Dynamic Chiropractic

Shoulder Impingement Syndrome in the Athlete

About the author: Dr. Souza is department chairman of diagnosis at Palmer College of Chiropractic West, and director of the college's Tasman Clinic. He is a diplomate of the American Chiropractic Board of Sports Physicians, and author/editor of Sports Injuries of the Shoulder.

Shoulder impingement, although a common diagnosis, is still poorly defined. What actually causes the impingement? Well-recognized is the impingement anteriorly that may occur under the coracoacromial (subacromial) arch. This arch or outlet is formed by the coracoid, acromion, and coracoacromial ligament superiorly; the head of the humerus inferiorly. The structures most often affected with this type of impingement are the occupants of the outlet, consisting of the underlying bursa (subacromial), the supraspinatus and the biceps tendon (less often the infraspinatus or teres minor). This space may be decreased by mechanical impingement from the acromion, by soft tissue hypertrophy or inflammation, elevation of the arm to 90 degrees with internal rotation, and weakness of the humeral head depressors (compressors).

More recently a posterior impingement has been described in throwing athletes where the rotator cuff is pinched between the posterosuperior glenoid labrum and the humeral head,[1,2] mainly involving the supraspinatus and infraspinatus. It is directly related to functional instability which allows excessive movement superiorly with arm elevation. Impingement of the subscapularis and subscapular bursa at the coracoid process is also a diagnostic consideration with anterior shoulder pain.[3]

Requirements for smooth stable shoulder movement with adequate tendon/bursal space are:

- a clear subacromial outlet
- proper synchrony and balance of muscles about the shoulder
- a coupling between the humerus and scapula
- an intact capsule and labrum
- accessory movement or joint play

Although past theories of shoulder impingement have focused on mechanical (surgical) components, the predominance of current literature suggests functional imbalance as the more common factor. In the past, acromial shape was the focus. Either an abnormally "hooked" acromion or osteophyte formation were considered to be mechanical irritants of the subacromial soft tissue: especially with elevation of the arm. Acromial involvement is rare in the young athlete: senior athletes are more often affected. The only exception is persistence of a growth center referred to

as an os acromiale in the younger athlete, usually before age 23.[4]

Evidence for an abnormally shaped acromium is best seen radiographically on an outlet view. This is essentially a lateral scapula view, however the central beam is at the glenohumeral joint with an angulation of about 10-15 degrees caudad.[5] Also subacromial osteophytes may be seen on a Zanca view. This is a unilateral AC view with a 10-15 degree cephalad tube tilt.[5]

Although the scapulohumeral ratio with shoulder elevation has been reported as a 2:1 ratio (gelnohumeral/scapula), this represents an average over the entire range of abduction. During the first 60 degrees, the scapula moves very little unless resistance is added. Doddy et al.,6 found the ratio was closer to 7:1 through the first 30 degrees. They found the ratio from 90 to 150 degrees was close to 1:1. Finally, at end-range abduction, the scapula contributes little to the last 30 degrees of elevation. The scapular movement that does occur is integral to proper positioning of the humeral head for optimum muscle/tendon length, angle, and positioning of the bony glenoid platform under the humeral head. This balancing act at the higher ranges of elevation requires contributions from the rotator cuff and serratus anterior. Therefore, one major component for proper shoulder elevation is scapular positioning and rotator cuff stabilization. This is a synchronous act with all of the rotator cuff participating. Athletes who focus on development of the larger muscles cause an imbalance due to neglect of the crucial (yet less visible) rotator cuff.

Capsular integrity and flexibility may be factors worth investigating. Although instability is often medically evaluated, restrictions to accessory movement are not usually evaluated. Ironically, accessory movement can often be evaluated with the same maneuver/position as instability testing: one finding is restriction, the other looseness.

Until recently, accessory movement at the shoulder was not considered an important factor with impingement. However, since the focus in research has switched to function, instability has been more often accused of causing impingement (secondary impingement). In the younger athlete, this is the most common cause. Although gross instability is an obvious factor, more subtle instability may also play a role although more indirect. This instability may be functional, resulting from the biomechanical demands of the sport or a congenitally loose capsule. Stretching of the anterior support structures, the capsule and the muscles, is found with many sports requiring overhead positions. Testing for instability can be accomplished simply by performing a load and shift or drawer maneuver.[5] Combining the apprehension test with the relocation test, the examiner may link underlying instability to a patient's clinical presentation of impingement. General tests for impingement include the painful arc (pain during the mid-range of abduction), Hawkin's test (pain with passive internal rotation of the arm at 90 degrees flexion), and the Neer test (pain at passive flexion end-range).[5]

Through cadaver studies, Harryman et al.,[7] found that shoulder joints demonstrating instability or other pathology often demonstrated no translation or excessive translation in the direction of capsular laxity. For example, subjects with anterior instability demonstrated anterior migration of a few millimeters when the arm was placed in the cocking position of abduction, external rotation, and horizontal abduction.

Until recently, accessory movement at the shoulder was not considered an important factor with impingement. New evidence suggests otherwise. One of the criteria for determining the need to adjust or mobilize a joint has been the direction that the joint normally translates during a specific active movement. It has been generally accepted that when the shoulder is abducted that the humerus translates downward; that when the arm is abducted and brought back into extension

(similar to the apprehension or cocking position) the humerus translates forward. Relatively new evidence by Harryman et al., and Howell et al.,[8] suggest otherwise. Through radiographic examination coupled with positioning Howell et al., have demonstrated humeral translation in the same direction of movement. Harryman et al., have found similar findings using cadavers. Specifically, when the arm of a supine subject was brought back into a cocking position of 90 degrees abduction and horizontal extension, the humerus translated posteriorly an average of four millimeters. In an attempt to re-center the humeral head, muscle contraction was added. All attempts at muscular re-setting of the humeral head were unsuccessful.

Furthermore, Harryman et al., found that by tightening the posterior capsule, translation occurred opposite the normal direction, often excessively. For example, when the arm was placed in the previous cocking position (abduction, external rotation,, horizontal extension), instead of translating posteriorly (via tension in the anterior capsule), the humeral head translated anteriorly and superiorly. They also found that muscular stimulation failed to correct this abnormal translation. This implies that capsular tension may be a major determinant of accessory movement at the glenohumeral joint. When normal tension exists, movement is on the opposite side of tension. However, when tension is excessive due to capsular adhesions, for example, movement may actually not occur due to competition with the opposite side of the capsule.

How does this affect motion palpation (accessory movement) testing of the shoulder and subsequent correction with regards to impingement? It would appear as though restrictions to end range joint play are still the abnormal finding used to determine need and direction of correction. But the restriction, besides possibly causing pain due to lack of movement, may also allow excessive translation opposite the location of restriction. For example, a restriction to posterior glide might indicate adhesion or fixation posteriorly, but the patient presentation may be anterior shoulder pain resulting from excessive anterior and superior translation. Similarly, as the arm is raised, tension develops in the inferior capsule. Adhesions may then cause superior migration of the humeral head with abduction. Therefore, the two restricted accessory movement patterns which may cause or aggravate impingement are posterior glide and inferior glide.

As an adjunct to adjusting the joint, capsular-specific stretching should be included. For example, if a restriction to posterior glide is felt, in addition to adjusting in the direction of fixation, posterior capsular stretching should be performed in the office and given as a daily home routine. This may be accomplished by using the fulcrum of a pillow placed in front of the chest while the well-arm hand stretches the involved arm across the chest over the pillow. An alternative stretch position places the patient in the prone position with their upper body weight support on their forearms, elbows at right angles to the table. The stretch of the posterior capsule would involve having the patient press their chest down towards the table.

Finally, it is important to guarantee proper length relationships and a balanced contribution by the shoulder musculature. Myofascial release techniques are often helpful. The technique as described by Leahy is especially useful for involvement of the subscapularis and infraspinatus/teres minor.[9]

An integrated approach is necessary to address all of the possible contributors to impingement. It is necessary to continually integrate basic science observations into a model that explains our empirical observations. This may often result in strong objection until one realizes that the argument is simply over theory and that the patient is not treated differently, but with a different paradigm.

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Thomas Souza, DC, DACBSP
San Jose, California

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