

## SECTION II

# ORIGINAL ARTICLES

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### Internal Impingement of the Shoulder in Flexion

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Contact of the rotator cuff to the superior glenoid with the arm in flexion has been described and postulated to be a source of rotator cuff disease. The goals of the current study were to document the existence of internal impingement in flexion arthroscopically and to determine its prevalence in patients with various diseases. Also, we attempted to determine the clinical significance of internal impingement in flexion by investigating the associations between internal impingement in flexion and the preoperative and intraoperative findings. A consecutive case series of 376 patients having arthroscopy of the shoulder were entered prospectively into this study. During arthroscopy, intraarticular lesions were evaluated and the presence of contact of the rotator cuff to the superior glenoid and the degree of flexion making the contact were noted. Statistical analysis was done with two dependent variables defined: the presence of internal impingement in flexion and the flexion degree making internal impingement in flexion. Of the 376 patients, 277 (74%) had internal impingement in flexion and 99 (26%) did not have internal impingement in flexion. There were no statistically significant differences in the prevalence of internal impingement in flexion according to the primary diagnoses. Statistical analysis revealed that the presence of internal impingement in flexion was associated with Type II superior labrum anterior posterior (SLAP) lesions and the presence of internal impingement of the rotator cuff to the superior glenoid in abduction and external rotation. There was a significant re-

lationship between rotator cuff disease and decreasing angle of contact for internal impingement in flexion. This study showed that internal impingement in flexion is common in a cohort of patients having shoulder surgery, with an overall prevalence of 74%, and that internal impingement in flexion may contribute to the development of Type II SLAP lesions and rotator cuff disease.

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The pathogenesis of rotator cuff disease continues to be controversial,<sup>13,25,30,32,33,48</sup> but an emerging consensus is that rotator cuff disease is multifactorial.<sup>16,41</sup> These etiologic factors have been grouped into intrinsic and extrinsic factors. Intrinsic factors involving the rotator cuff tendons or muscles include attrition,<sup>30</sup> aging accompanied with weakness and degeneration,<sup>17,35</sup> and vascular deterioration.<sup>12,17</sup> Extrinsic factors including trauma,<sup>13</sup> subacromial impingement,<sup>32,33</sup> and internal impingement on the glenoid rim have been suggested as causes of rotator cuff disease.<sup>22,24,25,48</sup>

In 1972, Neer<sup>32</sup> proposed that most rotator cuff injuries resulted from mechanical compression of the tendons under the coracoacromial arch. The clinical success of anterior acromioplasty and rotator cuff repair led to widespread acceptance of Neer's hypothesis. There have been numerous anatomic and clinical studies supporting impingement as a cause of rotator cuff diseases.<sup>5,6,15</sup> However, other studies have challenged Neer's hypothesis. In normal shoulders, the impingement of the greater tuberosity or adjacent rotator cuff to the acromial arch does not seem to occur.<sup>13,14,38</sup> In cadaveric studies by Ogata and Uthoff<sup>35</sup> and Ozaki et al,<sup>36</sup> specimens having articular-side partial-thickness rotator cuff tears had no pathologic degenerative changes on the undersurface of the acromion. Conversely, specimens with either bursal-side partial-thickness tears or full-thickness tears had no acromial

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changes present. Bright et al<sup>8</sup> questioned the role of the acromion shape in the pathogenesis of rotator cuff disease and contradicted the validity of the radiologic evaluation of the acromion shape as an etiologic factor in rotator cuff disease.

Although numerous case series have reported good results after arthroscopic subacromial decompression or open anterior acromioplasty, the rate of satisfaction ranges widely from 46–100%.<sup>5,6,9,33,42,43</sup> Some studies report greater than 90% satisfactory results after subacromial decompression, but a high percentage of unsatisfactory results has been reported in several other studies, particularly in young athletic patients.<sup>7,11,26,27,45</sup> The poor results of acromioplasty in young patients has been attributed to underlying glenohumeral instability.<sup>26,27,45</sup>

This association between instability and rotator cuff disease has been explained two ways: secondary impingement and internal impingement. The concept of secondary impingement suggests that in the unstable shoulder, forward elevation of the arm is accompanied by superior translation of the humeral head. This potentially leads to impingement of the greater tuberosity or rotator cuff on the coracoacromial arch.<sup>26,27</sup> The concept of internal impingement was introduced by Walch et al.<sup>48</sup> They described contact of the rotator cuff to the posterior and superior glenoid when the arm is in abduction and external rotation when viewing the joint arthroscopically during surgery. They hypothesized that this impingement might be physiologic, but in throwing athletes the repetitive nature of the impingement might lead to lesions on the undersurface of the rotator cuff and the posterior glenoid rim. Several investigators expanded the concept of internal impingement with findings of the anatomic, kinesiological,<sup>25</sup> MRI,<sup>23</sup> and arthroscopic data.<sup>37</sup>

Gerber and Sebesta<sup>19</sup> suggested that impingement of the rotator cuff may occur to the coracoid with the arm in flexion, elevation, and internal rotation. Jobe<sup>23</sup> suggested that impingement of the rotator cuff may occur to the superior glenoid with the arm in flexion. Contact of the rotator cuff to the superior glenoid was shown by MRI and cadaveric studies.<sup>23,47</sup> A recent study postulated that partial cuff tears were attributable to this impingement to the anterior superior glenoid.<sup>44</sup>

The pathogenic role of internal impingement in flexion for rotator cuff disease has not been elucidated fully. In a study by Jobe,<sup>23</sup> six volunteers who had no history of shoulder instability were placed in an MRI gantry in full shoulder elevation and internal rotation to mimic the Neer impingement sign. In all six volunteers, the greater tuberosity approximating the superior glenoid with some deformation of the soft tissues between these structures was observed. Internal impingement in flexion also was investigated in a cadaveric study by Valadie et al.<sup>47</sup> All five

shoulders placed in the flexion position showed contact between the articular surface of the rotator cuff tendons and the anterosuperior glenoid rim. In a study examining 1232 skeletal shoulder specimens, Edelson and Teitz<sup>14</sup> described the evidence of subacromial impingement and internal impingement. They suggested that internal impingement between the glenoid and the humeral head might be a significant mechanism in the development of rotator cuff disease.

When doing shoulder arthroscopy, we noticed this contact of the rotator cuff to the superior glenoid with the arm in a flexed position as described by Jobe.<sup>23</sup> However, the prevalence of this internal impingement in flexion has not been established because previous studies were of cadaveric specimens, of a limited number of subjects studied with MRI, or small case series.<sup>44,46</sup> There have been no studies evaluating this phenomenon as it relates to the clinical entities of impingement or instability. No study has evaluated the relationship of contact of the rotator cuff to the glenoid with the arm in flexion to symptoms or physical examination findings of rotator cuff disease. The goals of the current study were to document the existence of internal impingement in flexion arthroscopically, to determine its prevalence in patients with various diseases, and to investigate the association of internal impingement in flexion with the preoperative and intraoperative findings. Our hypotheses were that this impingement would be common and that it would be associated with symptoms in patients with rotator cuff disease.

## MATERIALS AND METHODS

Between 1995 and 2000, 590 patients having shoulder surgery were examined prospectively and their information was entered into a database. At our institution, most patients had arthroscopic evaluation of the joint before a more definitive procedure. Three hundred seventy-six patients who had diagnostic arthroscopy of the shoulder were included in this study and 214 patients were excluded. Excluded were 133 patients who did not have diagnostic arthroscopy because of fracture ( $n = 22$ ), arthroplasty ( $n = 77$ ), revision surgery of our patients ( $n = 13$ ), or isolated open procedures for any reason ( $n = 21$ ). The 49 patients who had arthroscopic evaluation at our institution, but had previous surgeries on the same shoulder were excluded. Thirty-two patients who had surgery in which the observation of internal impingement in flexion was not reliable because of severe synovial hypertrophy or labral fraying were excluded. There were 219 male and 157 female patients. The mean age of the patients was 41.5 ( $\pm 17.2$ ) years, ranging from 13–86 years. All patients provided informed consent before their procedures. This study received institutional review board approval.

All patients had a thorough preoperative assessment which included a detailed history and physical examination. In all patients this examination was within 4 weeks of the operative procedure. Included in the history were the etiology of their shoulder problem, sports activities, and occupation. The sports

activities were divided by level (recreational, high school, college, and professional levels) and whether the athlete was involved in an overhead sport. Provocative maneuvers such as an apprehension test and a relocation maneuver were done.

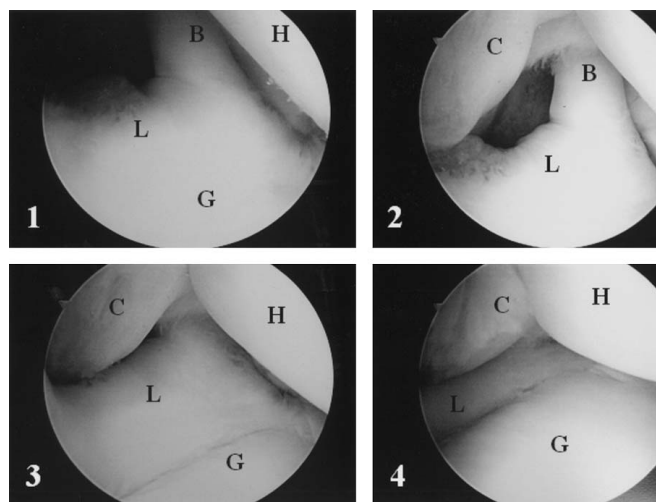
All patients had general anesthesia with or without a scalene block. The surgical procedure and examination were done by the senior author in all patients. Both shoulders of every patient were examined for ROM and laxity testing. Laxity tests included an anterior and posterior drawer as described by Gerber and Ganz.<sup>18</sup> The degree of laxity was expressed using a modified Hawkins scale (Grade I, to the rim; Grade II, over the rim, and Grade III, locks out) as previously reported.<sup>28</sup> Sulcus testing was done and graded using standard criteria (Grade I, < 1.0 cm; Grade II, 1.0–2.0 cm; and Grade III, > 2.0 cm).<sup>1,34</sup>

All procedures were done with the patient in a lateral decubitus position in a bean bag. Joint distension was provided with a pump set at 80 mm Hg.<sup>28,29</sup> All arms were suspended in an arm holder with 10 lb of traction. The arthroscope was placed in a posterior portal and a thorough diagnostic arthroscopy was done. Particular attention was paid to the presence of any rotator cuff disorder, biceps tendon or biceps tendon attachment disorder, labrum lesions either anteroinferiorly or posterosuperiorly, and to other signs of instability.

The surgical arm was removed from the arm holder so that there was no traction on the arm, and it was elevated passively by an assistant into forward flexion. Rotation was controlled by holding the arm with the palm down. During testing of contact of the rotator cuff to the glenoid the arthroscope was held at the posterior synovial margin to prevent any leverage of the scope against the humeral head as previously used by us and others reporting on rotator cuff contact to the superior glenoid.<sup>19,28,48</sup> Contact of the rotator cuff to the superior glenoid at the level of the biceps tendon or within 1 cm posteriorly of the biceps attachment was considered a positive sign for the presence of an internal impingement in flexion (Fig 1). The arm then was placed into abduction and external rotation, and internal contact of the rotator cuff to the posterosuperior glenoid was noted as previously reported.<sup>28,48</sup> The examiner stabilized the thorax, but not specifically the scapula. Subacromial arthroscopy was not done in patients in whom it was not indicated and observation of contact of the rotator cuff to the acromion was not done because it was not the focus of this study.

A primary diagnosis was made for every patient based on the history, physical examination, and arthroscopic findings. Patients who had more than one diagnosis were given a primary diagnosis dependent on their primary procedures. For example, a patient with instability and a partial cuff tear was considered to have a primary diagnosis of instability if the treatment was shoulder reconstruction. Primary diagnoses included: rotator cuff disease (impingement syndrome without tear, partial-thickness rotator cuff tear, and full-thickness rotator cuff tear), 182 patients (48%); glenohumeral instability, 125 patients (33%); acromioclavicular joint arthritis, 47 patients (13%); frozen shoulder, 13 patients (4%); and other diseases such as synovial cyst in nine patients (2%).

Statistical analysis using the SPSS (version 10.0; SPSS, Chicago, IL) was done with two dependent variables defined. The



**Fig 1.** Internal impingement in flexion is shown through a posterior portal in a right shoulder. (1) This photograph was taken at a lower flexion angle and the rotator cuff does not make contact. (2) In this photograph, taken at an increased flexion angle, the undersurface of the rotator cuff starts to make contact with the biceps tendon. (3) At an additionally increased flexion angle, the cuff nearly makes contact to the labrum and glenoid. (4) In this photograph, taken at the greatest flexion angle, the undersurface of the rotator cuff with partial tears makes close contact with the labrum. B, the biceps tendon; C, the undersurface of the rotator cuff; G, the glenoid; H, the humeral head; and L, the labrum.

first dependent variable was whether internal impingement in flexion was observed at the shoulder arthroscopy. The second dependent variable was the flexion degree making internal impingement in flexion. Independent variables included demographic data: the gender, age, involvement of dominant arm, occupation (high-demand versus low-demand occupations), participation in overhead sports (overhead throwing sports, overhead racket sports, and swimming), and participation level of sports (recreational level versus equal to or higher than high-school level). Other independent variables were subjective symptoms, physical examination findings, laxity testing findings, arthroscopic findings, and primary diagnoses. For the purpose of analysis, all categorical independent variables were dichotomized.

In the analysis with the presence of internal impingement in flexion defined as a dependent variable, univariate analysis was done with the use of Student's *t* test for continuous variables and chi square test for categorical variables. For the significant variables in these initial analyses, the 95% confidence interval and the odds ratios were calculated using univariate logistic regression. In the analysis with the flexion degree making internal impingement in flexion defined as a dependent variable, univariate analysis was done using Student's *t* test for the categorical independent variables and correlation analysis for the continuous independent variables. Statistical significance was set as *p* less than 0.05.

## RESULTS

The prevalence of internal impingement in flexion in the patients of this study was 74% (95% confidence interval, 73.2% – 78.1%). Of the 376 patients, 277 (74%) had an internal impingement in flexion and 99 (26%) did not. There were no statistically significant differences in the prevalence of internal impingement in flexion according to the primary diagnoses (Table 1).

In the univariate analysis with the presence of internal impingement in flexion, defined as a dependent variable, there were no significant differences between the internal impingement in flexion and noninternal impingement in flexion groups in all demographic variables except for patient age and high level of sports activity. The mean age of the patients with internal impingement in flexion was 40.2 years ( $\pm 17.1$ ; range, 13–86 years) whereas the mean age of the patients with no internal impingement in flexion was 44.9 years ( $\pm 17.0$ ; range, 14–83 years). Twenty (21%) of the 99 patients who did not have internal impingement in flexion and 91 (33%) of the 277 patients who had internal impingement in flexion had participated in sports activity at a level higher than the recreational level ( $p = 0.03$ ). There was no difference in the proportion of patients with overhead sports activity between the group with internal impingement in flexion and the group with noninternal impingement in flexion group (25%, 68/277 versus 19%, 19/99;  $p = 0.278$ ). In the analysis of prevalence of intraarticular disorders according to overhead sports activity, the patients with overhead sports activity had significantly higher prevalence of Type II SLAP lesions (14%, 12/87 versus 5%, 15/289;  $p = 0.006$ ).

Subjective symptoms evaluated by the visual analog scale did not show any significant differences between the internal impingement in flexion and noninternal impinge-

ment in flexion groups. Most of the physical examination findings did not show significant differences between the two groups (Table 2). A positive painful arc sign was less common in the contact group (40% in the internal impingement in flexion group versus 61% in the noninternal impingement in flexion group;  $p = 0.003$ ). Active and passive ROMs were increased in most positions, but passive external rotation and passive internal rotation at 90° abduction were significantly greater in the internal impingement in flexion group: mean passive external rotation, 97° ( $\pm 17.8^\circ$ ) in the internal impingement in flexion group versus 87° ( $\pm 25.9^\circ$ ) in the noninternal impingement in flexion group ( $p = 0.002$ ); and mean passive internal rotation, 50° ( $\pm 25.5^\circ$ ) in the internal impingement in flexion group versus 42° ( $\pm 25.8^\circ$ ) in the noninternal impingement in flexion group ( $p = 0.024$ ).

Univariate analysis of intraarticular disorders revealed that internal impingement in flexion was positively associated with Type II SLAP (superior labrum anterior posterior) lesions and negatively associated with humeral head OA (Table 3). Internal impingement in flexion also was associated significantly with the presence of internal impingement of the rotator cuff to the superior glenoid in abduction and external rotation. Whereas internal impingement in abduction and external rotation was observed in 61 (64%) of the 96 patients in the noninternal impingement in flexion group, 245 (92%) of the 265 patients in the internal impingement in flexion group were observed to have internal impingement in abduction and external rotation (odds ratio = 6.83,  $p = 0.000$ ). The mean degree of external rotation resulting in abduction and external rotation internal impingement was significantly lower in the patients who also had an internal impingement in flexion ( $p = 0.000$ ).

In the 277 patients of the internal impingement in flexion group, the mean flexion degree making an internal impingement in flexion was 124° ( $\pm 17.4^\circ$ ), ranging from 70° to 165°. Univariate analysis with the flexion degree making an internal impingement in flexion, defined as a dependent variable, was done. This revealed that the variables related to rotator cuff disease were associated significantly with decreasing flexion degree making an internal impingement in flexion (Table 4). A primary diagnosis of rotator cuff disease (mean flexion angle, 119  $\pm$  17.6 versus 127  $\pm$  16.3;  $p = 0.000$ ), increasing age, positive painful arc sign (117  $\pm$  18.0 versus 125  $\pm$  17.7;  $p = 0.003$ ), Type II SLAP lesion (117  $\pm$  15.1 versus 124  $\pm$  17.5;  $p = 0.033$ ), supraspinatus tears (119  $\pm$  17.3 versus 128  $\pm$  16.5;  $p = 0.000$ ), and humeral head OAs (118  $\pm$  17.5 versus 124  $\pm$  17.2;  $p = 0.048$ ) were associated significantly with decreasing flexion degree making an internal impingement in flexion.

**TABLE 1. Prevalence of Internal Impingement in Flexion in the Patients with Different Primary Diagnoses**

Primary Diagnosis	Prevalence of Internal Impingement in Flexion*
Rotator cuff disease (impingement syndrome, partial tears, and full tears)	131/182 (72)
Glenohumeral instability	92/125 (74)
Acromioclavicular disease	36/47 (77)
Frozen shoulder	10/13 (77)
Others (synovial cyst, ON, glenohumeral arthritis)	8/9 (89)
Total (376)	277/376 (74)

\*The data are given as the number of patients with the percentage in parentheses.

**TABLE 2. Univariate Analysis of the Physical Examination Findings in the Two Groups Divided by the Presence of Internal Impingement in Flexion**

Variable	Descriptive Comparison*		Univariate Analysis		
	Noninternal Impingement in Flexion (n = 99)	Internal Impingement in Flexion (n = 277)	Odds Ratio	Confidence Interval (95%)	p Value
Neer impingement sign (positive)	62/99 (63)	162/277 (58)			> 0.2
Hawkins impingement sign (positive)	67/99 (68)	178/272 (65)			> 0.2
Speed test (positive)	34/96 (35)	78/253 (31)			> 0.2
Painful arc test (positive)	48/79 (61)	65/163 (40)	0.43	0.25–0.74	0.003
Drop arm test (positive)	24/91 (26)	44/232 (19)	0.65	0.37–1.16	0.143
Compression rotation test (positive)	22/71 (31)	33/146 (23)	0.65	0.35–1.23	0.184
Anterior slide test (positive)	12/86 (14)	27/208 (13)			> 0.2
Active compression test (positive)	46/86 (53)	84/211 (40)	0.58	0.35–1.23	0.184
Apprehension test (positive)	18/95 (19)	64/268 (24)			> 0.2
Relocation test for anterior instability (positive)	11/68 (16)	37/143 (26)	1.81	0.86–3.81	0.120
Range of motion (odds ratio per degree)					
Active flexion	140 ± 32.9	146 ± 30.6	1.01	1.00–1.01	0.109
Passive flexion	149 ± 26.7	154 ± 24.6	1.01	1.00–1.02	0.123
Active abduction	135 ± 38.1	143 ± 33.0	1.01	1.00–1.01	0.079
Passive abduction	147 ± 29.6	150 ± 27.8			> 0.2
Active external rotation with arm 90° abducted	82 ± 25.3	82 ± 23.3			> 0.2
Passive external rotation with arm 90° abducted	87 ± 25.9	97 ± 17.8	1.02	1.01–1.04	0.002
Active internal rotation with arm 90° abducted	39 ± 28.6	42 ± 30.0			> 0.2
Passive internal rotation with arm 90° abducted	42 ± 25.8	50 ± 25.5	1.01	1.00–1.03	0.024

\*The values in parenthesis represent percentage.

## DISCUSSION

This study documents that internal impingement of the rotator cuff to the superior glenoid can occur with forward elevation of the shoulder in patients with various occupa-

tions, ages, and diagnoses. This study also documents that this contact is common in a cohort of patients having shoulder surgery, with an overall prevalence of 74%. The overall prevalence of 74% suggests that this contact is a very common phenomenon. In the current study, the

**TABLE 3. Univariate Analysis of Intraarticular Disorders in the Two Groups Divided by the Presence of Internal Impingement in Flexion**

Variables	Descriptive Comparison*		Univariate Analysis		
	Noninternal Impingement in Flexion (n = 99)	Internal Impingement in Flexion (n = 277)	Odds Ratio	Confidence Interval (95%)	p Value
Biceps tendon disorder	25 (25)	61 (22)			> 0.2
SLAP lesions (all lesions)	24 (24)	83 (30)			> 0.2
SLAP I	2 (2)	53 (19)			> 0.2
SLAP II	2 (2)	25 (9)	4.81	1.12–20.70	0.035
SLAP III-IV	1 (1)	5 (2)			> 0.2
Supraspinatus tear (all tears)	53 (52)	129 (47)			> 0.2
Partial thickness supraspinatus tear	27 (27)	82 (30)			> 0.2
Full-thickness supraspinatus tear	25 (25)	47 (17)	0.61	0.35–1.05	0.074
Infraspinatus tear	0 (0)	6 (2)			> 0.2
Subscapularis tendon tear	15 (15)	19 (7)	0.56	0.27–1.17	0.121
Bankart lesion	23 (23)	62 (22)			> 0.2
Hill-Sachs lesion	26 (26)	73 (26)			> 0.2
Humeral head OA	29 (29)	41 (15)	0.42	0.24–0.72	0.002
Glenoid OA	17 (17)	32 (12)	0.63	0.33–1.19	0.157

\*The data are given as the number of subjects, with the percentage in parentheses.

**TABLE 4. Statistical Analysis with the Flexion Degree Making Internal Impingement in Flexion Defined as a Dependent Variable\***

Variable#	
<i>Positive Association</i>	<i>Negative Association</i>
Primary diagnosis of rotator cuff disease (0.000)	Primary diagnosis of glenohumeral instability (0.002)
Increasing age (0.003)	Involvement of dominant arm (0.002)
Positive painful arc sign (0.002)	Positive sulcus sign (higher than Grade I) (0.040)
Type II SLAP lesion (0.050)	Positive apprehension sign (0.006)
Supraspinatus tears (0.000)	Hill-Sachs lesion (0.028)
Humeral head OA (0.042)	Anteroinferior labral fraying (0.009)

\*Positive association means that internal impingement in flexion was made at a lower flexion degree whereas negative association means that internal impingement in flexion was made at a higher flexion degree.

#The values in parenthesis represent p value.

observation of internal impingement in flexion was done through full arc of flexion until internal impingement in flexion occurred. The prevalence of 74% might have included internal impingement in flexion made by an extreme position of flexion, which may be a normal finding. Because we do not have a control group comprised of a normal population without shoulder symptoms, we cannot tell definitely whether this prevalence is abnormally higher than that of the normal population or whether this internal impingement in flexion is pathologic. However, the analysis with the degree of flexion making internal impingement in flexion defined as a dependent variable revealed that patients with rotator cuff disease or Type II SLAP lesions made contact at a lower flexion angle. These findings led us to speculate that internal impingement in flexion may contribute to the pathogenesis of rotator cuff disease or Type II SLAP lesions.

Contact of the rotator cuff to the posterosuperior glenoid was described by Perry in 1983.<sup>38</sup> He noted that this contact could occur during the cocking phase of throwing, but he did not mention whether the contact was physiologic or pathologic or whether it could cause lesions. Walch et al<sup>48</sup> reported their arthroscopic observations of this contact in 17 athletes who had pain on throwing motion. They interpreted that the lesions on the undersurface of the cuff were the result of impingement on the posterior edge of the glenoid when the arm was in the throwing position. They suggested that the impingement might be physiologic, but in throwing athletes the repetition of the contact in the dominant shoulder during sports could lead to mechanical damage and lesions. The relationship between contact of the rotator cuff to the superior glenoid and other disorders is controversial. Some authors have suggested that contact in abduction and external rotation may cause cuff tearing.<sup>20,22,25,48</sup> Others have suggested that this may cause labrum lesions.<sup>3,23</sup>

The role of internal impingement in abduction and external rotation in causing rotator cuff disease or SLAP

lesions has been questioned. Previous studies showed that contact of the rotator cuff to the posterosuperior glenoid in abduction and external rotation was observed in subjects who were asymptomatic or had nonthrowing shoulders.<sup>20,28,31,39</sup> Many patients with contact in abduction and external rotation had never been involved in throwing sports<sup>28,39</sup> and most of the shoulder functions in daily activities are done in forward elevation.<sup>33</sup> Some authors suggested that internal impingement in abduction and external rotation was not a primary cause of SLAP lesions or rotator cuff disease, but rather secondary to instability resulting from superior labrum injury associated with posterior capsule tightness and the peel-back mechanism.<sup>3,10,31</sup>

The findings of the current study may explain the contribution of internal impingement to rotator cuff disease or SLAP lesions in the general population and in the throwing athletes. This impingement also occurs in flexion and in abduction and external rotation. Patients with rotator cuff disease or Type II SLAP lesions had internal impingement in flexion at a lower flexion angle, which may mean that contact is more frequent in daily activities in these patients. Patients who had internal impingement in flexion also had internal impingement in abduction and external rotation at a lower degree of external rotation. These suggest that impingement in flexion and in abduction and external rotation, can be of a repetitive nature even in the nonathletic general population, and this may contribute to the pathogenesis of rotator cuff disease. In this study, patients with rotator cuff disease had internal impingement in flexion at a lower flexion and internal impingement in abduction and external rotation at a lower external rotation. However, the finding that 51 (38%) of the 182 patients with the primary diagnosis of rotator cuff disease did not have internal impingement in flexion suggests that internal impingement in flexion is not the sole causative factor for rotator cuff disease.

The etiology of SLAP lesions is controversial and this study supports the contention that contact of the rotator cuff to the superior glenoid rim may be a contributing factor. Andrews et al<sup>2</sup> suggested that tensile overloading on the biceps long head occurring in the deceleration phase of throwing causes detachment of the biceps anchor. Others<sup>3,23</sup> advocated a compressive load theory of SLAP lesions based on the findings of previous EMG studies, biomechanical studies, and clinical reports.<sup>4,21,49</sup> The findings of the current study indicate an association between internal impingement in flexion and Type II SLAP lesions. Our findings support the theory of compression load<sup>23,40</sup> for the etiology of Type II SLAP lesions rather than the peel-back mechanism<sup>31</sup> or tensile overloading.<sup>2</sup> Decreased internal rotation, suggestive of posterior capsule tightness previously associated with Type II SLAP lesions, was not found in patients in this study.<sup>3,10,31</sup>

There are several limitations of this study. Like some previous studies on this topic, the arthroscopic observation was done with the patients under anesthesia. Contact in patients who are awake and can make muscle activity might have different patterns. Passive flexion may have different results from active flexion. Our flexion maneuver was done with patients in the lateral decubitus position, which may differ from the motion in a standing or sitting position. The joint was insufflated with a pump which might have altered the normal relationships of the glenohumeral joint structures. We did not observe the subacromial space in the flexion maneuver, so we are unable to discuss the relationship of the bursal side of the rotator cuff to the acromion. We think that arthroscopic observation of subacromial impingement with arm flexion can be difficult because the space closes with flexion. The position of the arm was measured with a handheld goniometer and this may not have accurately measured arm position in a draped patient in the lateral position. Investigation of accuracy and reproducibility of the measurement of flexion angle using a goniometer was not done in this study. More detailed three-dimensional evaluations of the relationships of these structures to each other with different shoulder positions are needed. The patient population studied represents the practice of only one surgeon in an academic medical center and these findings may be influenced by the type of patients seen. This study reflected only patients who were symptomatic enough to have surgery. We do not have a percent of the general population with this pathologic finding. The use of patients with symptoms as a control group may have influenced the statistical analysis, but it would have been ethically and practically impossible to do arthroscopy on the shoulders of asymptomatic patients as normal controls.

We found that internal impingement of the rotator cuff to the superior glenoid in flexion is common in this cohort

of patients having shoulder surgery. Type II SLAP lesions were associated with the presence of internal impingement in flexion. The patients with a primary diagnosis of rotator cuff disease and Type II SLAP lesions had internal impingement in flexion at a significantly lower degree of flexion. The patients who had internal impingement in flexion had internal impingement at a lower degree of external rotation when the shoulder was abducted. These findings lead us to speculate that the patients with rotator cuff disease and Type II SLAP lesions might have had internal impingement in flexion or abduction and external rotation more repetitively than the patients without the diseases. This study provides the first statistical evidence that internal impingement in flexion may be an etiologic factor of rotator cuff disease and Type II SLAP lesions in nonathletes and in throwing athletes.

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