Appendix

Exclusion Criteria, Arthroscopic Surgical Technique and Findings, Postoperative Rehabilitation, and Outcomes Assessment

Exclusion Criteria

Exclusion criteria were (1) acute arthroscopic Bankart repair and capsular shift for first-time anterior shoulder instability (102 patients); (2) previous failed arthroscopic stabilization (seventeen patients); (3) concomitant rotator cuff tear (nine patients); (4) clinical evidence of bidirectional, multidirectional, or voluntary instability (twenty-two patients); (5) a history of epilepsy or cognitive impairment (twelve patients); (6) hyperlaxity (sixteen patients) with instability developing in the absence of a clinical history of external trauma to the shoulder (patients with hyperlaxity and a history of trauma were included if they had a Bankart lesion); (7) no Bankart lesion at surgery, or unusual soft-tissue lesions contributing to instability such as a humeral avulsion of the glenohumeral ligaments (six patients); and (8) bilateral shoulder instability, as it was thought that the natural history might be altered by the activity modification and treatment required following the first stabilization procedure (seventeen patients)

Arthroscopic Surgical Technique

The patient was placed in the beach-chair position, and the affected upper extremity was prepared. Examination was performed with the patient under anesthesia to determine the direction and severity of the instability. A posterior viewing portal was created inferomedial to the posterolateral corner of the acromion, and the anterosuperior instrumentation portal was created lateral to the coracoid process, through the rotator cuff interval. A complete arthroscopic evaluation of the shoulder was performed to assess the soft-tissue and osseous lesions associated with instability as well as any other pathology.

To accomplish the repair of the Bankart lesion, the labral detachment was completed to the six o'clock position (of the right shoulder), with use of a Bankart rasp and electrocautery. The anterior aspect of the glenoid neck was then decorticated with use of a motorized shaver and burr to create a cancellous bed to encourage soft-tissue healing. Three, four, or five holes were drilled from the eleven o'clock to the five o'clock position (of the right glenoid), depending on the size of the detachment of the capsulolabral complex. The drill-holes were placed at the margin of the glenoid articular surface to allow recreation of the glenoid concavity. With use of the single anterior portal, a suture passer (Linvatec, Largo, Florida) was used to deliver a PDS suture (polydioxanone; DePuy Mitek EMEA, Johnson & Johnson Medical, Rome) through the detached capsulolabral complex to allow superior shift of the capsule. The extent of the capsular shift was such that the suture, once tightened to its native anchor, would retension the capsule and obliterate the inferior capsular recess with the shoulder placed in 10° of forward flexion, 45° of abduction, and 30° of external rotation. A Panalok absorbable anchor (Mitek, Johnson & Johnson) was placed onto the limb of the suture on the glenoid side and was then inserted into the most superior drill-hole close to the glenoid articular margin. The arthroscopic core suture was then tied on the capsulolabral side, to keep the knot away from the articular surface. The same maneuver was

subsequently performed to pass the other anchors and sutures, proceeding in a superior-to-inferior direction. Tensioning of the repair could be adjusted in this manner if required. When the anteroinferior Bankart lesion was associated with a superior labral anteroposterior detachment (a SLAP lesion), the latter was repaired first, to anatomically reduce and stabilize the superior pole of the labrum.

Additional instrumentation for suture management and anchor placement was carried out percutaneously, without formal arthroscopic portal incisions. When the detachment was predominantly anteroinferior, three anchors were used, whereas when there was a SLAP lesion in continuity, one or two additional anchors were placed in the superior quadrant of the glenoid. The average number of anchors used was 4.8 (range, three to five). Associated osseous Bankart lesions were repaired by incorporating them into the soft-tissue capsulolabral repair. Sutures were placed through the capsulolabral sleeve superior and inferior to the osseous lesion, and the corresponding anchors were inserted into the area superior and inferior to the area of osseous detachment from the glenoid rim. Tying of these sutures produced approximation of the osseous lesion to the area of detachment. Associated glenoid bone loss and posterior humeral head defects (so-called Hill-Sachs lesions) were not addressed by any supplementary arthroscopic procedures during the course of this study.

All of the operative procedures were recorded in real time and anonymously stored as digital files. The intraoperative parameters depicted in Table E1-B were blindly assessed at the conclusion of the study by one of us (C.M.R.) postoperatively. The extent of the Bankart lesion was recorded on the basis of a clock-face model with 12 o'clock being superior and 6 o'clock, inferior. The tear was graded on the basis of whether it involved just the anteroinferior portion, whether it involved an associated SLAP lesion, or whether there was an associated anterior labroligamentous periosteal sleeve avulsion (ALPSA). An anteroinferior Bankart lesion was encountered in 241 patients; forty of these patients had an associated osseous Bankart lesion, three had a panlabral detachment, twenty had an associated SLAP lesion in continuity, and thirty-eight had an anteroinferior ALPSA lesion. Seven patients had an articular-sided deep-surface rotator cuff lesion, and twenty-seven patients had a chondral split defect at the 3 o'clock position of the glenoid. The amount of glenoid bone loss was quantified with use of a validated methodology based on the glenoid bare spot, which is the center of the inferior glenoid circle[3]. The percentage of glenoid bone loss was graded as 0% to 5%, 6% to 24%, or ≥25%. The volumetric measurements of the Hill-Sachs defects was estimated on magnetic resonance imaging and graded as I (< 1.5 cm^2), II (1.5 to 2.5 cm^2), or III (>2.5 cm^2). We defined an engaging Hill-Sachs lesion as a posterior humeral defect that could be seen arthroscopically as engaging the anterior rim of the glenoid when the arm was brought into a position of 90° of abduction combined with 90° of external rotation[3].

Postoperative Rehabilitation

All patients followed the same rehabilitation program, with the shoulder in an immobilizer for six weeks. Active-assisted range-of-motion exercises were begun at three weeks postoperatively, with the patient avoiding abduction of the shoulder beyond 90° and external rotation beyond neutral. Isometric rotator cuff exercises and graduated active range-of-motion exercises, performed under the supervision of a physiotherapist,

commenced after removal of the sling and continued for at least three months. Patients were advised to avoid contact sports and high-risk activities for six months.

Outcomes Assessment

At six months, one year, two years, and annually thereafter, the patients were interviewed by a research assistant not involved in the study analysis. They were asked whether they had sustained additional dislocations, had symptoms of recurrent anterior instability, or had received additional treatment in another center. All patients were asked to complete a questionnaire incorporating the Disabilities of the Arm, Shoulder and Hand (DASH) questionnaire[24] and the Western Ontario Shoulder Instability Index (WOSI)[25] at each visit up to the two-year assessment.

The patients were also examined by the senior author for signs of recurrent instability at each follow-up visit. We defined postoperative recurrence as either an additional radiographically confirmed anterior dislocation or symptoms of recurrent anterior subluxation accompanied by positive findings on both an anterior apprehension test[26] and an anterior load-and-shift test[27].

After the final two-year functional assessment, we sought to continue annual follow-up. If patients did not return as planned, we used a telephone interview or postal questionnaire to attempt to re-establish contact. The number of patients lost at two years was thirty-three. Another four patients were lost to follow-up at the end of the second year and another seventy-eight, by the end of the fifth year. Therefore, a total of thirty-three patients (10.9%) were lost to follow-up at two years and a total of 115 patients (38%) were lost at five years (Table E5).

Statistical Analysis

Since the development of recurrent instability was a time-dependent outcome variable and the duration of patient follow-up was variable, we used survival methodology to examine the probability of recurrent instability occurring after stabilization. For the patients who had recurrent instability, that outcome was considered to be present when the shoulder redislocated for the first time or when the patient first developed symptoms of recurrent subluxation. Patients who did not have recurrent instability were censored from further analysis when they were lost to follow-up.

Cox proportional hazards models were used to estimate the effects of patient and injury-related factors on the time to recurrence, and product functions were used to test for synergistic effects via interaction terms in the regression. All variables that were predictive of recurrence at $p < 0.10$ were included in multivariate models (with use of forward conditional methodology) to determine the factors that were independently predictive. The lax p value of 0.1 was used because variables that just fail to reach significance with use of a lower cutoff may achieve significance when adjusted for other variables in the multivariate analysis. The goodness of fit of the model was assessed with use of the p value of the chi-square of the log likelihood ratio test. A prognostic index (PI) was calculated for the three factors that remained in the final model from the summated products of the regression coefficients and the factor levels. The predicted probability (S_t) of recurrence at any given time (t) was calculated from the expression: $S_t = S_0(t)\exp(PI)$, where $S_0(t)$ is the baseline cumulated survival function at time t.

The percentage probability of recurrence within two years was then calculated for each patient with use of the above formula. From this, we were able to divide patients into two groups, those above and those below different chosen cutoff levels for the predicted probability of recurrence within two years. This enabled us to produce classification tables of predicted and actual outcomes to estimate the sensitivity, specificity, positive predictive value, and negative predictive value of the model at different cutoff levels for the predicted probability of recurrence within two years. A receiver operating characteristic curve (Fig. 2) was constructed to determine the accuracy of the model by demonstrating the limits of the model's ability to predict whether a patient had a stable or unstable repair. The performance of the model was quantified by calculating the area under the curve. An area under the curve of 1 demonstrates an ideal test with 100% sensitivity and specificity, while an area under the curve of <0.5 indicates that the test is less useful. We used Wilcoxon nonparametric estimates to determine the 95% confidence intervals (95% CI) for the area under the curve. The receiver operating characteristic curve correlates the true-positive and false-positive rates for a series of data points. The optimum diagnostic cutoff level for the model was chosen as the values that corresponded to the points on the receiver operating characteristic curve nearest the upper left-hand corner of the graph. This cutoff level reflects the optimal mathematical balance between sensitivity and specificity. However, it may not reflect the optimal cutoff level for a particular patient in the clinical setting.

Paired-sample t tests were used to compare the preoperative and postoperative functional outcome (WOSI and DASH) scores. Using linear regression analysis, we tested each patient-related and injury-related parameter along with the recurrence status against each of these two functional outcome measures.

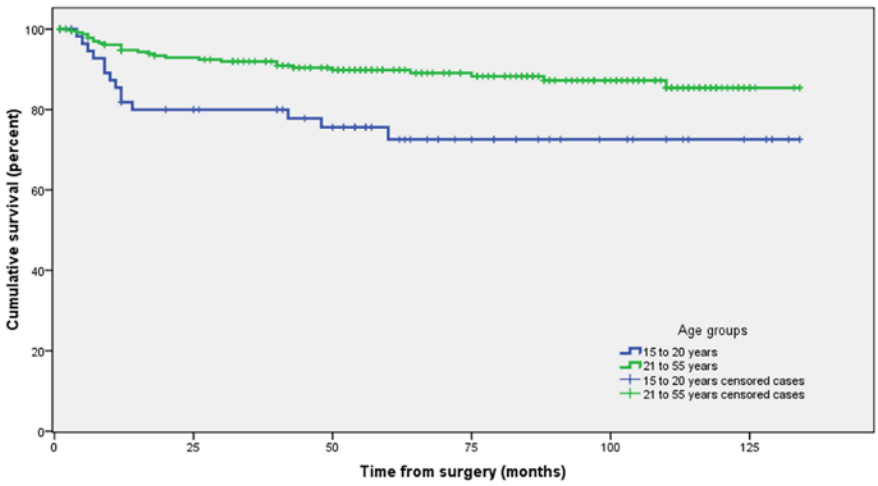

Fig. E1-A

Kaplan-Meier survivorship curves for two age groups: fifteen to twenty years and twenty-one to fifty-five years.

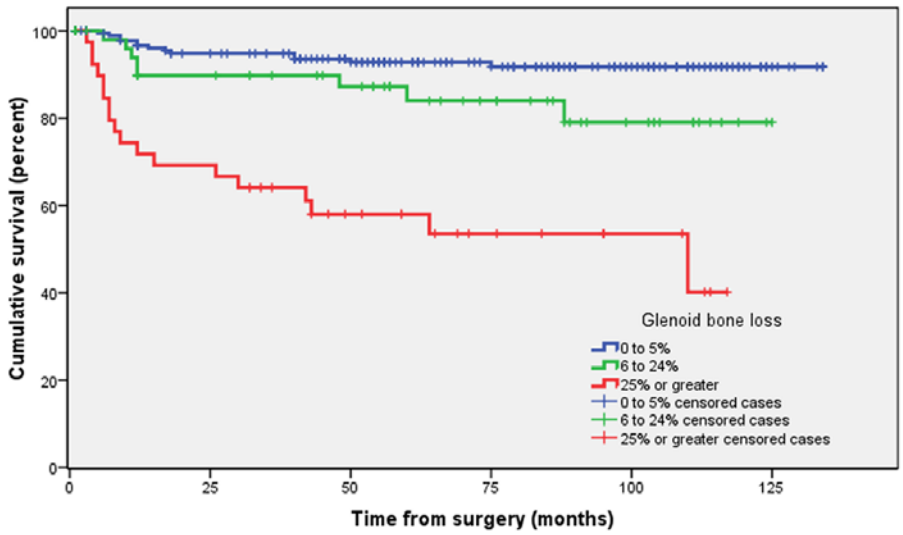

Fig. E1-B

Kaplan-Meier survivorship curves for groups based on the percentage of glenoid bone loss.

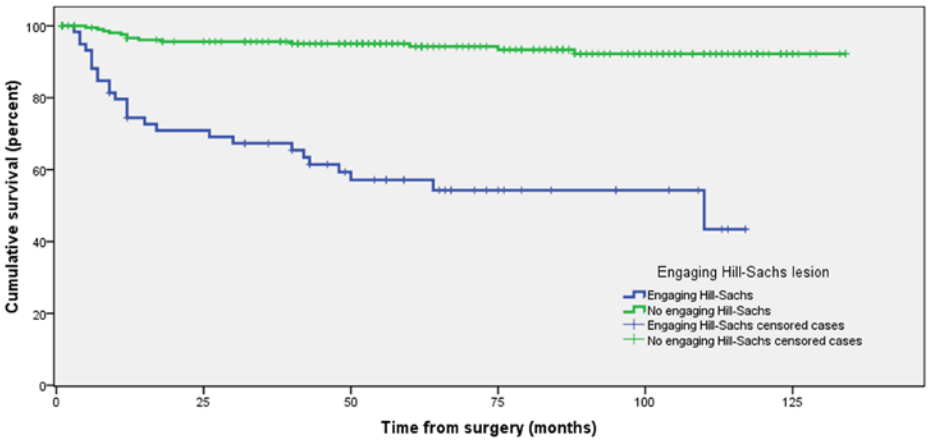

Fig. E1-C

Kaplan-Meier survivorship curves for groups with and without an engaging Hill-Sachs lesion.

TABLE E1-A Coding of Patient-Related Risk Factors for Recurrent Instability After Arthroscopic Bankart Repair and Capsular Shift

Variable	Categories
Sex	Male
	Female
Age at time of surgery	Continuous variable
Handedness	Left
	Right
Dislocation side	Left
	Right
Affected shoulder	Dominant
	Nondominant
Instability type	Subluxation
	Dislocation
	Both subluxation and dislocation
No. of dislocations or episodes of subluxation	<5
	5-10
	>10
Injury mechanism	Fall from <2 m
	Fall from >2 m
	Sports injury
	Motor-vehicle accident
	Assault
	Other
Previous instability of other shoulder	Yes
	No
History of dislocation in first-degree relative	Yes
	No
Evidence of generalized ligamentous laxity	Beighton[15] score of ≥4 (hyperlaxity)
	Beighton[15] score of <4
Positive Gagey sign	Yes
	No
Recurrent injury mechanism	No additional injury
	Additional injury
Occupation	Sedentary work or unemployed
	Light work
	Medium work
	Heavy work
	Very heavy work
Return to work or full activities of daily living	Did not return
	Returned
Level of participation in sports	None
	Occasional/social
	Regular amateur
	Professional
Level of risk of main sport played	None
	Noncontact/general fitness sport
	Contact or overhead sport
Return to sport after surgery	Yes
	No

TABLE E1-B Coding of Injury-Related Risk Factors for Recurrent Instability After Arthroscopic Bankart Repair and Capsular Shift

Variable	Categories
Bankart lesion extent	Anterior-inferior only
	Anterior-inferior & superior labral tear from anterior to posterior (SLAP) lesion
	Anterior labroligamentous periosteal sleeve avulsion (ALPSA)
Glenoid bone loss	0%-5%
	6%-24%
	≥25%
Size of Hill-Sachs lesion	Grade I (< 1.5 cm^2)
	Grade II (1.5-2.5 cm^2)
	Grade III (>2.5 cm^2)
Engaging Hill-Sachs lesion*	Yes
	No
No. of anchors used	1
	2
	3
	4

*An engaging Hill-Sachs lesion was defined as a posterior humeral defect that could be seen arthroscopically to be engaging the anterior rim of the glenoid when the arm was brought into a position of 90° of abduction combined with 90° of external rotation[3].

TABLE E2 Final Cox Regression Model Used to Predict Recurrent Instability After Arthroscopic Bankart Repair and Capsular Shift

Risk Factor	Regression Coefficient (B)	Standard Error of B	P Value	Exp (B) (with 95% CI)
Age (in years)	–0.090	0.031	0.004	0.91 (0.86-0.97)
Glenoid bone loss (percentage)*	0.582	0.217	0.007	1.79 (1.15-2.65)
Engaging Hill-Sachs lesion	1.697	0.409	<0.001	5.46 (2.45-12.17)

*Although glenoid bone loss was graded as a percentage of glenoid bone loss, it was considered as a categorical variable when used in the final model.

TABLE E3 Percentage Sensitivity, Specificity, and Positive and Negative Predictive Values of the Model at Different Cutoff Levels of Predicted Probability of Recurrence from the Statistical Model within Two Years After Arthroscopic Bankart Repair and Capsular Shift

Model Cutoff Level	Sensitivity	Specificity	Percentage of Entire Cohort Identified as Being at Risk for Failed Repair	Positive Predictive Value	Negative Predictive Value	Overall Percentage of Cohort Correctly Diagnosed
0.50	14	98	3	44	91	91
0.45	31	98	6	43	92	89
0.35	35	95	10	42	93	87
0.25	50	91	14	35	94	86
0.15	64	87	18	34	96	84
0.10	72	83	22	31	97	82
0.05	92	56	28	19	98	76

TABLE E4 Mean Functional Scores at Two Years After Arthroscopic Bankart Repair and Capsular Shift for the Recurrence and Nonrecurrence Groups*

Measurement	Recurrence Group	Nonrecurrence Group	P Value
Mean percentage deficit in WOSI score	40.5 (18.8-55.5)	27.5 (12.0-42.0)	<0.001
Mean percentage deficit in DASH score	12.9 (3.0-20.5)	5.0 (2.0-9.0)	<0.001

*The mean WOSI and DASH scores are expressed as a percentage deficit compared with normal function. The 95% CI is shown in parentheses.

TABLE E5 Drop-Out Analysis*

Variable	P Value
Age at time of surgery	0.565
Sex	0.263
No. of dislocations or episodes of subluxation	0.831
Injury mechanism	0.297
Level of participation in sports	0.335
Level of risk of main sport played	0.474
Return to sport after surgery	0.662
Evidence of generalized ligamentous laxity	0.196
Positive Gagey sign	0.744
Bankart lesion extent	0.846
Glenoid bone loss	0.626
Size of Hill-Sachs lesion	0.851
Engaging Hill-Sachs lesion	0.161
No. of anchors used	0.712

*There were no significant differences between the patients who were lost to follow-up and those who were followed.