Quantification of Posterior Capsule Tightness and Motion Loss in Patients with Shoulder Impingement

Timothy F. Tyler,*† MS, PT, ATC, Stephen J. Nicholas,*‡§ MD, Timothy Roy,‖ PT, and Gilbert W. Gleim,* PhD

*From the *Nicholas Institute of Sports Medicine and Athletic Trauma at Lenox Hill Hospital, New York, ‡Hofstra University, Hempstead, §New York Jets Football Club, and ‖Eastside Sports Physical Therapy, New York, New York*

ABSTRACT

The relationship between posterior capsule tightness and dysfunction has long been recognized clinically but has not been biometrically quantified. The purpose of this study was to quantify changes in range of motion and posterior capsule tightness in patients with dominant or nondominant shoulder impingement. Measurements of posterior capsule tightness and external and internal rotation range of motion were made in 31 patients with shoulder impingement and in 33 controls without shoulder abnormality. Patients with impingement in the nondominant arm had increased posterior capsule tightness and decreased internal and external rotation range of motion compared with controls. Patients with impingement in their dominant arm had increased posterior capsule tightness and reduced internal rotation range of motion but no significant loss of external rotation range of motion compared with controls. Posterior capsule tightness in impingement patients showed a significant correlation with loss of internal rotation range of motion. Patients with shoulder impingement in their nondominant arm had a more global loss of range of motion compared with patients having impingement in their dominant arm. We believe we have described a valid clinical measurement for identifying posterior capsule tightness in patients with shoulder impingement.

† Address correspondence and reprint requests to Timothy F. Tyler, MS, PT, ATC, NISMAT at Lenox Hill Hospital, 130 East 77th Street, New York, NY 10021.
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The shoulder plays a vital role in many athletic activities. Overhead motions such as throwing, swimming, and serving in tennis repetitively place the shoulder in vulnerable positions possibly leading to impingement syndrome. This syndrome, also called "subacromial impingement," has been classified as primary or secondary. Primary impingement refers to mechanical encroachment into the subacromial space by the humeral head, often seen in middle-aged patients.[2] Different acromial types may contribute to the encroachment as a result of the development of spurs.[3,4,16,31] Secondary impingement syndrome symptoms are thought to be a result of shoulder instability, scapulothoracic weakness, and posterior capsule tightness, which may contribute to subtle anterior instability.[1–4,14,16,25,26,30]

Although the factors contributing to secondary shoulder impingement are multiple, the manifestation of posterior capsule tightness is thought to contribute to altered arthrokinematics. The effect of tight capsular and muscular structures of the shoulder on normal shoulder range of motion has been well documented.[11,19,20,23,27,30] Clinically, much attention has been given to how a tight posterior capsule might affect normal glenohumeral arthrokinematics. The posterior capsular structures have been shown to play a significant role in allowing and controlling normal arthrokinematics between the humeral head and the glenoid.[9,18,20,22,27,30] Harryman et al.[9] state that oblique glenohumeral translations are not the result of ligament insufficiency or laxity; instead, translation results when the capsule is asymmetrically tight. Asymmetrical tightness is thought to cause anterior and superior migration of the humeral head during forward elevation of the shoulder, possibly contributing to impingement.

The purpose of this study was to document changes in range of motion and posterior capsule tightness in patients with dominant or nondominant shoulder impingement.

MATERIALS AND METHODS

Subjects

All patients agreed to have measurements made on them as part of a routine clinical examination. The controls consisted of 33 volunteer subjects (20 men, 13 women) with no history of shoulder injury or disease in their lifetime. No subject had any history of shoulder surgery. The subjects were between 21 and 57 years old (33 ± 9.3 years, mean $\pm$ SD). The average height was 165 ± 10 cm; the average weight was 68 ± 15 kg.

Secondary subacromial impingement syndrome was diagnosed in 31 patients. The diagnosis was made on the basis of the patients' history and a clinical examination including full passive forward flexion and a positive Neer impingement sign. Patients who had concomitant adhesive capsulitis, cervical radiculopathy, or evidence of bone spurs on radiographs were excluded, as were patients who had weakness on testing of the rotator cuff or a full-thickness rotator cuff tear on MRI.[18] All patients had radiographs but not an MRI scan. Patients were between 19 and 74 years old (44 ± 16.5 years). The average height was 160 ± 9 cm; the average weight was 50 ± 14 kg.

Equipment

All measurements were made on a standard examination table. A standard carpenter's square was used for marking the location of the medial epicondyle in relation to the surface of the examination table. The 90° angle of the square ensured that a perpendicular line from the examination table to the medial epicondyle was measured. Standard goniometers were used to measure internal and external rotation.

Measurement of Posterior Capsule Tightness

We have previously described the measurement of posterior capsule tightness.[28] Proper positioning of the subject was crucial to a reliable measurement. Any rotation of the torso forward or backward would result in a corresponding increase or decrease in the measurement.

Figure 1 shows the scapula being grasped at the lateral border and stabilized in the retracted position. Figure 2 shows that the humerus is lowered until the motion has ceased or there is rotation of the humerus, indicating the end of posterior shoulder tissue flexibility. Any humeral rotation may indicate that other portions of the capsule are being tensioned rather than the posterior capsule, which is positioned in a direct line of pull. The tester is cautioned to palpate for a firm end feeling while lowering the arm and not to be fooled by the proximal humerus abutting into the chest in larger subjects.

Figure 3 shows the level of inferior border of the medial epicondyle being marked on the carpenter's square. The tester was blinded to the mark indicating the distance from the examination table to the medial epicondyle. The distance from the bottom of a carpenter's square to the mark identifying the medial epicondyle was recorded. The distance measured indicates the amount of flexibility of

Figure 1. The starting position for the posterior capsule flexibility measurement with the subject positioned correctly on his side.


Figure 2. The maximum passive range of motion of the posterior capsule. Note the scapular stabilization with the torso perpendicular to the examination table.

the posterior capsule tissues. A greater distance between the medial epicondyle and the examination table indicates less flexibility of the posterior capsule. Conversely, the closer the medial epicondyle is to the table (smaller distance), the more flexible the posterior capsule is. This measurement was taken bilaterally.

The tester also assessed passive bilateral external rotation of the shoulder with the humerus at 90° of abduction in the coronal plane and passive bilateral shoulder internal rotation with the humerus at 90° of abduction in the coronal plane. These measurements were taken with the patient supine. The tester was not blinded to the measurements of shoulder rotation range of motion.

The Reliability and Validity of Measuring Posterior Capsule Tightness

We previously described the reliability and validity of the posterior capsule measurement.[28] The validation testing
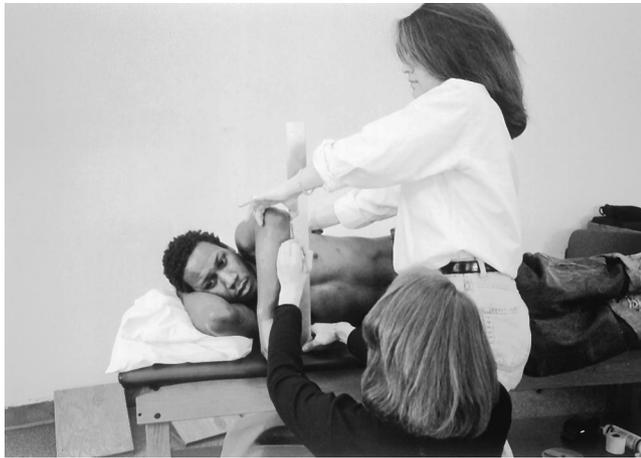
Figure 3. The recorder marks on the square how far the medial epicondyle is from the examination table.

was performed on NCAA division IA baseball pitchers by comparison with a control group. Baseball pitchers are a population known to have a decrease in internal rotation range of motion and an increase in external rotation range of motion in their dominant arm.[15, 17, 23, 24] In our previous study, the comparison of groups showed that pitchers demonstrated increased external rotation range of motion and decreased internal rotation range of motion and posterior capsule flexibility compared with the controls. On the basis of our regression analysis in that study, a clinician can expect that for every 4° of internal rotation range of motion lost, posterior capsule tightness will increase 1 cm using the method of measuring capsular tightness that we developed.[28]

Data Analysis

Independent *t*-tests were used to determine differences between impingement patients and controls and the effect of dominance on internal rotation range of motion, external rotation range of motion, and posterior capsule flexibility. Side-to-side differences in impingement patients

were calculated as the measurement for the affected side minus the measurement for the unaffected side, so that negative values indicate loss of motion in the affected side. For comparison of dominant-side impingement with controls, the difference in range of motion of controls was calculated as range of motion on the dominant side minus the range of motion on the nondominant side. The same controls were compared with patients with nondominant arm impingement by calculating range of motion on the nondominant side minus the range of motion on the dominant side. A Pearson's product-moment correlation coefficient was used to determine whether a relationship existed between shoulder rotation range of motion and posterior capsule tightness for each group. All values are shown as mean ± standard error of the mean (SEM) and alpha was set at $P < 0.05$.

RESULTS

Nineteen patients had impingement in their dominant extremity and 12 in their nondominant extremity. Table 1 presents measurements of posterior capsule tightness, internal rotation range of motion, and external rotation range of motion bilaterally and the difference between the involved and uninvolved shoulders in impingement patients and controls. Patients with impingement in their dominant shoulder demonstrated a statistically significant loss of internal rotation range of motion ($P < 0.001$) and greater posterior capsule tightness ($P = 0.011$) compared with controls (Fig. 4). Patients with dominant-arm impingement did not exhibit a significant loss of external rotation range of motion ($P = 0.47$). Patients with impingement in their nondominant shoulder demonstrated a statistically significant loss of internal rotation range of motion ($P = 0.04$), external rotation range of motion ($P = 0.02$) and greater posterior capsule tightness ($P = 0.03$) compared with controls (Fig. 5).
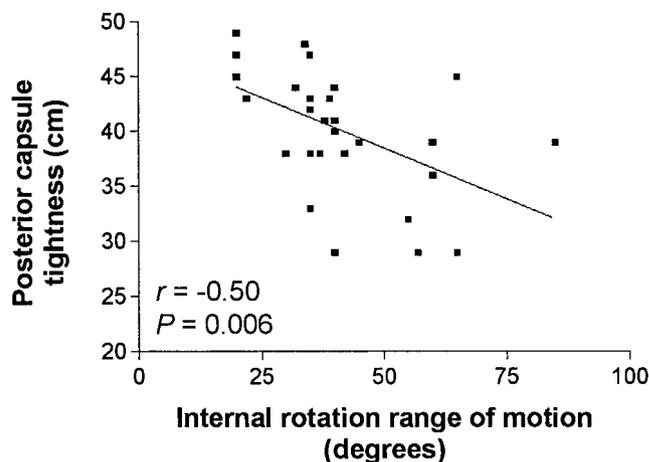
If posterior capsule tightness is related to a decrease in internal rotation range of motion, these values should be correlated. In fact, we observed a relationship between decreased internal rotation range of motion of increased

TABLE 1
Posterior Capsule Tightness and Range of Motion for All Groups (Mean ± SEM)

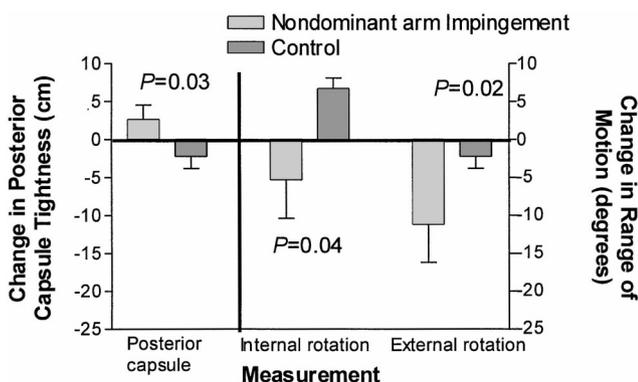| Group	Posterior capsule tightness (cm)	Range of motion	
		Internal rotation (degrees)	External rotation (degrees)
Control			
Dominant	31.65 ± 0.97	47.67 ± 1.49	96.09 ± 1.90
Nondominant	30.68 ± 0.90	54.36 ± 1.37	93.91 ± 2.02
Dominant–nondominant	0.97 ± 0.54	−6.69 ± 1.34	2.19 ± 1.62
Nondominant–dominant	−0.97 ± 0.54	6.69 ± 1.34	−2.19 ± 1.62
Impingement (dominant)			
Dominant	39.94 ± 1.27	38.71 ± 2.80	96.18 ± 2.02
Nondominant	33.50 ± 1.35	61.00 ± 2.64	95.82 ± 1.93
Dominant–nondominant	6.44 ± 1.87	−22.29 ± 2.03	0.36 ± 1.50
Impingement (nondominant)			
Dominant	37.71 ± 1.65	49.58 ± 4.29	95.58 ± 2.09
Nondominant	40.33 ± 1.84	44.58 ± 5.53	84.42 ± 4.64
Nondominant–dominant	2.62 ± 1.85	−5.00 ± 5.05	−11.16 ± 5.03
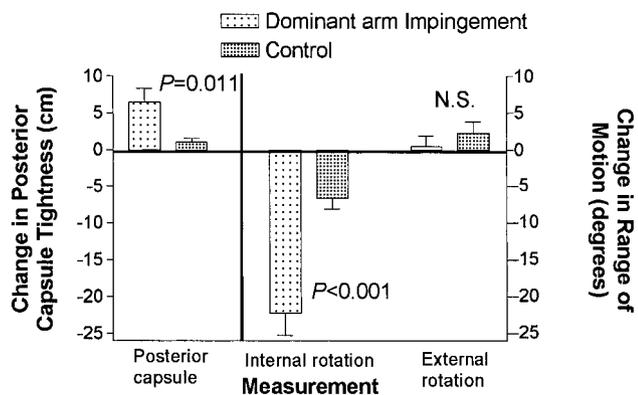
Figure 4. Comparison of range of motion and posterior capsule tightness deficits between patients with dominant-arm impingement and controls.

Figure 5. Comparison of range of motion and posterior capsule tightness deficits between patients with nondominant-arm impingement and controls.

Figure 6. Scatter diagram showing relationship of posterior shoulder tightness to internal rotation range of motion in shoulders with impingement. The solid line represents the least-squares regression.

cifically, the connection between posterior capsule tightness and dysfunction has long been recognized clinically but had not been empirically quantified until now.[9, 14, 18, 23, 29] Warner et al.[30] demonstrated a significant limitation of internal rotation range of motion and cross-chest adduction in a group of impingement patients compared with patients with instability and control patients. However, the correlation between these two measurements was not the aim of the study. Our results showed a significant correlation between internal rotation range of motion losses and increased posterior capsule tightness in patients with shoulder impingement using a measurement we previously validated.[28] On the basis of the results in this study and our previous study,[28] a clinician can expect that for every 4° of internal rotation range of motion lost there will be 1 cm of posterior capsule tightness.

Warner et al.[30] were unable to demonstrate posterior capsule tightness in the group of impingement patients as measured by a clinical drawer test. This test may not be sensitive enough to determine tightness in an arm without instability. In fact, the drawer test was described to determine anterior and posterior laxity, not tightness.[1,5] The grading system of 0 to 3+ commonly used to quantify the drawer test refers only to the amount of laxity, and there is no mention of grading tightness.[1,8] Our measurement of posterior capsule tightness was able to detect significant tightness when comparing patients with impingement to controls, inversely correlating tightness to the loss of internal rotation range of motion.

In agreement with previous studies on range of motion measurements in the upper extremity, our controls demonstrated a decrease in internal rotation range of motion in their dominant shoulder.[17, 28] To date there have been no published data on range of motion differences between dominant and nondominant shoulders in patients with impingement. Our results found that patients with impingement in their dominant arm did not exhibit a loss of

posterior capsule tightness ($r = -0.50$, $P = 0.006$) in the shoulders of patients with impingement (Fig. 6). Alternatively, there was no relationship found between external rotation range of motion and posterior capsule tightness ($r = -0.153$, $P = 0.496$).

DISCUSSION

Range of motion losses have been documented in patients with shoulder impingement.[18,30] It has been postulated, though previously unmeasured, that impingement patients lose internal rotation range of motion in their involved arm as a result of posterior capsule tightness.[15, 23, 24] The results of the present study are the first to clinically link posterior capsule tightness to the loss of internal rotation range of motion. Although patients with impingement in their non-dominant arm had less loss of internal rotation than patients with impingement in their dominant arms, patients with impingement in their nondominant arms also had loss of external rotation, resulting in a more profound loss of range of motion in the affected shoulder.

Patients with secondary impingement often ask the treating clinician to stretch the posterior capsule.[6, 7, 10−13, 18] Spe-

external rotation range of motion but had a lack of internal rotation range of motion accompanied by posterior capsule tightness. In like fashion, patients with impingement in their nondominant arm had a significant loss of internal rotation with posterior capsule tightness. In addition, patients with impingement in their nondominant arm demonstrated reduced external rotation range of motion. We believe that the increased loss of external rotation range of motion in these patients is a result of the reduced demand on the nondominant extremity to perform activities of daily living. The global motion loss in patients with impingement in their nondominant arm could also be attributed to these patients seeking treatment later than those who have impingement in their dominant arm. Patients may not seek treatment until there is a very significant loss of function in the nondominant extremity.

In previous studies, the methods used to evaluate the flexibility of the posterior capsule have not been precise.[23,30] Measuring with a goniometer, Warner et al.[30] used the cross-chest adduction test in the supine position and moved the tested arm into horizontal flexion until the scapula began to lift off the table. Scapular lifting would indicate the end of posterior capsule flexibility and the contribution of scapulothoracic motion. Warner et al.[30] did not account for glenohumeral rotation, nor did they closely monitor scapular motion that may occur beneath the skin before the scapula began to lift off the table. Furthermore, a goniometric measurement of horizontal flexion may be inexact because of the difficulty of keeping the fulcrum aligned on the glenohumeral joint while keeping the arms of the goniometer aligned with bony landmarks. Similarly, Pappas et al.[23] assessed posterior capsule tightness in baseball pitchers by goniometrically measuring supine subjects with manual stabilization of the scapula. A disadvantage of this technique is the inability to palpate and determine a reliable starting position of the scapula. Scapulae starting in a more protracted position may be mistaken for increased posterior shoulder flexibility. The reliability of those goniometric techniques has not been documented.

Our objective measurement of posterior capsule tightness has several advantages. Our results showed excellent reproducibility; in addition, our method allows the tester to constantly monitor scapulothoracic motion, thus ensuring that any increase in glenohumeral motion is a reflection of posterior capsule flexibility. When we measure range of motion with the subject in a side-lying position, the scapula lies retracted, thereby reproducing the scapular starting position for each test. Also, this method gives the tester the ability to detect glenohumeral rotation. Making the quantitative measure of elbow height in centimeters eliminates the possible inexact goniometric measurement. A drawback of the test is that variability of patient size does not allow for comparison of an absolute measurement of tightness between patients. Also, increased humeral head retroversion may be present in habitual throwers and this may resemble posterior capsule tightness.

A measurement of internal rotation motion alone is not enough to determine posterior capsule tightness. Based on the circle concept,[4] if there is anterior capsule tightness, a loss of internal rotation will result as the capsule winds on itself. O'Brien et al.[21] demonstrated that the posterior capsule is the primary restraint to any posterior force when the arm is positioned at 90° of abduction. It is likely that the primary structure that limits motion during the test is the posterior capsule of the shoulder. By putting the posterior capsule in a direct line of pull, keeping the rotation neutral, and getting a capsular end-feel, we are confident the posterior capsule is the primary restraint to additional adduction. In addition, distinguishing muscle tightness from capsule tightness may be achieved by accessing glenohumeral range of motion in different degrees of shoulder abduction. For example, patients who have intrinsic posterior capsule tightness will have more limitation of motion in 90° of abduction than they would at neutral because of the increased tension on the capsule in this position.

The results of this study demonstrate a relationship between posterior capsule tightness and a limitation in glenohumeral range of motion. While it is not known which adaptation came first, it is possible that patients may avoid putting their arm in a position of internal rotation to avoid pain caused by a mechanical impingement of the greater tuberosity on the subacromial arch and structures. This restriction of internal rotation motion may result in posterior capsule tightness. Conversely, posterior capsule tightness already present may be forcing the humeral head forward, causing mechanical impingement and a loss of range of motion as a result of the avoidance of painful movements. It is not clear which comes first, secondary shoulder impingement or posterior capsule tightness. In fact, there were a few subjects in our reproducibility study who had unilateral posterior capsule tightness but did not have impingement symptoms. It is our opinion that many factors contribute to secondary shoulder impingement. Three of the most prevalent findings are posterior capsule tightness, external rotation weakness in a position of 90° of shoulder abduction and 90° of elbow flexion, and abnormal scapulohumeral rhythm. Identifying which of these contributing factors surfaces first may be important in preventing secondary shoulder impingement and may be an area for future research.

The role that stretching the posterior capsule plays in restoring internal rotation range of motion has yet to be determined. There are numerous clinical applications for the new objective measurement of posterior capsule tightness that we developed.[28] Longitudinal measurements are important to further validate the clinical use of this measurement.

CONCLUSIONS

This study used a previously described clinically valid measurement technique to identify posterior capsule tightness in patients with shoulder impingement. Posterior capsule tightness measured clinically in impingement

patients showed a significant correlation to the loss of internal rotation range of motion. Patients with shoulder impingement in their nondominant arm demonstrated a more global loss of range of motion compared with patients having impingement in their dominant arm.

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