Correction of Posterior Shoulder Tightness Is Associated With Symptom Resolution in Patients With Internal Impingement

Timothy F. Tyler,*†‡ PT, ATC, Stephen J. Nicholas,‡ MD, Steven J. Lee,‡ MD, Michael Mullaney,‡ PT, and Malachy P. McHugh,‡ PhD
From †PRO Sports Physical Therapy, Scarsdale, New York, and the ‡Nicholas Institute of Sports Medicine and Athletic Trauma, New York, New York

Background: Glenohumeral internal rotation deficit (GIRD) and posterior shoulder tightness have been linked to internal impingement.

Purpose: To determine if improvements in GIRD and/or decreased posterior shoulder tightness are associated with a resolution of symptoms.

Study Design: Cohort study; Level of evidence, 3.

Methods: Passive internal rotation and external rotation (ER) range of motion (ROM) at 90° of shoulder abduction and posterior shoulder tightness (cross-chest adduction in side lying) were assessed in 22 patients with internal impingement (11 men, 11 women; age 41 ± 13 years). Treatment involved stretching and mobilization of the posterior shoulder. The Simple Shoulder Test (SST) was administered on initial evaluation and discharge. Changes in GIRD, ER ROM, and posterior shoulder tightness were compared between patients with complete resolution of symptoms versus patients with residual symptoms using independent t tests.

Results: Patients had significant GIRD (35°), loss of ER ROM (23°), and posterior shoulder tightness (35°) on initial evaluation (all $P < .01$). Physical therapy (7 ± 2 weeks; range, 3-12 weeks) improved GIRD (26° ± 14°; $P < .01$), ER ROM loss (14° ± 20°), and posterior shoulder tightness (27° ±19°). The SST improved from 5 ± 3 to 11 ± 1 ($P < .01$). A greater improvement in posterior shoulder tightness was seen in patients with complete resolution of symptoms ($n = 12$) compared with patients with residual symptoms (35° vs 18°; $P < .05$). Improvements in GIRD and ER ROM loss were not different between groups (GIRD, 25° vs 28°, $P = .57$; ER ROM, 14° vs 15°, $P = .84$).

Conclusion: Resolution of symptoms after physical therapy treatment for internal impingement was related to correction of posterior shoulder tightness but not correction of GIRD.

Keywords: impingement; posterior shoulder; glenohumeral internal rotation deficit (GIRD)

Glenohumeral internal rotation deficit (GIRD) and posterior shoulder tightness (PST) have been linked to internal impingement.[11] This link between GIRD and/or PST and internal impingement was based on the original series by Burkhart et al[2–4] that was published in the journal *Arthroscopy*. The mechanism by which GIRD and PST are associated with internal impingement has been elucidated in both cadaveric and clinical studies. In cadaveric studies, tightening the posterior capsule by plication has been shown to increase anterior glenohumeral translation during flexion and cross-body adduction, cause superior glenohumeral translation with flexion and external rotation of the glenohumeral joint, and markedly decrease internal rotation.[5–7] Similarly, GIRD and PST have been demonstrated in throwing athletes with internal impingement[11] and in patients with secondary impingement.[14] These cadaveric and clinical studies provide support for the theory that internal impingement results from altered glenohumeral mechanics secondary to PST. In line with this theory, arthroscopic release of the posterior capsule in patients with painful throwing shoulders was shown to provide symptomatic relief and allow successful return to preinjury sport levels.[17]

Considering that the majority of patients with internal impingement are initially treated nonsurgically, it is important

*Address correspondence to Timothy F. Tyler, PT, ATC, 2 Overhill Road, Scarsdale, NY 10583 (e-mail: tyler@nismat.org).

Presented at the 34th annual meeting of the AOSSM, Orlando, Florida, July 2008.

No potential conflicts of interest declared.

The American Journal of Sports Medicine, Vol. 38, No. 1
DOI: 10.1177/0363546509346050

to establish if a treatment focused on mobilizing and stretching the posterior shoulder provides successful outcomes. Traditionally, the treatment focus has been on resolving GIRD with less attention paid to PST. The extent to which improvements in glenohumeral internal rotation range of motion (ROM) versus improvements in posterior shoulder extensibility are associated with successful resolution of internal impingement is unknown. Therefore, the purpose of this study was to determine if improvements in GIRD and/or decreased PST after a course of physical therapy are associated with a resolution of symptoms in patients with internal impingement. We hypothesized that symptom resolution would be associated with decreased PST.

METHODS

Twenty-two patients (11 men and 11 women; age, 41 ± 13 years) with a diagnosis of internal impingement (18 in dominant arms, 4 in nondominant arms) were studied. The average duration of symptoms was 5 ± 5 months with a range of 1 to 24 months. The sports or activities primarily responsible for the injuries are listed in Table 1. Six patients had a superior labrum, anterior and posterior (SLAP) repair more than 6 months before this study and presented currently with residual internal impingement. The other 16 patients had no previous surgery on the involved shoulder.

The diagnostic criteria described by Heyworth and Williams[8] for internal impingement was used in this study. The diagnosis was made by an orthopaedic surgeon experienced in treating shoulder injuries (S.J.N.). Specific inclusion criteria based on physical examination were positive relocation test, positive posterior impingement sign, and posterior glenohumeral joint line tenderness. Specific inclusion criteria based on MRI findings were the presence of a posterosuperior glenoid labral lesion. Exclusion criteria were anterior instability, full-thickness rotator cuff tear, and subacromial impingement as determined by physical examination and MRI. Additionally, all patients reported subjective clicking in their shoulder on active movement. Four of the 22 patients had subtle anterior instability with no frank instability.

Procedures

At initial evaluation GIRD, PST, and external rotation ROM were documented for all patients. Patients also completed the Simple Shoulder Test (SST) questionnaire. After a course of physical therapy all measures were repeated and a posttreatment SST score was obtained. All subjects gave informed consent and the study was approved by institutional review board.

Measurement of Posterior Shoulder Tightness

The PST was measured with the patient in the side-lying position.[12,14,15] The patient laid with the thorax aligned perpendicular to the treatment table and the spine in neutral flexion, extension, and rotation. With the tester facing the patient, the lateral border of the scapula was manually stabilized in a retracted position. From a position of 90° of humeral abduction and neutral humeral rotation, the tester passively lowered the arm into horizontal adduction by gripping the participant's forearm just distal to the humeral epicondyles. The arm was lowered until the humeral horizontal adduction end range was reached or until the humerus started to rotate internally. At this end ROM, a second tester recorded the angle of the humerus in degrees relative to the horizontal plane. A positive number indicated tightness, that is, the arm was above the horizontal plane. Previously, PST has been documented with this test by measuring the distance between the medial epicondyle of the elbow and the table on which the patient is lying on his or her side, with a larger number indicating tightness. However, intersubject differences in trunk and chest width are confounding factors. Therefore, an angular measurement was used here to control for differences in the size of the patients (eg, women with a small frame vs men with a large frame). A digital level was used for all measurements (Smart Tool Technology, Oklahoma City, Oklahoma).

Glenohumeral Internal and External Range of Motion

Glenohumeral internal and external rotation ROM was measured with the patient supine, the arm abducted to 90°, and the elbow flexed to 90°. The forearm was held in supination and the digital level was placed on the dorsal aspect of the forearm for external rotation ROM and the ventral aspect of the forearm for internal rotation. Also, for internal rotation the scapula was monitored by palpating the spine of the scapula with the fingers and the coracoid with the thumb of that same hand while the opposite hand moved the tested arm into internal rotation.[16] For each measurement the arm was rotated passively to the end of the ROM. The ROM measurements were made with a digital level as opposed to a goniometer; therefore, it is important to appreciate the reliability of the measurements. The repeated measures on the noninvolved arm (pretreatment and posttreatment) provided an indication of measurement reliability. Test-retest Pearson product moment correlations were $r = .69$ for external rotation, $r = .70$ for internal rotation, and $r = .89$ for PST.

Glenohumeral rotation and PST measurements were made bilaterally and the same tester made all measurements, blinded to previous measurements.

Intervention

Patients were seen in the clinic 3 times a week and also prescribed a daily home exercise program. The primary component of the physical therapy intervention was manual mobilization and stretching of the posterior shoulder. Specific exercises included grade IV posterior glides of the glenohumeral joint in the scapular plane as described by Maitland[10] and in maximum glenohumeral internal rotation in 90° shoulder abduction[9] (Figure 1), active-assisted cross-chest adduction with manual stabilization of the scapula[1] (Figure 2), and the "sleeper stretch" (Figure 3). Also included were external rotation and scapular-stabilization strengthening exercises. The home exercise program included

TABLE 1
Study Population Injury Detail[a]

Injury Mechanism/ Sport	Involved Side	Age (y)	Gender	Duration of Symptoms (mo)	Prior Surgery	PST Deficit (deg)		GIRD (deg)		SST	
						Pre	Post	Pre	Post	Pre	Post
Ice skating (fall)	Dom	42	F	3	SLAP	67	41	49	8	1	11
Weight lifting	Dom	45	F	1	None	77	1	–6	9	1	12
Swimming	Dom	16	F	5	None	40	3	37	8	5	12
Basketball	Dom	40	M	8	None	38	1	24	10	8	12
Tennis	Dom	62	F	24	None	30	4	47	6	5	12
Tennis	Dom	39	F	1	None	42	15	18	7	4	12
Ice hockey	Nondom	22	M	6	None	9	–6	32	1	6	11
Tennis	Dom	17	F	2	None	13	9	24	0	7	12
Snowboarding	Dom	36	M	4	SLAP	8	3	25	6	9	11
Golf	Nondom	28	M	2	None	42	13	18	–10	9	12
Swimming	Nondom	48	M	12	None	31	–3	32	5	4	12
Tennis	Dom	54	M	4	SLAP	20	11	37	12	8	11
Tennis	Dom	53	M	9	SLAP	73	10	37	–7	9	12
Weight lifting	Nondom	57	F	4	None	42	6	45	15°	3	11
Softball	Dom	45	F	4	None	24	16	70	23	5	12
Golf	Dom	45	F	3	None	30	1	37	12°	6	9
Golf	Dom	59	M	12	None	48	0	26	3	2	12
Golf	Dom	46	M	2	None	18	20	14	9°	7	9
Softball	Dom	47	F	6	None	–3	–26	85	53	6	9
Tennis	Dom	45	F	3	SLAP	44	22	51	12°	0	11
Golf	Dom	41	M	12	None	33	7	38	3	2	9
Baseball	Dom	19	M	3	SLAP	51	17	29	6	5	12

[a] PST, posterior shoulder tightness; Pre, before treatment; Post, after treatment; GIRD, glenohumeral internal rotation deficit; SST, simple shoulder test; SLAP, superior labrum, anterior and posterior repair; Dom, dominant arm; Nondom, nondominant arm.

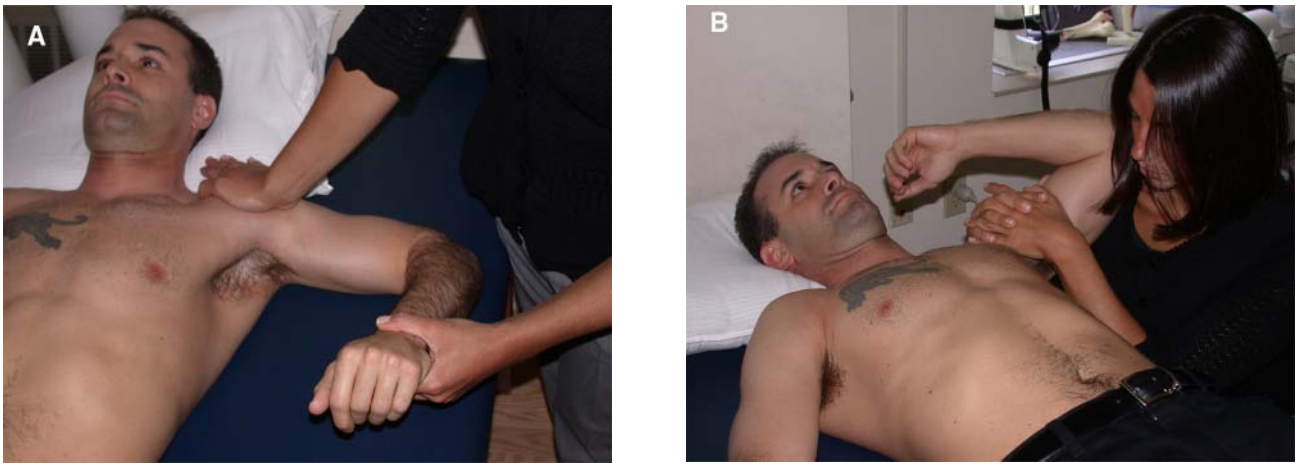

Figure 1. A, posterior glide in maximal passive, internal rotation range of motion with the humerus in the plane of the scapular. B, technique to provide distraction with a posterior glide at 90° of abduction. This is the authors' preferred technique to provide low load long duration mobilization.

the sleeper stretch, cross-chest adduction, external rotation, and scapular strengthening exercises. Physical therapy was continued until symptom relief and return to full activities or until a plateauing effect was apparent and patients were referred back to their physician.

Outcome Measure

The SST was administered on initial evaluation and discharge. This is a 12-component questionnaire with "yes" and "no" responses regarding symptoms and function with activities of daily living and functional tasks.

Data Analysis

Based on the authors' clinical experience it was expected that some patients would have complete resolution of symptoms and some would have residual symptoms. The SST score is a 0 to 12 score. Because it was expected that many

Figure 2. Cross-chest adduction with manual stabilization of the scapula at the acromion and the inferior angle.

Figure 3. Sleeper stretch.

patients would score 12 after treatment (complete resolution) and the range of scores would be quite small, it was determined that it would be more appropriate to categorize patients based on their final SST score rather than treat it as a continuous variable. Therefore, changes in GIRD and PST with physical therapy were compared between patients with and without residual symptoms using a mixed model analysis of variance, with Treatment (pretreatment vs posttreatment) as the within-subjects factor and Group (patients with complete resolution of symptoms vs patients with residual symptoms) as the between-subjects factor. These analyses provided a determination of how improvements in GIRD and PST affected treatment outcome. Measurement reliability for glenohumeral internal and external rotation ROM and PST was documented by computing the standard error of the measurement for repeated measures (pretreatment vs final follow-up) of the noninvolved arm.

The optimal sample size estimate for this study would be based on the documented intersubject variability in the change in GIRD and PST with treatment. Because such data are not available in the literature, the intersubject variability in GIRD and PST was used; it was assumed that these factors would be similar between groups before treatment and, thus, posttreatment measures would reflect treatment effects. Based on previously reported intersubject variability in GIRD for patients with shoulder impingement,[14] it was estimated that an 11° difference in GIRD between patients with residual symptoms and patients with resolution of symptoms could be detected with 80% power at $P < .05$ in a sample of 20 patients (assuming relatively equal group sizes). The modified PST measurement used here (angular vs linear measurement) was used previously to document PST in patients with various shoulder lesions (C. J. Johnson, M. J. Mullaney, and M. P. McHugh, unpublished data, 2007). Based on those data it was estimated that an 8° difference in GIRD between patients with residual symptoms and patients with resolution of symptoms could be detected with 80% power at $P < .05$ in a sample of 20 patients (assuming relatively equal group sizes).

RESULTS

Before treatment, the SST score for the patient sample was 5 ± 3 (of a maximum score of 12). Patients had significant GIRD (35° ± 19°, $P < .001$), loss of external rotation ROM (23° ± 35°, $P < .001$), and increased PST (35° ± 21°, $P < .001$) (data are differences between the involved and noninvolved sides for each test with a positive number indicating loss of motion on the involved side). Pretreatment SST score was correlated with PST ($r = –.42$, $P < .05$) with lower SST scores associated with greater PST. Glenohumeral internal rotation deficit and loss of external rotation ROM were not correlated with the SST score ($r = –.09$, $P = .69$ and $r = –.23$, $P = .3$, respectively).

The average duration of physical therapy was 7 ± 2 weeks (range, 3-12 weeks). There was a significant improvement in SST scores (5 ± 3 to 11 ± 1, $P < .01$), with 12 patients having complete resolution of symptoms (12 of 12) and 10 patients having residual symptoms (9 of 12 in 4 patients and 11 of 12 in 6 patients). The residual symptoms reported on the SST at final follow-up were relatively consistent among the patients. Seven of 10 patients gave a negative response to the question, "Do you think you can toss a softball overhand 20 yards with the affected extremity?" Four of 10 patients gave a negative response to the question, "Can you wash the back of your opposite shoulder with your affected extremity?" Nine of the 10 patients with residual symptoms gave a negative response to 1 or both of these questions.

Glenohumeral internal rotation deficit, PST, and loss of external rotation ROM were all significantly improved after the period of physical therapy ($P < .01$). At final follow-up GIRD was 9° ± 12° (deficit $P < .01$), PST was 7° ± 13° (deficit $P < .05$), and loss of external rotation ROM was 9° ± 21° (deficit $P = .06$). Improvement in PST was greater in patients who had complete resolution of symptoms versus patients with residual symptoms ($P < .05$) (Figure 4). Improvements in GIRD (Figure 5) and external rotation ROM (Figure 6) were not different between groups ($P = .57$

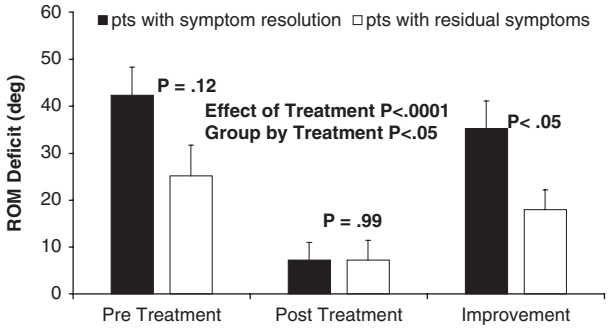

Figure 4. Effect of treatment on posterior shoulder tightness in patients with and without resolution of symptoms. Mean and standard error for involved/uninvolved deficits are shown.

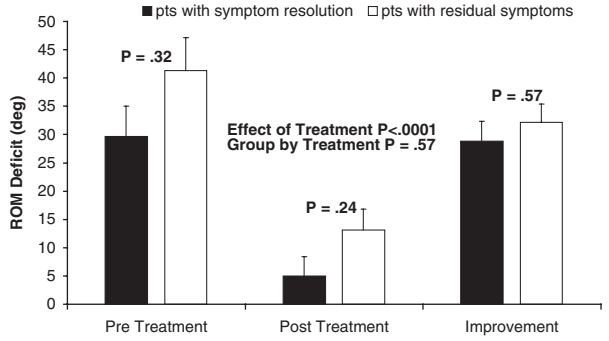

Figure 5. Effect of treatment on glenohumeral internal rotation deficit in patients with and without resolution of symptoms. Mean and standard error for involved/uninvolved deficits are shown.

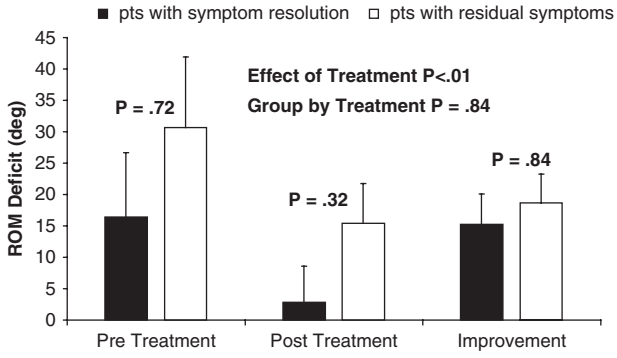

Figure 6. Effect of treatment on loss of external rotation range of motion in patients with and without resolution of symptoms. Mean and standard error for involved/uninvolved deficits are shown.

and P = .84, respectively). The average improvement in PST for all 22 patients was 27°. Nine of 12 patients (75%) with complete resolution of symptoms improved their PST by at least 27° compared with 2 of 10 patients (20%) with residual symptoms (P < .05). The average improvement in GIRD was 26°. Six of 12 patients (50%) with resolution of symptoms had improved GIRD by at least 26° compared with 5 of 10 patients (50%) with residual symptoms (P = .99). The duration of symptoms was 7 ± 7 months for patients with complete resolution of symptoms and 5 ± 3 months for patients with residual symptoms (P = .36).

The standard error of the measurement was 4.0° for both glenohumeral internal and external rotation ROM, and 4.2° for PST, indicating good reliability for these measures repeated over a period of several weeks.

DISCUSSION

The physical therapy treatment intervention focusing on stretching and mobilizing the posterior shoulder resulted in a decrease in PST, a decrease in GIRD, a decrease in the loss of external rotation ROM, and a marked improvement in symptoms, as measured by the SST score. Twelve patients (55%) had complete resolution of symptoms and 10 patients (45%) had some residual symptoms. Before treatment, PST was associated with lower SST scores (worse symptoms), and GIRD and loss of external rotation ROM were unrelated to symptoms. More importantly, patients with complete resolution of symptoms had greater improvements in PST than patients with residual symptoms, and improvements in GIRD and loss of external rotation ROM were unrelated to outcome.

The association between improvement in PST and resolution of symptoms appeared to be related to the fact that patients who subsequently had complete resolution of symptoms tended to have greater PST before treatment compared with patients who subsequently had residual symptoms (Figure 5). Therefore, it might be that patients with internal impingement and marked PST are more likely to have a successful outcome with physical therapy than patients with less pronounced PST. These patients may be more responsive to a treatment focused on the posterior shoulder. In the present study, 13 patients with a PST deficit greater than 25° also had an improvement in PST deficit of greater than 25°; 10 had complete resolution of symptoms (77% success) and 3 had residual symptoms. Of the other 9 patients whose pretreatment PST was less than 25°, only 2 had a complete resolution of symptoms (22% success). It is important to note that there were no baseball pitchers in this patient population. In our experience, pitchers with impingement may have less PST and more humeral head retroversion and increased GIRD.

A confounding factor in assessing deficits between involved and noninvolved arms in patients with shoulder injury is whether the pathologic changes are in the dominant or nondominant arm. Deficits in PST and GIRD are more pronounced in patients whose dominant arm is involved.[14] In the present study the nondominant arm was involved in 5 patients. Two of these patients had a PST deficit less than 25° before treatment (9° and –2°) and subsequent improvements in PST deficits were less than 25° (24° and 15°). Both patients had residual symptoms. The other 3 patients whose nondominant arm was involved had marked PST on the involved side (deficits of 31° to 42°) that improved by 29° to 36° after treatment, and 2 of the 3 patients had complete resolution of symptoms. Therefore, it appears that inclusion of patients with internal impingement in the nondominant arm did not confound the results of the study.

Based on the standard deviation of test-retest differences in the present study, there was 80% power to detect a 9° change in internal and external rotation ROM and a 13° change in PST at $P < .05$. These post hoc estimates are in line with a priori power estimates reported in the "Methods" section. Therefore, the digital level was shown to be sufficiently reliable to detect clinically relevant changes in glenohumeral ROM and PST.

There were 6 patients in this population who had a SLAP repair more than 6 months earlier and were seen with internal impingement; all 6 responded to this treatment approach. Residual pain after this type of surgery has been reported in the literature.[13] Sonnery-Cottet et al[13] documented symptomatic posterosuperior glenoid impingement in 28 tennis players, which limited their participation. Diagnostic arthroscopic examination revealed posterosuperior glenoid impingement in all patients. All patients underwent debridement of the lesions. This consisted of smoothing the posterosuperior labral lesion in 14 patients and resection of a bucket-handle type of lesion in 11 patients. All patients underwent debridement of the damaged deep surface of the supraspinatus tendon. After arthroscopic shoulder surgery, 22 patients had returned to tennis; despite their return, 20 of the 22 patients reported some persistent pain with participation. It may be possible that surgery in this subset of patients is addressing the structural deficiencies of the shoulder but not the residual PST.

In conclusion, the treatment technique used in this study was an effective method of improving symptoms, decreasing PST, and decreasing GIRD. Resolution of symptoms after physical therapy treatment for internal impingement was related to correction of PST but not correction of GIRD.

REFERENCES

1. Bang MD, Deyle GD. Comparison of supervised exercise with and without manual physical therapy for patients with shoulder impingement syndrome. *J Orthop Sports Phys Ther.* 2000;3(30):126-137.
2. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology. Part I: pathoanatomy and biomechanics. *Arthroscopy.* 2003;19(4):404-420.
3. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology. Part II: evaluation and treatment of SLAP lesions in throwers. *Arthroscopy.* 2003;19(5):531-539.
4. 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(6): 641-661.
5. Clabbers KM, Kelly JD, Bader D, et al. Effect of posterior capsule tightness on glenohumeral translation in the late-cocking phase of pitching. *J Sport Rehabil.* 2007;16(1):41-49.
6. Grossman MG, Tibone JE, McGarry MH, Schneider DJ, Veneziani S, Lee TQ. A cadaveric model of the throwing shoulder: a possible etiology of superior labrum anterior-to-posterior lesions. *J Bone Joint Surg Am.* 2005;87(4):824-831.
7. Harryman DT, Sidles JA, Clark JM, McQuade KJ, Gibb TD, Matsen FA III. Translation of the humeral head on the glenoid with passive glenohumeral motion. *J Bone Joint Surg Am.* 1990;72(9): 1334-1343.
8. Heyworth BE, Williams RJ 3rd. Internal impingement of the shoulder. *Am J Sports Med.* 2009;37(5):1024-1037.
9. Hsu AT, Ho L, Ho S, Hedman T. Joint position during anterior-posterior glide mobilization: its effect on glenohumeral abduction range of motion. *Arch Phys Med Rehabil.* 2000;81(2):210-214.
10. Maitland GD. *Peripheral Manipulation.* London: Butterworth-Heinmann; 1991.
11. Myers JB, Laudner KG, Pasquale MR, Bradley JP, Lephart SM. Glenohumeral range of motion deficits and posterior shoulder tightness in throwers with pathologic internal impingement. *Am J Sports Med.* 2006;34(3):385-391.
12. Myers JB, Oyama S, Wassinger CA, et al. Reliability, precision, accuracy, and validity of posterior shoulder tightness assessment in overhead athletes. *Am J Sports Med.* 2007;35(11):1922-1930.
13. Sonnery-Cottet B, Edwards TB, Noel E, Walch G. Results of arthroscopic treatment of posterosuperior glenoid impingement in tennis players. *Am J Sports Med.* 2002;30(2):227-232.
14. Tyler TF, Nicholas SJ, Roy T, Gleim GW. Quantification of posterior capsule tightness and motion loss in patients with shoulder impingement. *Am J Sports Med.* 2000;28(5):668-673.
15. Tyler TF, Roy T, Nicholas SJ, Gleim GW. Reliability and validity of a new method of measuring posterior shoulder tightness. *J Orthop Sports Phys Ther.* 1999;29(5):262-269.
16. Wilk KE, Reinold MM, Macrina LC, et al. Glenohumeral internal rotation measurements differ depending on stabilization techniques. *Sports Health.* 2009;1(2):131-136.
17. Yoneda M, Nakagawa S, Mizuno N, et al. Arthroscopic capsular release for painful throwing shoulder with posterior capsular tightness. *Arthroscopy.* 2006;22(7):801.e1-5. *doi:10.1016/j.arthro. 2005. 12.056.*