PARTIAL ROTATOR CUFF TEARS

W. BEN KIBLER MD

MEDICAL DIRECTOR
LEXINGTON SPORTS MEDICINE CENTER
LEXINGTON, KY

Articular sided partial rotator cuff tears have been identified very commonly in throwing athletes, most frequently in association with other shoulder injuries, especially superior glenoid labral tears (1). It was originally thought that the rotator cuff lesions were caused by “internal impingement” of the articular surface of the rotator cuff against the superior glenoid (2). However, just as with “external” impingement, “internal” impingement is now being regarded as a symptom, secondary to altered mechanics in the scapuloglenohumeral articulation (1),(3).

One proposed causative mechanism of the altered glenohumeral mechanics is acquired anterior micro-instability due to stretching of the anterior capsule (3). This capsular stretch would allow the glenohumeral articulation to move into a “hyperangulation” position of increased external rotation and horizontal abduction and create the rotator cuff lesion due to mechanical impingement. However, there is no hard scientific evidence that this occurs, and treatments based on this rationale have not been uniformly successful.

A second proposed causative mechanism of the altered glenohumeral mechanics is acquired glenohumeral internal rotation deficit (GIRD). This deficit is created by progressive contracture of the posterior glenohumeral capsule and decreased static and dynamic flexibility of the posterior shoulder muscles (1),(4). This tight capsule creates a superior shift in the glenohumeral contact point- posterosuperiorly in cocking (1) and anterosuperiorly in followthrough (5). This initiates a “pathologic cascade” (6) that climaxes in the cocking phase of throwing. As the shoulder abducts and excessively externally rotates around this new contact point, shear forces at the biceps anchor and poaterosuperior labrum increase through a peel back action of the biceps on the labrum, producing a posterior SLAP lesion; the anterior capsule becomes lax due to the altered contact point, and may secondarily stretch due to the hyper external rotation; and there are increased shear and torsional forces on the rotator cuff as the other joint constraints fail, creating a “hypertwist” load mechanism of injury rather than a straight tensile load mechanism. All of these consequences are worsened by scapular dyskinesis, producing a protracted scapula that creates an antetilted glenoid, increases anterior capsular tensile loads, and magnifies the peel back action (1). About half of these patients will also demonstrate hip and trunk weakness on examination.

Partial thickness rotator cuff tears in throwers have also been shown to be “lesion specific”. The anterior PRCT were associated with anterior SLAP lesions, and posterior PRCT were associated with posterior SLAP lesions. This adds credence to the idea that the hypertwist of the rotator cuff in the areas of superior subluxation due to labral injury, either anterior or posterior, may contribute to tearing of the cuff. In this situation, the mechanically based tensile/shearing/torsional load may create enough internal strain to initiate apoptotic changes in the cell, adding degenerative damage to mechanical damage.

Clinical Implications

Throwing athletes who develop signs and symptoms of PRCT, or who are found to have these lesions in surgery should have a comprehensive clinical examination to evaluate the associated alterations. This should include a screening exam for hip and trunk strength and flexibility, evaluation of scapular position and dynamic motion, a scapula stabilized determination of glenohumeral internal rotation, and testing for superior labral tears(7). Optimal treatment of the rotator cuff injury is based on the size of the lesion and its thickness. Lesions that involve more than half of the thickness should probably be taken done and repaired like full thickness tears. Surgical treatment should also be directed at the associated pathology, especially the labral injuries. Post operative rehabilitation should include all areas of the kinetic chain, including the legs, trunk scapula, and shoulder. Rehabilitation of the trunk and scapula can begin while the arm is in the sling.

REFERENCES

1. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: Spectrum of pathology part I: Pathoanatomy and biomechanics. **Arthroscopy** 2003; 19: 404- 420.
2. Walch G, Boileau J, Noel E et al. Impingement of the deep surface of the supraspinatus tendon on the posterior superior glenoid rim: An arthroscopic study. **J Shoulder Elb Surg** 1992; 1: 238- 243.
3. Jobe CM. Posterior superior glenoid impingement: Expanded spectrum. **Arthroscopy** 1995; 11: 530- 537.
4. Kibler WB, Chandler TJ, Livingston BP et al. Shoulder rotation in elite tennis players- effect of age and tournament play. **Am J Sports Med** 1996; 24: 279- 285.
5. Harryman DT, Sidles JA, Clark JM. Translation of the humeral head on the glenoid with passive glenohumeral motion. **J Bone Joint Surg** 1990; 72: 1334- 1343.
6. Leadbetter WB. Cell matrix response in tendon injury, **Clin Sports Med** 1992; 11: 533- 578.
7. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: Spectrum of pathology part III: The SICK scapula, scapular dyskinesis, the kinetic chain, and rehabilitation. **Arthroscopy** 2003; 19: 641- 661.