. Cross-body stretch for posterior shoulder tightness: stabilize shoulder blade against wall and gradually pull arm across body.

Figure 13. Phase 1: shoulder mobility and stability. Sleeper stretch for posterior shoulder tightness: adjust elbow down to avoid painful position.

Figure 14. Phase 1: shoulder mobility and stability. Add towel under elbow to create more of a stretch.
Fig 15. Phase 1: shoulder mobility and stability. Doorway stretch for tight anterior musculature.

Fig 16. Phase 1: shoulder mobility and stability. Adjust elbow position based on level of stretch and symptoms; instruct patient to squeeze scapula together and step forward until a stretch is felt across front of shoulder.

Fig 17. Phase 1: scapular stability series. Scapular repositioning to teach neuromuscular control.

Fig 18. Phase 1: scapular stability series. Repetitively retract the scapula from a protracted position.
Fig 19. Phase 1: scapular stability series. Low-row scapular retraction with isometric contraction.

Fig 20. Phase 1: scapular stability series. Retract and depress scapula while pushing down and back on a stable surface with hand.

Fig 21. Phase 1: scapular stability series. Inferior glide with scapular adduction.

Fig 22. Phase 1: scapular stability series. Retract and depress scapula while pushing downward with arm slightly away from body. Criteria to progress to phase 2 are as follows: patient has minimal to no pain with activities of daily life; patient demonstrates good voluntary scapular control during exercises with minimal to no verbal cueing; flexibility is approaching acceptable values for the individual; and patient demonstrates proper performance of hip exercises without losing balance during a complete set of repetitions.

Fig 23. Phase 2: core mobility/stability/balance. Dynamic stretching for hip flexor and rotator tightness.
**Fig 24.** Phase 2: core mobility/stability/balance. Prone hip extension with right foot touching left hip (scorpion), alternating legs.

**Fig 25.** Phase 2: core mobility/stability/balance. Dynamic stretching for hip mobility and balance.

**Fig 26.** Phase 2: core mobility/stability/balance. Backward lunge with trunk rotation to opposite side.

**Fig 27.** Phase 2: core mobility/stability/balance. Hip abductor strengthening.

Fig 29. Phase 2: core mobility/stability/balance. Balance and control—power position; start in stretch position.

Fig 30. Phase 2: core mobility/stability/balance. Lift lead leg to 90°; hold for 3 seconds.

Fig 31. Phase 2: core mobility/stability/balance. Stride forward, keeping toe closed; hold for 3 seconds.
Fig 32. Phase 2: core mobility/stability/balance. Rotate torso forward while pinching shoulder blades together; hold for 3 seconds. Do not internally rotate arm forward.

Fig 33. Phase 2: core mobility/stability/balance. Hip strength and balance.

Fig 34. Phase 2: core mobility/stability/balance. Lateral planks stabilizing on bent or extended elbow or over ball if shoulder pain is present.

Fig 35. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Three level rows for scapular retraction, starting in a protracted scapular position and trunk flexed.

Fig 36. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Move toward trunk extension and scapular retraction with low rows, with elbow straight at side.
Fig 37. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Mid rows with elbow flexed at side.

Fig 38. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). High rows with elbow flexed and arm abducted to 90°.

Fig 39. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Shoulder adduction, starting position.

Fig 40. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Actively adduct for serratus depression.
Fig 41. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). External rotation to strengthen rotator cuff without pain.

Fig 42. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Perform at 0° of abduction.

Fig 43. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Perform at 45° of abduction.

Fig 44. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Perform at 90° of abduction.
Fig 45. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Internal rotation to strengthen rotator cuff without pain.

Fig 46. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Perform at 0° of abduction.

Fig 47. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Perform at 45° of abduction.

Fig 48. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Perform at 90° of abduction.
Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Serratus strengthening.

Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Shoulder flexion with a punch to facilitate scapular protraction to just above shoulder level.

Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Rotator cuff elevation with force couple.

Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Shoulder abduction in scapular plane with weight or band.
Fig 53. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Wrist extension.

Fig 54. Phase 2: shoulder mobility and stability (flexibility exercises are continued from previous phase). Wrist flexion. Criteria to progress to phase 3 are as follows: patient has minimal to no pain with all exercises; patient demonstrates normal motor control without substitution during exercises; and elimination of flexibility deficits in upper and lower extremities during static and dynamic stretching.

Fig 55. Phase 3: core mobility/power. Dynamic stretching for hamstring and hip mobility. Walk on hands out as far as possible.

Fig 56. Phase 3: core mobility/power. Then move feet toward hands to feel a stretch in the hamstring.

Fig 57. Phase 3: core mobility/power. Torso rotation and latissimus stretch.
Fig 58. Phase 3: core mobility/power. While sitting on heels and with lumbar spine flexed, place 1 arm out straight, with other arm behind head, to rotate toward flexed elbow side.

Fig 59. Phase 3: core mobility/power. Single leg squats. Stand on 1 leg.

Fig 60. Phase 3: core mobility/power. Squat down, keeping knee in line with foot and not in front of foot. Go as low as you can without losing your balance. Start by holding onto bat for balance, but work to be able to perform 5 to 10 repetitions without assistance.

Fig 61. Phase 3: core mobility/power. Core stability: chop in half, kneeling diagonal. Focus on keeping core stable.
**Fig 62.** Phase 3: core mobility/power. Core stability: lift in half, kneeling diagonal. Focus on keeping core stable.

**Fig 63.** Phase 3: core mobility/power. Core stability: start position for chop in half, standing diagonal. Focus on keeping core stable.

**Fig 64.** Phase 3: core mobility/power. Core stability: end position for chop in half, standing diagonal. Focus on keeping core stable.

**Fig 65.** Phase 3: core mobility/power. Core stability: start position for lift in half, standing diagonal. Focus on keeping core stable.
Fig 66. Phase 3: core mobility/power. Core stability: end position for lift in half, standing diagonal. Focus on keeping core stable.

Fig 67. Phase 3: shoulder endurance and eccentrics. Shoulder and scapular endurance: prone alphabet on a ball or over a bench. The IWTC acronym (“I would throw you a curve-ball”) can be applied: “I,” shoulder extension with scapular retraction; “W,” scapular retraction with elbows flexed; “T,” prone horizontal abduction with thumbs up; “Y,” scapular retraction with shoulder flexion at 135° with thumbs up; “C,” supine crunch with scapula punch holding a weight and abdominals drawn in.

Fig 68. Phase 3: shoulder endurance and eccentrics. Prone scapular retraction (“W”).

Fig 69. Phase 3: shoulder endurance and eccentrics. Prone horizontal abduction with scapular retraction (“T”).

Fig 70. Phase 3: shoulder endurance and eccentrics. Prone shoulder flexion (“Y”).
Fig 71. Phase 3: shoulder endurance and eccentrics. Supine crunch with scapular protraction.

Fig 72. Phase 3: shoulder endurance and eccentrics. “C” emphasize abdominal hollowing with each crunch.

Fig 73. Functional phase: shoulder endurance and eccentrics. Plyometric overhead toss of 5-lb medicine ball.

Fig 74. Functional phase: shoulder endurance and eccentrics. Toss into rebounder or wall or with teammate with 2 hands.

Fig 76. Functional phase: shoulder endurance and eccentrics. Toss into rebounder or with teammate.

Fig 77. Functional phase: shoulder endurance and eccentrics. Eccentric strengthening: prone 90/90 ball drops.
Fig 78. Functional phase: shoulder endurance and eccentrics. Move arm from perpendicular to parallel position (with respect to floor).

Fig 79. Functional phase: shoulder endurance and eccentrics. Eccentric strengthening. In half kneeling position, start in arm “power position.”

Fig 80. Functional phase: shoulder endurance and eccentrics. Lower 1- to 2-lb weight into follow-through position slowly over an 8 count.
Table 2. MRI Findings of Asymptomatic Throwers

<table>
<thead>
<tr>
<th>Citation</th>
<th>Population</th>
<th>Supraspinatus Signal/Partial Tears</th>
<th>Full Tears</th>
<th>Labral Abnormality</th>
<th>Abnormal Fluid</th>
<th>AC Arthritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Connor et al., 126 2003</td>
<td>40 throwing athletes Age, 26.5 yr</td>
<td>40% of dominant arms had PTRCTs or full-thickness rotator cuff tears</td>
<td>7.5%</td>
<td>90%</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>Halbrecht et al., 127 1999</td>
<td>10 asymptomatic collegiate baseball players</td>
<td>40% had abnormal signal, 20% had tendinosis, 20% had PTRCTs</td>
<td>0%</td>
<td>30% had posterior SLAP</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Jerosch et al., 128 1993</td>
<td>11 elite water polo players Age, 25.4 yr</td>
<td>45%</td>
<td>0%</td>
<td>100% had anterior labrum pathology</td>
<td>64%</td>
<td>NR</td>
</tr>
<tr>
<td>Jost et al., 124 2005</td>
<td>30 throwing athletes (both asymptomatic athletes and athletes with symptoms but no limitations in throwing) Age, 25.4 yr</td>
<td>43% had supraspinatus partial tears, 27% had infraspinatus partial tears, 15% had subscapularis partial tears</td>
<td>0%</td>
<td>40% had anterior labrum pathology, 30% had posterior labrum pathology, 37% had posterosuperior glenoid impingement</td>
<td>NR</td>
<td>40%</td>
</tr>
<tr>
<td>Miniaci et al., 129 2002</td>
<td>28 professional pitchers Age, 20.1 yr</td>
<td>86% had abnormalities with 7% infraspinatus partial tears</td>
<td>0%</td>
<td>79% had signal changes, 45% classified as having tears</td>
<td>79%</td>
<td>36%</td>
</tr>
</tbody>
</table>

NOTE. The data suggest that in asymptomatic throwers, the findings that might be considered abnormal may be present in the absence of symptoms. AC, acromioclavicular; NR, not reported; PTRCT, partial-thickness rotator cuff tear.

Table 3. Outcome After Surgery for Superior Labral Tears in Throwers

<table>
<thead>
<tr>
<th>Citation</th>
<th>LOE</th>
<th>No. of Patients and Type of Population</th>
<th>Technique</th>
<th>Other Pathology</th>
<th>Follow-up</th>
<th>Return to Same or Better Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brockmeier et al., 132 2009</td>
<td>IV</td>
<td>47 with 34 athletes (28 overhead athletes including 16 baseball or softball players)</td>
<td>Suture anchor</td>
<td>24/47 PTRCT debrided, 33/47 subacromial bursectomy, 4/47 DCE</td>
<td>2.7 yr</td>
<td>71% of all athletes returned to preinjury level; RTP occurred in 11/12 with acute injury (92%) and 14/22 with chronic injury (64%)</td>
</tr>
<tr>
<td>Cerynik et al., 133 2008</td>
<td>IV</td>
<td>42 MLB pitchers with isolated glenoid labral injuries Age, 28.4 yr</td>
<td>“Surgical repair of torn labrum”</td>
<td>NR</td>
<td>3 yr</td>
<td>69% overall RTP rate (73% of starters and 63% of relievers); ERA and IP showed decline after surgery</td>
</tr>
<tr>
<td>Cohen et al., 134 2006</td>
<td>IV</td>
<td>39 (29 athletes with 8 throwers)</td>
<td>Bioabsorbable tack</td>
<td>NR</td>
<td>44 mo</td>
<td>45% for all athletes</td>
</tr>
<tr>
<td>Cohen et al., 135 2011</td>
<td>IV</td>
<td>23 patients with labral tears (all baseball players)</td>
<td>Suture anchor repairs</td>
<td>None</td>
<td>&gt;24 mo</td>
<td>32% returned to same level after labral repair; RTP rate of 17% for double-A ball or higher</td>
</tr>
</tbody>
</table>

(continued)
<table>
<thead>
<tr>
<th>Citation</th>
<th>LOE</th>
<th>No. of Patients and Type of Population</th>
<th>Technique</th>
<th>Other Pathology</th>
<th>Follow-up</th>
<th>Return to Same or Better Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cordasco et al.,136 1993</td>
<td>IV</td>
<td>52 patients with labral tears; 27 had SLAP lesions, with 17 type II (9 throwers, 4 tennis players, 4 swimmers) Age, 29 yr</td>
<td>Debridement</td>
<td>25/52 patients (48%) had subacromial bursectomy</td>
<td>36 mo</td>
<td>63% RTP rate</td>
</tr>
<tr>
<td>Field and Savoie,137 1993</td>
<td>IV</td>
<td>20 patients (6 throwers) Caspari transglenoid suture repair</td>
<td>Debridement of PTRCT in 8/20, 5 had acromioplasty, 3 had DCE</td>
<td>21 mo</td>
<td>All 6 throwers returned without limitations or loss of velocity</td>
<td></td>
</tr>
<tr>
<td>Friel et al.,138 2010</td>
<td>IV</td>
<td>48 patients with type II SLAP lesions; 13 collegiate overhead athletes Age, 33.1 yr</td>
<td>Bioabsorbable suture anchor repair</td>
<td>3.4 yr</td>
<td>7/13 collegiate overhead athletes (54%) returned to previous level</td>
<td></td>
</tr>
<tr>
<td>Glasgow et al.,139 1992</td>
<td>IV</td>
<td>8 overhead or throwing athletes Debridement</td>
<td>1 had instability</td>
<td>&gt;24 mo</td>
<td>88% (1 failure in patient with instability)</td>
<td></td>
</tr>
<tr>
<td>Ide et al.,233 2005</td>
<td>IV</td>
<td>40 patients (all overhead athletes) Suture anchor</td>
<td>30/40 had PTRCT debrided</td>
<td>41 mo</td>
<td>60% for baseball players with overuse SLAP lesions</td>
<td></td>
</tr>
<tr>
<td>Kim et al.,141 2002</td>
<td>IV</td>
<td>34 patients with isolated superior labral tears (18 throwing athletes)</td>
<td>Suture anchor</td>
<td>None</td>
<td>33 ± 9 mo</td>
<td>4/18 throwers (22%) returned to previous level</td>
</tr>
<tr>
<td>Lintner, 2011*</td>
<td>IV</td>
<td>36 SLAP repairs in professional throwers in 1 MLB organization</td>
<td>Suture anchor</td>
<td>11 had RCT repair, 6 had RCT debridement, 4 had anterior labral repair, 2 had exostosis excision, 1 had thermal capsulorrhaphy, 1 had capsule plication, 1 had distal clavicle excision, 1 had biceps tenodesis</td>
<td>34 mo</td>
<td>64% returned to prior level</td>
</tr>
<tr>
<td>Morgan et al.,116 1998</td>
<td>IV</td>
<td>53 patients, all overhead athletes including 44 pitchers</td>
<td>Suture anchor</td>
<td>31% of all SLAP repairs had PTRCT or FTRCT</td>
<td>12 mo</td>
<td>RTP rate of 84% for pitchers; all failures had PTRCT</td>
</tr>
<tr>
<td>Neri et al.,133 2011</td>
<td>IV</td>
<td>23 patients (20 baseball players, 1 volleyball player, 1 tennis player, 1 water polo player)</td>
<td>Suture anchor</td>
<td>8/23 had PTRCT</td>
<td>38 mo</td>
<td>57% returned at previous level; 80% without partial-thickness cuff tear and 12.5% with PTRCT</td>
</tr>
<tr>
<td>Neuman et al.,142 2011</td>
<td>IV</td>
<td>30 patients, all overhead athletes including 21 baseball or softball players with 14 pitchers Age, 24 yr</td>
<td>Suture anchor</td>
<td>NR</td>
<td>3.5 yr</td>
<td>“Perception of normal,” 79.5%; RTP rate of 84.1% of athletes</td>
</tr>
</tbody>
</table>

(continued)
<table>
<thead>
<tr>
<th>Citation</th>
<th>LOE</th>
<th>No. of Patients and Type of Population</th>
<th>Technique</th>
<th>Other Pathology</th>
<th>Follow-up</th>
<th>Return to Same or Better Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pagnani et al., 143 1995</td>
<td>IV</td>
<td>22 patients with 16 type II and 6 type IV (13 overhead athletes)</td>
<td>Bioabsorbable tack</td>
<td>2 had Bankart repairs, 4 had SAD, 1 had mini-open FTRCT repair</td>
<td>&gt;24 mo</td>
<td>92% in overhead athletes</td>
</tr>
<tr>
<td>Paletta, 144 2010</td>
<td>IV</td>
<td>67 throwing athletes with 54 type II, 10 type III, and 3 type IV SLAP lesions; 60 of 67 were pitchers</td>
<td>Bioabsorbable tacks in 15, knotless anchors in 52</td>
<td>40/67 had PTRCT, 4 had internal impingement, 3 had subacromial bursitis, 2 had paralabral cysts</td>
<td>&gt;24 mo</td>
<td>86.5% return to throwing in first season, 78% returned at same or higher level</td>
</tr>
<tr>
<td>Rhee et al., 118 2005</td>
<td>IV</td>
<td>44 patients with 31 type II, 9 type III, and 4 type IV lesions (33 athletes including 15 throwers)</td>
<td>Bioabsorbable tacks in 14, suture anchors in 30</td>
<td>None</td>
<td>33 mo</td>
<td>50% of throwers returned to same level of play</td>
</tr>
<tr>
<td>Ricchetti et al., 145 2010</td>
<td>III</td>
<td>51 MLB pitchers Age, 28.1 yr</td>
<td>“Surgery to repair torn glenoid labrum”</td>
<td>NR</td>
<td>13.1 mo</td>
<td>72.5% returned to Major League play</td>
</tr>
<tr>
<td>Sanders, 146 2008</td>
<td>IV</td>
<td>67 patients, with 19 professional, 32 collegiate, and 16 high school baseball players; 60/67 were pitchers; 54 type II, 3 type IV Age, 22.5 yr</td>
<td>Type II treated with bioabsorbable suture anchors</td>
<td>47/67 (70%) had associated pathology, most commonly PTRCT</td>
<td>30 mo</td>
<td>83.5% returned to same level of throwing</td>
</tr>
<tr>
<td>Yoneda et al., 147 1991</td>
<td>IV</td>
<td>10 athletes (7 baseball players) Age, 17.8 years</td>
<td>Staple</td>
<td>3 (30%) had PTRCT, 2 (20%) had subacromial bursitis</td>
<td>37.4 mo</td>
<td>2/10 (20%) had normal function</td>
</tr>
<tr>
<td>Yung et al., 148 2008</td>
<td>IV</td>
<td>16 patients (13 overhead athletes)</td>
<td>Suture anchor</td>
<td>None</td>
<td>27.6 mo</td>
<td>94%</td>
</tr>
</tbody>
</table>

NOTE. High variation (20% to 94%) is noted in the rates of return to play. Only 4 of 21 series (19%) had return-to-play rates at or above 85%. The presence of rotator cuff disease affects outcomes adversely.

DCE, distal clavicle excision; ERA, earned run average; FTRCT, full-thickness rotator cuff tear; IP, innings pitched; LOE, level of evidence; MLB, Major League Baseball; NR, not reported; PTRCT, partial-thickness rotator cuff tear; RCT, rotator cuff tear; RTP, return to play; SAD, subacromial decompression.

*Information presented at the consensus meeting.
Table 4. Outcome After Surgery for Partial-Thickness Rotator Cuff Tears in Throwing Athletes

<table>
<thead>
<tr>
<th>Citation</th>
<th>LOE</th>
<th>No. of Patients and Type of Population</th>
<th>Technique</th>
<th>Other Pathology</th>
<th>Follow-up</th>
<th>Return to Same or Better Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Andrews et al., 234 1985</td>
<td>IV</td>
<td>34 athletes (23 pitchers) Age, 22 yr</td>
<td>Debride PTRCT</td>
<td>All had tearing of some part of labrum, 3 with tendonitis of biceps, 6 with partial tear of biceps</td>
<td>13.1 mo</td>
<td>76% returned to play at same level</td>
</tr>
<tr>
<td>Conway, 152 2001</td>
<td>IV</td>
<td>14 professional baseball players (13 pitchers)</td>
<td>Repair delaminated tendon, not to bone</td>
<td>100% had superior labral tears: 29% type I, 29% type II, and 7% type III; 50% had anterior instability; 57% had subacromial bursitis</td>
<td>16 mo</td>
<td>8/9 players with &gt;12 months follow-up (89%) returned</td>
</tr>
<tr>
<td>Ide et al., 140 2005</td>
<td>IV</td>
<td>17 (6 throwing athletes) Age, 42 yr</td>
<td>Repair PTRCT to bone</td>
<td>None</td>
<td>39 mo</td>
<td>2/6 returned to same level, 3/6 returned at lower level, 1/6 did not return</td>
</tr>
<tr>
<td>Payne et al., 153 1997</td>
<td>IV</td>
<td>29 overhead athletes Age, &lt;40 yr</td>
<td>Debride PTRCT</td>
<td>17 had labral tears: 11 posterior, 3 anterior, and 3 SLAP lesions</td>
<td>24 mo</td>
<td>13/29 (45%) returned to sports</td>
</tr>
<tr>
<td>Reynolds et al., 154 2008</td>
<td>IV</td>
<td>67 professional pitchers Age, 25.6 yr</td>
<td>Debride PTRCT</td>
<td>Only 4% had isolated partial cuff tears; labral debridement in 26%, labral repair in 60%, thermal shrinkage in 33%, capsule release in 6%</td>
<td>39.2 mo</td>
<td>37/67 (55%) returned at same or higher level</td>
</tr>
<tr>
<td>Rian et al., 155 2002</td>
<td>IV</td>
<td>67 throwing athletes</td>
<td>Debride PTRCT</td>
<td>90% of athletes had labral lesions; most were posterior labrum</td>
<td>22.3 mo</td>
<td>12/67 (16%) returned to sports at former level</td>
</tr>
</tbody>
</table>

NOTE. Most patients had additional pathology that can affect outcomes. The best results use a nonanatomic transtendon repair of the delaminated tendon without repair to bone. LOE, level of evidence; PTRCT, partial-thickness rotator cuff tear.
<table>
<thead>
<tr>
<th>Citation</th>
<th>LOE</th>
<th>No. of Patients and Type of Population</th>
<th>Technique</th>
<th>Other Pathology</th>
<th>Follow-up</th>
<th>Return to Same or Better Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liem et al.,156 2008</td>
<td>III</td>
<td>21 amateur throwing athletes Age, 58.9 yr</td>
<td>FTRCT repair to bone</td>
<td>DCE in 4/21, biceps tenotomy in 4/21</td>
<td>25.7 mo</td>
<td>Preinjury and postoperative participation levels no different; retear rate was 23.8% but did not influence return to play</td>
</tr>
<tr>
<td>Mazoue and Andrews,157 2006</td>
<td>IV</td>
<td>16 professional baseball players with 12 pitchers</td>
<td>Mini-open repair of FTRCT to bone</td>
<td>2 had SLAP repairs plus thermal capsulorrhaphy, 2 had labral debridement plus thermal capsulorrhaphy, 2 had thermal capsulorrhaphy, 4 had labral debridement</td>
<td>66.6 mo</td>
<td>1/12 (8%) of pitchers returned; 1/2 position players with dominant arm involved (50%) returned</td>
</tr>
</tbody>
</table>

NOTE. Return-to-play rates of repair of full-thickness rotator cuff tears to bone are exceptionally poor.

DCE, distal clavicle excision; FTRCT, full-thickness rotator cuff tear; LOE, level of evidence.
<table>
<thead>
<tr>
<th>Professional baseball pitchers (n = 40) and position players (n = 40)</th>
<th>Pitchers</th>
<th>Position Players</th>
</tr>
</thead>
<tbody>
<tr>
<td>Throwing Arm</td>
<td>Non-Throwing Arm</td>
<td>Throwing Arm</td>
</tr>
<tr>
<td>Internal rotation sitting (°)</td>
<td>35 ± 6</td>
<td>34 ± 6</td>
</tr>
<tr>
<td>External rotation sitting (°)</td>
<td>41 ± 6</td>
<td>41 ± 8</td>
</tr>
<tr>
<td>Professional baseball pitchers (n = 101)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Throwing Arm</td>
<td>Non-Throwing Arm</td>
<td>Throwing Arm</td>
</tr>
<tr>
<td>Internal rotation prone (°)</td>
<td>23 ± 8</td>
<td>22 ± 9</td>
</tr>
<tr>
<td>External rotation prone (°)</td>
<td>35 ± 9</td>
<td>34 ± 11</td>
</tr>
<tr>
<td>Collegiate baseball pitchers (n = 16)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Throwing Arm</td>
<td>Non-Throwing Arm</td>
<td>Throwing Arm</td>
</tr>
<tr>
<td>Internal rotation sitting (°)</td>
<td>38 ± 7</td>
<td>34 ± 7</td>
</tr>
<tr>
<td>External rotation sitting (°)</td>
<td>30 ± 5</td>
<td>30 ± 4</td>
</tr>
<tr>
<td>Hip extension prone (°)</td>
<td>22 ± 5</td>
<td>20 ± 4</td>
</tr>
<tr>
<td>Hip flexion supine (°)</td>
<td>119 ± 9</td>
<td>123 ± 11</td>
</tr>
<tr>
<td>Professional baseball pitchers (n = 19)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Throwing Arm</td>
<td>Non-Throwing Arm</td>
<td>Throwing Arm</td>
</tr>
<tr>
<td>External rotation prone (°)</td>
<td>44 ± 9</td>
<td>36 ± 6</td>
</tr>
<tr>
<td>Internal rotation prone (°)</td>
<td>50 ± 9</td>
<td>31 ± 6</td>
</tr>
<tr>
<td>Adduction supine (°)</td>
<td>51 ± 8</td>
<td>32 ± 6</td>
</tr>
<tr>
<td>Abduction supine (°)</td>
<td>43 ± 12</td>
<td>36 ± 11</td>
</tr>
</tbody>
</table>
Table 7. Shoulder ROM Descriptive Data (Mean ± Standard Deviation) for Softball and Baseball Athletes

<table>
<thead>
<tr>
<th></th>
<th>Baseball Pitchers</th>
<th>Baseball Position Players</th>
<th>Softball Players</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Throwing Arm</td>
<td>Non-Throwing Arm</td>
<td>Throwing Arm</td>
</tr>
<tr>
<td>High school softball players (n = 103) and baseball players (n = 143)</td>
<td>53° ± 11°</td>
<td>61° ± 9°</td>
<td>60° ± 14°</td>
</tr>
<tr>
<td>Passive internal rotation in supine with scapula stabilized at 90°</td>
<td>126° ± 11°</td>
<td>118° ± 12°</td>
<td>124° ± 14°</td>
</tr>
<tr>
<td>Passive external rotation in supine with scapula stabilized</td>
<td>30° ± 11°</td>
<td>33° ± 12°</td>
<td>34° ± 13°</td>
</tr>
<tr>
<td>Passive horizontal adduction in supine with scapula stabilized</td>
<td>12° ± 11°</td>
<td>11° ± 12°</td>
<td>14° ± 13°</td>
</tr>
<tr>
<td>Collegiate baseball players (n = 33)</td>
<td>56° ± 7°</td>
<td>69° ± 5°</td>
<td>58° ± 7°</td>
</tr>
<tr>
<td>Passive internal rotation in supine with scapula stabilized at 90°</td>
<td>62° ± 4°</td>
<td>72° ± 3°</td>
<td></td>
</tr>
<tr>
<td>Passive external rotation in supine with scapula stabilized at 90°</td>
<td>132° ± 12°</td>
<td>117° ± 11°</td>
<td>122° ± 11°</td>
</tr>
<tr>
<td>Passive horizontal adduction in supine with scapula stabilized*</td>
<td>127° ± 11°</td>
<td>114° ± 11°</td>
<td></td>
</tr>
<tr>
<td>Collegiate baseball pitchers (n = 15) and players (n = 23)</td>
<td>48° ± 11°</td>
<td>59° ± 11°</td>
<td></td>
</tr>
<tr>
<td>Passive internal rotation in supine with scapula stabilized at 90°</td>
<td>48° ± 11°</td>
<td>59° ± 11°</td>
<td></td>
</tr>
<tr>
<td>Passive external rotation in supine with scapula stabilized at 90°</td>
<td>136° ± 11°</td>
<td>128° ± 11°</td>
<td></td>
</tr>
</tbody>
</table>

*Passive horizontal adduction with a 0° reference angle occurs when the humerus is perpendicular to the table; a negative value indicates that horizontal adduction did not reach the 0° reference angle position.