Anterosuperior impingement of the shoulder as a result of pulley lesions: A prospective arthroscopic study

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Lesions of the biceps pulley and the rotator cuff have been reported to be associated with an internal anterosuperior impingement (ASI) of the shoulder. The purpose of this study was to determine the factors influencing the development of an ASI. Eighty-nine patients with an arthroscopically diagnosed pulley lesion were prospectively included in this study. Four patterns of intraarticular lesions could be identified. Twenty-six patients (group 1) showed an isolated lesion of the superior glenohumeral ligament (SGHL). In 21 patients (group 2) an SGHL lesion and a partial articular-side supraspinatus tendon tear were found. Twenty-two patients (group 3) had an SGHL lesion and a deep surface tear of the subscapularis tendon, and in twenty patients (group 4) a lesion of the SGHL combined with a partial articular-side supraspinatus and subscapularis tendon tear was diagnosed. Of the patients, 80 (89.9%) showed involvement of the long head of the biceps tendon including synovitis, subluxation, dislocation, and partial or complete tearing. In 43.8% of all patients, ASI was observed. Whereas ASI was seen in 26.6% and 19.1% of patients in groups 1 and 2, respectively, 59.1% of patients in group 3 and 75% of patients in group 4 were found to have an ASI. ASI was significantly more often seen in patients with additional partial articular-side subscapularis tendon tears ($P < .0001$). In patients with acromioclavicular (AC) arthritis, ASI (62.5%) was more frequently observed than in patients without AC arthritis ($P = .0309$). In the multivariate analysis the stepwise selection procedure revealed only AC arthritis and the deep surface tear of the subscapularis (groups 3 and 4) to be significant influencing factors for an ASI. Our findings indicate that a progressive lesion of the pulley system, including partial tears of the subscapularis and supraspinatus tendons, contributes significantly to the development of an ASI. A pulley lesion leads to instability of the long head of the biceps tendon, causing increased passive anterior translation and upward migration of the humeral head, resulting in an ASI. In addition, a partial articular-side subscapularis and supraspinatus tendon tear reinforces the ASI. (J Shoulder Elbow Surg 2004;13:5-12.)

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1058-2746/2004/$35.00 + 0
doi:10.1016/j.jse.2003.09.013

Anterosuperior impingement (ASI) was first described by Gerber and Sebesta[2] as a form of intraarticular impingement responsible for painful structural disease of the shoulder. In a position of horizontal adduction and internal rotation of the arm, the undersurface of the reflection pulley and of the subscapularis tendon impinges against the anterosuperior glenoid rim. This appears to be a possible cause of pulley lesions and subscapularis tendon lesions.

The pulley system, a tendoligamentous sling, represents an important part of the rotator interval, which consists of four major structures. These four structures are the coracohumeral ligament (CHL), the superior glenohumeral ligament (SGHL), fibers of the supraspinatus tendon, and fibers of the subscapularis tendon. The CHL arises from the coracoid process as a broad but thin ligament. The CHL divides into two major bands; one inserts into the tendinous anterior edge of the supraspinatus and the greater tuberosity, and the other inserts into the superior border of the subscapularis, the transverse humeral ligament, and the lesser tuberosity.[4] The SGHL originates from the anterosuperior labrum adjacent to the supraglenoid tubercle and crosses the floor of the rotator interval. In the medial aspect of the ligament, it forms a fold parallel to the long head of the biceps tendon (LHB). In the lateral region, it changes into a U-shaped sling crossing under the biceps tendon before it inserts into the proximal aspect of the lesser tuberosity just above the insertion site of the subscapularis tendon.[22] At the entrance of the bicipital groove, the SGHL and the CHL blend together, forming the reflection pulley[19] (Figure 1). Histologically, the orientation of the fibers

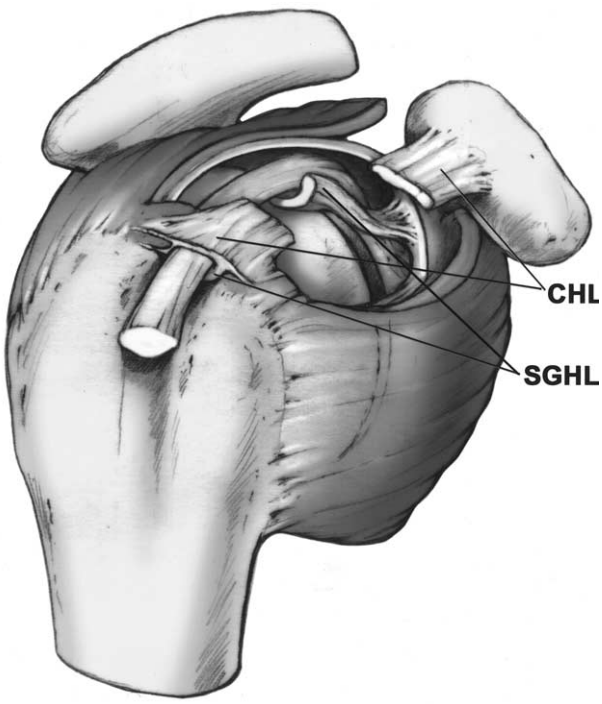

Figure 1 Anatomy of rotator interval. The CHL and SGHL blend together, forming the reflection pulley, which encloses the LHB at the entrance of the intertubercular groove.[22]

of the SGHL seems to withstand anterior shearing forces in the area proximal to the bicipital groove, indicating that the most important function of the SGHL is the stabilization of the LHB in its intraarticular course.[22]

The rotator cuff reinforces this stabilizing system by fiber bundles that arise from its transverse band (fasciculus obliquus). These fibers form the roof of the rotator interval capsule together with the CHL and SGHL.[22] In the posterior aspect of the roof, fibers from the supraspinatus intermingle with the ligamentous structures. The floor of this system is made up of fibers derived from the posterior aspect of the subscapularis. These fibers again blend together with parts of the SGHL and CHL at the entrance to the groove.[22]

A lesion of the pulley system can be caused by trauma or degenerative change. A fall on the outstretched arm in combination with full external or internal rotation, as well as a fall backward on the hand or elbow, can cause a pulley lesion.[7] Furthermore, a forcefully stopped overhead throwing motion may terminate in a pulley lesion. During active contraction of the biceps in internal rotation, the strain increases while elbow extension is decelerated. By this deceleration, a maximal contraction of the LHB is provoked and can cause tears in the rotator interval capsule. Gerber and Sebesta[2] described the pulley lesion resulting from repetitive forceful internal rotation above the horizontal plane. This causes friction damage between the pulley system and subscapularis on the one hand and the anterior superior glenoid rim on the other.

As a result of intrinsic degenerative changes, the rotator interval shows partial tears involving the SGHL and the articular side of the supraspinatus. Partial tears of the supraspinatus often commence at the lateral aspect of the bicipital groove and, therefore, also cause lesions of the pulley system by involving the fasciculus obliquus.

Tears of the SGHL lead to instability of the LHB in its intraarticular course and to subluxation of the LHB medially, causing partial articular-side tears of the subscapularis tendon and lesions of the LHB itself. In 1996 Habermeyer and Walch[3] showed that 50% of all biceps subluxations were associated with degenerative changes in the anterosuperior aspect of the labrum, suggesting a correlation between these intrinsic structural changes.

Lesions of the LHB, the reflection pulley, and the rotator cuff have been associated with an internal ASI of the shoulder.[2,17] The purpose of this study was to determine the factors influencing the development of an ASI.

Table I Clinical and arthroscopic findings

Group	PL	PL and SSP	PL and SSC	PL, SSP and SSC
No. of patients	26	21	22	20
Age (y)	46.4	47.5	50.7	55.4
Traumatic	9 (34.6%)	4 (19.1%)	6 (27.3%)	3 (15%)
Atraumatic	17 (65.4%)	17 (80.9%)	16 (72.7%)	17 (85%)
ASI	7 (26.9%)	4 (19.1%)	13 (59.1%)	15 (75%)
Positive O'Brien sign	18 (69.2%)	15 (71.5%)	14 (63.6%)	12 (60%)
Positive palm-up test	20 (76.9%)	11 (52.4%)	16 (72.7%)	12 (60%)
Positive Hawkins test	13 (50%)	9 (42.9%)	13 (59.1%)	12 (60%)
Subacromial fraying	13 (50%)	14 (66.7%)	16 (72.2%)	16 (80%)
AC arthritis	8 (30.8%)	6 (28.6%)	6 (27.3%)	4 (20%)

PL, Pulley lesion; *SSP*, partial articular-side suprasinatus tendon tear; SSC, partial articular-side subscapularis tendon tear.

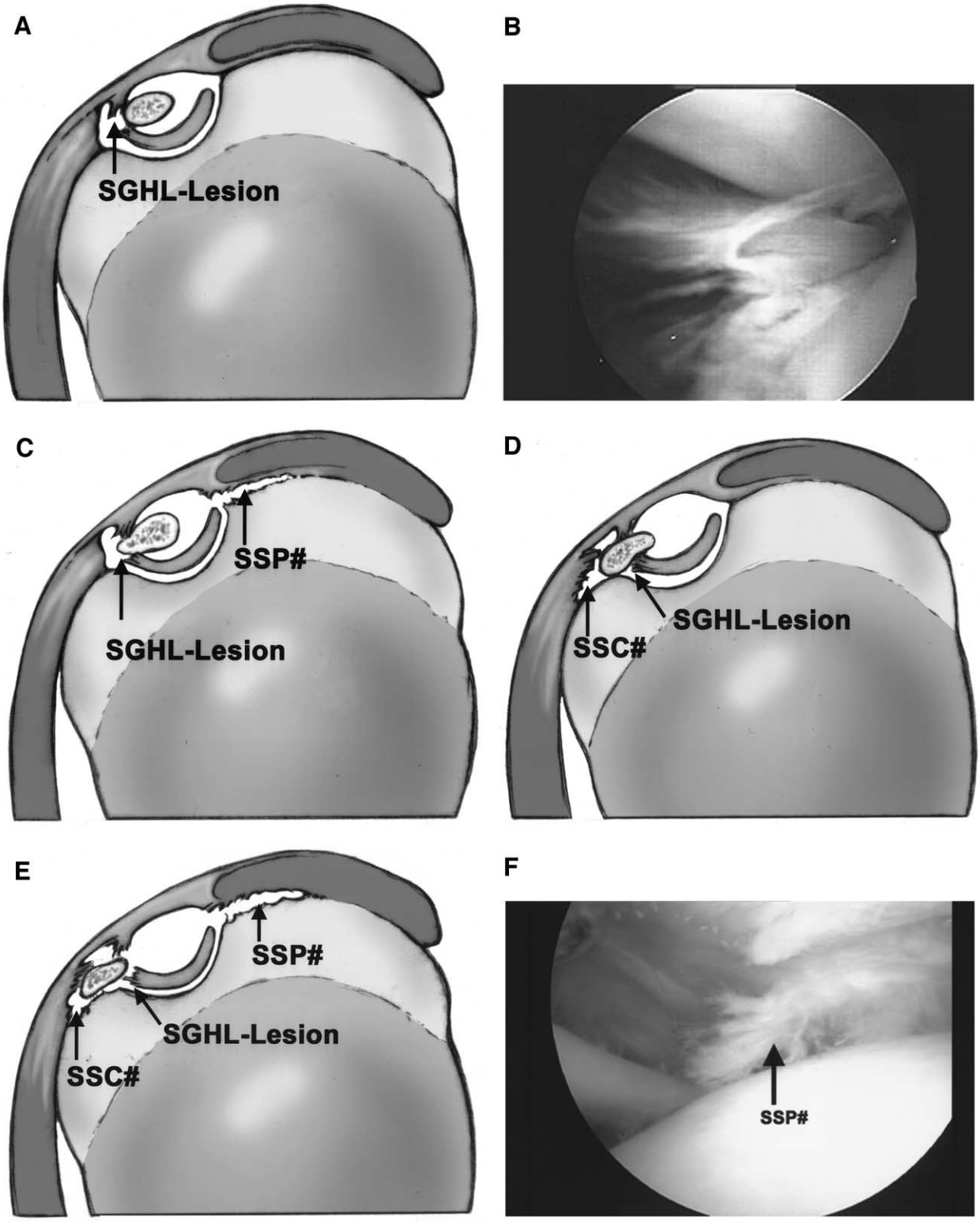

Figure 2 A, Group 1: SGHL lesion only. The SGHL lesion leads to instability of the LHB during internal rotation. **B**, Arthroscopic view of a group 1 lesion. **C**, Group 2: SGHL lesion and partial articular-side supraspinatus tendon tear (SSP#). **D**, Group 3: SGHL lesion and partial articular-side subscapularis tendon tear (SSC#). The medially subluxated LHB leads to a deep surface tear of the subscapularis tendon. **E**, Group 4: SGHL lesion with partial articular-side supraspinatus (SSP#) and subscapularis tendon tear (SSC#). **F** .

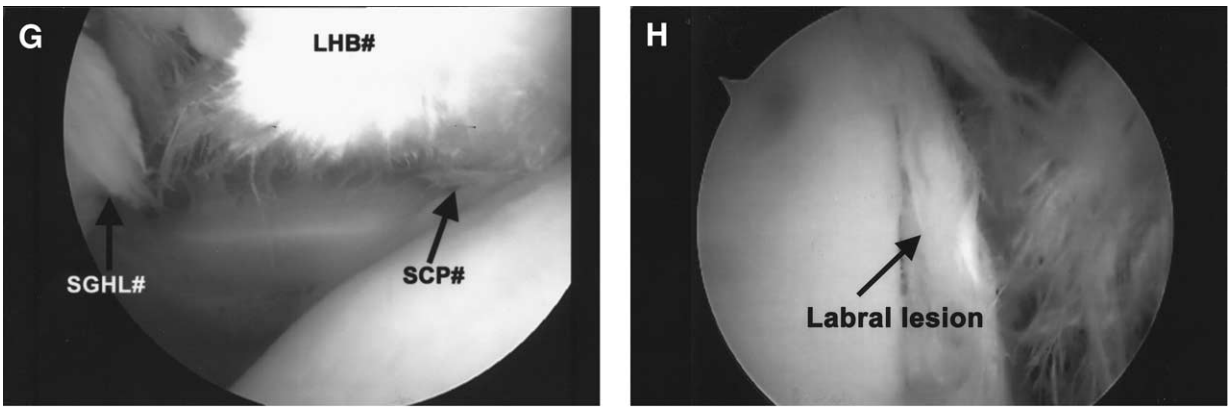

Figure 2 (Continued) **G**, Arthroscopic view of group IV lesion. **H**, Anterosuperior labral lesion resulting from ASI.

MATERIAL AND METHODS

Since 1998, 89 consecutive patients (23 women and 66 men) with a mean age of 47.7 years (range, 16-77 years) and an arthroscopically verified pulley lesion were prospectively included in this study. Twenty-two patients had a history of trauma, whereas sixty-seven had none. Clinical examination showed a positive impingement sign[5] in 47 patients, a positive O'Brien test[11] in 59, and a positive palm-up test in 59. One of the tests was positive in 67 patients (75.3%), and both tests were positive in 51 (57.3%). None of the patients had any signs of instability or laxity, and all had a full range of motion.

All patients underwent standardized radiographic examination in three planes (true anteroposterior, outlet, and axial views); this revealed osteoarthritic changes of the acromioclavicular joint in 24 patients (27%). No signs of glenohumeral osteoarthritis were observed.

For the standardized arthroscopic examination, all patients were placed in the beach-chair position. Examination under anesthesia showed neither instability nor laxity in any patient (Table I).

Arthroscopic technique

For diagnostic arthroscopy, the scope was introduced through a standard posterior portal. The glenohumeral inspection started from the LHB origin. Its intraarticular and intertubercular course was assessed with a probe. After the examination of the LHB, the articular side of the supraspinatus tendon and the articular side of the subscapularis tendon were inspected. (A complete tear of the supraspinatus or subscapularis tendon was an exclusion criterion.) The anterosuperior glenoid rim and labral structures were examined. Dynamic testing of the impingement of the subscapularis and lesser tuberosity against the anterosuperior glenoid rim and labrum was carried out under arthroscopy during flexion, horizontal adduction, and internal rotation. Patients showing an anterosuperior labral lesion together with positive dynamic testing of impingement of the subscapularis and the lesser tuberosity against the anterosuperior glenoid rim and labrum were diagnosed as having ASI.

The subacromial space was arthroscopically evaluated through the posterior portal. All arthroscopic findings were documented by use of a standardized intraoperative shoulder documentation sheet.

Statistical methods

Univariate analyses of an association between ASI and the factors described above were carried out by Pearson χ^2 test for categorical data and Mann-Whitney *U* test for age. The combined influence of the different factors was investigated by multiple logistic regression. A stepwise selection procedure described by Lemeshow and Hosmer[8] was used to select independent factors for the presence of an ASI. All statistical analyses were performed with SAS, version 8.0 for Windows (SAS Institute Inc, Cary, NC).

RESULTS

In all 89 patients selected according to our study inclusion criteria (100%), a lesion of the pulley system was confirmed at arthroscopy. Four different patterns of intraarticular lesions could be identified (Figure 2) (Table II).

ASI was observed in 39 of 89 patients (43.8%). In group 1 (isolated SGHL lesion) ASI occurred in 26.9%, and in group 2 (SGHL lesion and partial articular-side lesion of the supraspinatus tendon) ASI occurred in 19.1%. The combination of an SGHL lesion and a deep surface tear of the subscapularis tendon (group 3) or this combination plus a partial tear of the supraspinatus tendon (group 4) increased the percentage of ASI to 59.1% (group 3) and 75% (group 4) (Figure 3).

Overall, involvement of the LHB was demonstrated in 80 patients (89.9%). There were 5 complete tears, 13 partial tears with concomitant subluxation, 29 subluxations, and 33 cases of synovitis of the LHB. Superior labrum anterior-posterior (SLAP) II and higher graded SLAP lesions[16] were not observed in any patient. Lesions of the LHB were found in 22 of 26 patients in group 1 (84.6%), in 18 of 21 patients in

Table II Classification of intraarticular lesions

	Classification of intraarticular lesion	No. of patients	No. of ASI
Group 1	SGHL lesion	26 (29.2%)	7 (26.9%)
Group 2	SGHL lesion + SSP	21 (23.6%)	4 (19.1%)
Group 3	SGHL lesion + SSC	22 (24.7%)	13 (59.1%)
Group 4	SGHL lesion + SSP + SSC	20 (22.5%)	15 (75%)

SSP, Partial articular-side supraspinatus tendon tear; *SSC*, partial articular-side subscapularis tendon tear.

group 2 (85.7%), in all patients in group 3, and in 18 of 20 patients in group 4 (90%).

Patients with ASI were slightly older (mean age, 51.6 years; range, 16-77 years) than those without ASI (mean age, 48.3 years; range, 29-77 years). ASI was slightly more frequently observed in men (45.5%) than in women (39.1%) ($P = .19$). Of the patients with ASI, 89.7% (35/39) had a degenerative history.

Clinical examination revealed a positive impingement test in 66.7% of patients with ASI and 42% without ASI. Of patients with ASI, 79.5% had a positive palm-up test or a positive O'Brien test. Both tests were positive in 58.9% of patients with ASI.

ASI was seen more often in patients with an additional partial articular-side subscapularis tendon tear. This was statistically significant ($P < .0001$).

Diagnostic arthroscopy of the subacromial space showed no bursal-side rotator cuff tear in any patient. In 59 of 89 cases (66.3%) subacromial fraying indicating subacromial impingement was observed.

Acromioclavicular (AC) arthritis was diagnosed by clinical examination and by standard radiographs in 24 patients (27%).

The number of patients with ASI in the above-defined subgroups and in relation to radiologically diagnosed AC arthritis or arthroscopically discovered subacromial fraying is shown in Table III. ASI was significantly more often observed in patients with AC arthritis (62.5%) than in patients without AC arthritis (36.9%) ($P = .0309$). No significant difference could be found between patients with and without subacromial fraying. In the multivariate analysis the stepwise selection procedure revealed only AC arthritis and the differently defined groups to be significant influencing factors for ASI. No interactions were included in the model.

The results of the logistic regression analysis are presented in Table IV. The odds ratio of patients in group 3 compared with group 1 with an isolated SHGL lesion was 4.99, showing the significant impact ($P = .0171$) of an isolated partial articular-side tear of the subscapularis tendon on the development of ASI. In contrast, the isolated partial articular-side tear of the supraspinatus tendon (group 2) did not have a significant influence. However, patients with a partial articular-side tear of both the supraspinatus and subscapularis tendons (group 4) had an odds ratio of 12.0 compared with group 1.

In summary, ASI as defined above occurred significantly more often in patients with concomitant deep surface partial subscapularis tendon tears (group 3) and increased in number with additional partial articular-side supraspinatus tendon tears (group 4). In addition, it has to be underlined that AC arthritis was observed significantly more often in patients with ASI than in those without ASI, suggesting a significant impact on the development of ASI.

DISCUSSION

Different forms of internal impingement of the glenohumeral joint have been described over the last decade.[1,10,17] ASI represents a new clinical entity responsible for unexplained anterior shoulder pain.[2]

In a cadaveric study Valadie et al[18] demonstrated contact between the articular surface of rotator cuff tendons and the anterosuperior glenoid with the arm placed in the Hawkins position. Struhl[17] suggested that this contact between the rotator cuff and the superior labrum is physiologic. In cases of partial rotator cuff defects the contact may become pathologic as the fragmented tissue is sheared and compressed between the superior humeral head and the glenoid.

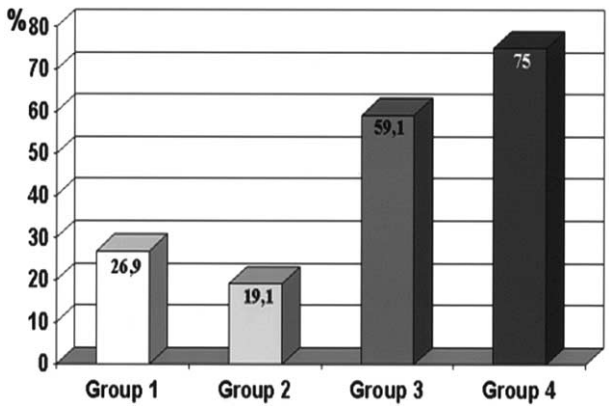

Figure 3 Relative frequency of anterosuperior labral lesion (ASI) in the 4 groups.

Table III Frequencies of patients with ASI in the 4 groups, in those with subacromial fraying, and in those with AC arthritis

	No.	ASI	No ASI	*P* value (χ^2 test)
Group 1	26	7 (26.9%)	19 (73.1%)	
Group 2	21	4 (19.1%)	17 (80.9%)	
Group 3	22	13 (59.1%)	9 (40.9%)	
Group 4	20	15 (75.0%)	5 (25.0%)	.0004
Subacromial fraying	59	28 (47.5%)	31 (52.5%)	
No subacromial fraying	30	11 (36.7%)	19 (63.3%)	.3321
AC arthritis	24	15 (62.5%)	9 (37.5%)	
No AC arthritis	65	24 (36.9%)	41 (63.1%)	.0309

Table IV Results of logistic regression analysis

Parameter	Odds ratio	95% Wald confidence interval	*P* value
Group 2*	0.63	0.14-2.75	.5399
Group 3*	4.99	1.33-18.74	.0171
Group 4*	12.02	2.82-51.27	.0008
AC arthritis	4.98	1.54-18.08	.0073

*Compared with group 1.

Gerber and Sebesta[2] defined the ASI of the deep surface of the subscapularis tendon as a form of intraarticular impingement responsible for painful structural disease of the shoulder. They evaluated 16 patients with a mean age of 45 years by physical examination, arthro–magnetic resonance imaging, and diagnostic arthroscopy. All patients had pain on a modified impingement test that could not be relieved by subacromial infiltration of lidocaine. In all patients the internal ASI of the pulley system and the subscapularis tendon against the anterosuperior glenoid rim with the arm brought into horizontal adduction, internal rotation, and various degrees of anterior elevation was observed. Of the patients, 3 (18.8%) had an isolated pulley lesion. In 10 cases (62.5%) there was a pulley lesion associated with a partial articular-side subscapularis tendon tear, and in 3 cases (18.8%) an isolated partial articular-side subscapularis tendon tear was found. Overall, 81.3% of their patients with an ASI had a partial articular-side subscapularis tendon tear. Of their 16 patients, 12 (75%) were engaged in manual labor involving regular overhead activity. Therefore the authors concluded that because of repetitive, forceful internal rotation maneuvers above the horizontal plane, friction damage results from impingement between the pulley system and subscapularis on the one hand and the anterior superior glenoid rim on the other, leading to the above-mentioned pathologic lesions.

Our study shows that the anterosuperior labral lesion seems to be a result of an impingement of the LHB and the subscapularis tendon with the anterosuperior glenoid rim. An important factor for the development of ASI is the additional partial articular-side lesion of the subscapularis tendon in the presence of a pulley lesion. A pulley lesion in combination with a partial articular-side supraspinatus tendon tear without a partial articular-side subscapularis tendon lesion does not lead to ASI. Contrary to this, the combination of a partial articular-side subscapularis and supraspinatus tendon tear (defined in our logistic regression model as a single character) in addition to the pulley lesion increases the risk of the incidence of ASI. Age and gender were not influencing factors for the development of the ASI.

Our study showed that patients with ASI (mean age, 51.6 years) were slightly, but not significantly, older than patients without ASI (mean age, 48.3 years). The age distribution was comparable to other studies with a mean patient age of 45.3 years.[2] In contrast to the initial study by Gerber and Sebestas,[2] none of our patients performed regular overhead activity during daily work or in sports.

Lesions of the pulley system, the LHB, and the supraspinatus tendon, as well as the subscapularis, are commonly associated.[14,15,19,21] The pulley lesion can be caused by trauma or degenerative changes.[2,7] After the reflection pulley is torn, the LHB becomes unstable in its intraarticular course, leading to medial subluxation and alteration of the LHB itself. Physiologically, the LHB is thought to be an anterior stabilizer of the glenohumeral joint during rotation of the arm.[13] The subluxated LHB loses its anterior stabilizing effect on the glenohumeral joint and allows anterior humeral translation.[12] The subluxated LHB with concomitant decentralization of the humeral head seems to be the initial event of a progressive cascade resulting in ASI. Sakurai et al[14] found a statistically significant correlation between tears of the subscapularis tendon and the presence of lesions of the LHB and found most pathologic changes of the subscapularis tendon on the articular side of the up-

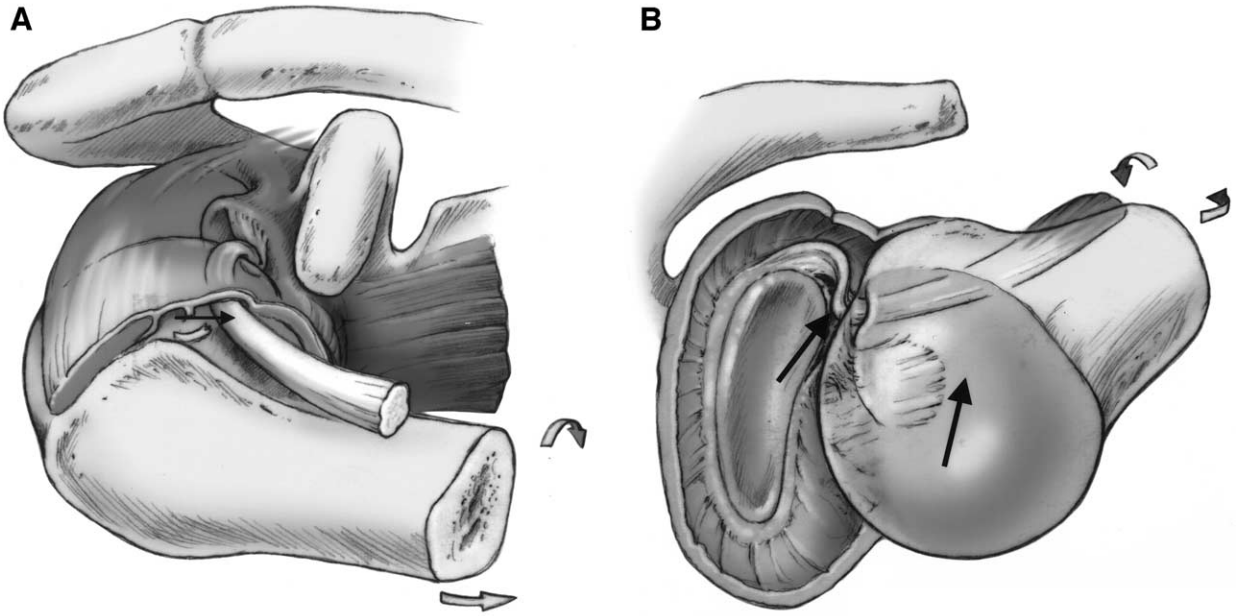

Figure 4 A, Pathomechanism of ASI. **B**, Pathomechanism of ASI. During internal rotation and adduction in the horizontal plane, the torn pulley system leads to medial subluxation of the LHB, causing a deep surface tear of the subscapularis tendon. Because of the unstable medially subluxated LHB, the posterior and compressive joint retraction mechanism of the LHB is lost. As a result, the humeral head migrates into an anterosuperior position against the glenoid rim, causing ASI.

per portion. The medially subluxated LHB has been shown to lead to partial articular-side tears of the subscapularis tendon.[20] Tears of the articular side of the subscapularis tendon permit further anterior-superior translation of the humeral head, resulting in ASI. In 71.8% of our patients with ASI, a partial articular-side subscapularis tendon tear was observed, and logistic regression analysis verified the important influence of the partial articular-side subscapularis tendon tear in ASI. An additional articular-side supraspinatus tendon tear reinforces this pathomechanism.

Our study confirmed that intraarticular changes have a significant influence on the development of an ASI. The subacromial findings in this study revealed no significant difference in 47.5% of patients with subacromial fraying and 36.7% without subacromial fraying in combination with ASI, suggesting that subacromial fraying does not appear to be an influencing factor for the development of an ASI. Snyder et al[16] reported subacromial pathologic findings in 58.1% of patients with partial rotator cuff tears.

Of the patients in our study group, 24 (27%) had signs of AC arthritis. Gerber and Sebesta[2] found AC arthritis in 43.8% of their patients with an ASI. In our series 15 of 24 patients (62.5%) diagnosed with AC arthritis had an ASI. Multivariate analysis revealed AC arthritis to be a determining factor for ASI. The pathophysiologic association between AC arthritis and the development of an ASI is unclear. Matsen et al[9] stated that symptomatic AC arthritis is often associated with a posterior capsular pattern. In cases of tightness of the posterior capsule, an anterior-superior translation of the humeral head may be produced with shoulder flexion reinforcing the above-described pathomechanism for ASI. Nevertheless, the observed statistical association between AC arthritis and the development of ASI requires further study.

We conclude that a lesion of the pulley system that leads to instability of the LHB causes a partial articular-side subscapularis tendon tear. The medially displaced LHB results in increasing anterior translation of the humeral head. In addition, the LHB can passively control the upward migration of the humeral head.[8] Thus, in the case of a medially subluxated LHB, increased passive anterior translation and upward migration of the humeral head may occur. Subluxation of the LHB results in a process that reinforces the ASI (Figure 4). The results of multiple logistic regression analysis imply a progressive character of lesions of the pulley system, the LHB, and the rotator cuff. The more lesions, the higher the frequency of ASI.

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