Original Article With Video Illustration

Traumatic Glenohumeral Bone Defects and Their Relationship to Failure of Arthroscopic Bankart Repairs: Significance of the Inverted-Pear Glenoid and the Humeral Engaging Hill-Sachs Lesion

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Purpose: Our goal was to analyze the results of 194 consecutive arthroscopic Bankart repairs (performed by 2 surgeons with an identical suture anchor technique) in order to identify specific factors related to recurrence of instability. **Type of Study:** Case series. **Materials and Methods:** We analyzed 194 consecutive arthroscopic Bankart repairs by suture anchor technique performed for traumatic anterior-inferior instability. The average follow-up was 27 months (range, 14 to 79 months). There were 101 contact athletes (96 South African rugby players and 5 American football players). We identified significant bone defects on either the humerus or the glenoid as (1) "inverted-pear" glenoid, in which the normally pear-shaped glenoid had lost enough anterior-inferior bone to assume the shape of an inverted pear; or (2) "engaging" Hill-Sachs lesion of the humerus, in which the orientation of the Hill-Sachs lesion was such that it engaged the anterior glenoid with the shoulder in abduction and external rotation. **Results:** There were 21 recurrent dislocations and subluxations (14 dislocations, 7 subluxations). Of those 21 shoulders with recurrent instability, 14 had significant bone defects (3 engaging Hill-Sachs and 11 inverted-pear Bankart lesions). For the group of patients without significant bone defects (173 shoulders), there were 7 recurrences (4% recurrence rate). For the group with significant bone defects (21 patients), there were 14 recurrences (67% recurrence rate). For contact athletes without significant bone defects, there was a 6.5% recurrence rate, whereas for contact athletes with significant bone defects, there was an 89% recurrence rate. **Conclusions:** (1) Arthroscopic Bankart repairs give results equal to open Bankart repairs if there are no significant structural bone deficits (engaging Hill-Sachs or inverted-pear Bankart lesions). (2) Patients with significant bone deficits as defined in this study are not candidates for arthroscopic Bankart repair. (3) Contact athletes without structural bone deficits may be treated by arthroscopic Bankart repair. However, contact athletes with bone deficiency require open surgery aimed at their specific anatomic deficiencies. (4) For patients with significant glenoid bone loss, the surgeon should consider reconstruction by means of the Latarjet procedure, using a large coracoid bone graft. **Key Words:** Instability—Arthroscopic instability repair—Shoulder instability—Bone defect—Bone graft—Latarjet reconstruction.

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NOTE: To access the video illustration accompanying this report, visit the October on-line issue of *Arthroscopy* at www.arthroscopyjournal.org

The debate over the supremacy of open versus arthroscopic surgical repair for traumatic anterior instability rages now more energetically than ever. After more than a decade of unfocused confrontation, the debate has finally crystallized into a classic conflict between "lumpers" (the open proponents) and "splitters" (the arthroscopic proponents). On the one hand, the lumpers view every report of high recurrence rates from outdated transglenoid repairs as evidence that all arthroscopic repairs are somehow inherently inferior to the "gold standard" open repairs. On the other hand, the arthroscopists, who have evolved

into splitters, acknowledge that there have been problems with prior arthroscopic techniques but insist that they have now identified the essential surgical element that will produce results as good as the open gold standard.

Unfortunately for the arthroscopist, the issue is not so simple as using a single elusive technical innovation that will assure success in each case. Such a simplistic approach might be satisfactory for a handyman, but not for a scientist. The arthroscopic surgeon must recognize the pathology in each case and incorporate techniques that restore both the anatomy and the biomechanical function of the damaged structures.

You would think we would have made what we feel is our most critical observation before now. We are, after all, bone surgeons. What we are referring to is the fact that most of our arthroscopic Bankart repair failures have resulted from traumatic bone defects, either on the glenoid side or on the humeral side. The underlying cause of our failures has not been inadequate soft tissue fixation, but rather traumatic bone deficiency. The basic cause of postoperative recurrent dislocation has less to do with whether the repair is performed open or arthroscopically than it has to do with recognition and adequate treatment of mechanically significant bone defects.

At the close of the 1990s, we witnessed a phenomenal amount of effort being directed at shrinkage or plication of capsular tissues in the treatment of instability. The focus of technical refinements has been almost entirely on the soft tissues. We strongly believe that this focus has been misdirected and has diverted our attention from the main problem: traumatic glenohumeral bone defects. Therefore, we undertook this study in an attempt to analyze traumatic bone deficiency as a factor in redislocation after Bankart repair.

MATERIALS AND METHODS

We analyzed 194 arthroscopic Bankart repairs performed by the two of us between July 1992 and June 1998. One of us (S.S.B.) performed 43 of these surgeries between July 1992 and June 1998, and the other (J.F.DeB.) performed 151 of the surgeries between January 1994 and May 1998. This was a consecutive series of traumatic anterior instability for each author, with the following exclusions: anterior instability due to humeral avulsion of the glenohumeral ligaments (HAGL lesions) and cases with associated subscapularis tendon avulsions were deemed not to be appropriate for arthroscopic repair. Eight of the patients had had previous surgery for instability. One author (S.S.B.) had an average follow-up of 31 months (range, 14 to 79 months) and the other author had an average follow-up of 26 months (range, 14 to 54 months). For the combined series of 194 patients, the average follow-up was 27 months (range, 14 to 79 months). All patients had a minimum follow-up of 14 months.

The average age at the time of surgery was 27.9 years (range, 15 to 64 years). There were 170 male patients (87.6% of the total) and 24 female patients (12.4% of the total). The dominant arm was involved in 141 of the 194 shoulders (72.7%). All patients had sustained at least 1 traumatic anterior dislocation. There were 101 contact athletes (96 South African rugby players and 5 American football players). The other dislocations were sustained in various noncontact athletic activities or in industrial or household accidents.

Significant bone defects were either on the humerus or the glenoid. We defined significant humeral bone defects to be "engaging" Hill-Sachs lesions. These lesions, to be explained in greater detail later in this article, were oriented in such a way that they engaged the anterior glenoid in a position of athletic function (defined as 90° abduction combined with external rotation anywhere between 0° and 135°). We defined a significant glenoid bone defect as one in which the arthroscopic appearance of the glenoid (as viewed through a posterior or anterior portal with the arthroscope placed superiorly and viewing inferiorly) was that of an "inverted pear." The normal glenoid is pear shaped, with a larger diameter below the midglenoid notch than above it. With the inverted-pear glenoid, the inferior glenoid had a smaller diameter than the superior glenoid. There were significant bone defects in 21 patients (3 engaging Hill-Sachs lesions, and 18 inverted-pear Bankart lesions). Nine of these 21 patients were contact athletes (all rugby players). There were 16 first-time dislocations, 4 of which had significant bone defects (all were glenoid bone defects).

Both authors performed an identical arthroscopic Bankart repair using a variety of metallic suture anchors. The glenoid neck was lightly prepared with a high-speed burr, being careful not to remove significant amounts of bone and thus contribute to bone deficiency. The capsuloligamentous complex was dissected free of the underlying subscapularis so that it could be easily reapproximated anatomically to the "corner" of the glenoid, with suture anchors that were placed approximately 1 cm apart. Care was taken not to medialize the suture anchors. Braided polyester suture was used for the repair. An average of 3 suture

anchors was used per case. Anatomic reconstruction was the goal, without any attempt to take up additional slack in the tissues by techniques such as rotator interval closure or thermal shrinkage.

Patients with significant bone defects discovered arthroscopically underwent immediate open surgery. Those with engaging Hill-Sachs lesions underwent open capsular shift reconstruction,[1] and those with inverted-pear glenoids had an open Latarjet procedure.[2,3] Although these patients were not a part of the 194 patients in this series, their open treatment modalities are outlined in the Discussion for completeness of the treatment protocol.

The postoperative protocol required immobilization of the operated extremity in a sling or shoulder immobilizer for 3 weeks. Forward flexion was begun 3 weeks postoperatively, and external rotation at 6 weeks. Strengthening exercises were begun 8 weeks postoperatively.

Statistical analysis was used to compare recurrence rates for patients with bone deficiency versus those without bone deficiency. Pearson's χ-square test and the Fisher exact probability test were used.

RESULTS

We had 21 recurrent dislocations and subluxations (14 dislocations, 7 subluxations), for a 10.8% recurrence rate. However, of those 21 recurrent dislocations and subluxations, 14 had significant bone defects (3 engaging Hill-Sachs lesions and 11 inverted-pear Bankart lesions).

We found it useful to divide our patients into 2 groups: those without significant bone defects (173 patients) and those with significant bone defects (21 patients). For the group without significant bone defects, there were 7 recurrences in 173 patients, for a 4% recurrence rate. For the group with significant bone defects, there were14 recurrences in 21 patients, for a 67% recurrence rate. Thus, the recurrence rate for patients without significant bone defects was found to be significantly lower than the recurrence rate for patients with significant bone defects, with a P value approaching zero ($P < .0001$), when analyzed both by Pearson's χ-square test and the Fisher exact probability test.

Of the 101 contact athletes (96 rugby players and 5 American football players), there were 9 significant bone defects, all in rugby players, 3 of which were first-time dislocators. Of the 9 rugby players with significant bone defects, 8 have redislocated, for an 89% recurrence rate in rugby players with bone defects. For rugby players without bone defects (87 patients), there were 6 recurrences, for a 7% recurrence rate. For American football players (5 patients), none of whom had bone defects, there were no recurrences. The combined recurrence rate for contact athletes (rugby and American football players) without bone defects was 6 of 92, or 6.5%. This recurrence rate for contact athletes without significant bone defects is significantly lower than that for contact athletes with significant bone defects, when analyzed by the aforementioned statistical methods ($P < .0001$).

DISCUSSION

Arthroscopic Versus Open Repairs

Arthroscopic Bankart repairs remain controversial primarily because of reports of high recurrence rates, ranging up to 44% for transglenoid repairs.[4,5] However, on critical analysis of the literature, the recurrence rate is quite variable and can be very low even with transglenoid sutures.[6,7] A report on a recent series of arthroscopic suture anchor repairs[8] described excellent results with only a 7% recurrence rate in an athletic population. The results in this arthroscopic suture anchor study equal those of the gold standard open Bankart repair.[9]

If one looks at arthroscopic reports, the best results are those that emphasize repair of the capsulolabral tissue to the corner of the glenoid, or even onto the face of the glenoid.[6-8,10-12] This should not be surprising, since Neviaser[13] described a fairly common pattern of medialized capsulolabral healing (the ALPSA lesion, or anterior labroligamentous periosteal sleeve avulsion) associated with recurrent dislocation. If we repair the capsule in a medialized position, the position of an ALPSA lesion, we would expect a higher recurrence rate. Indeed, this is exactly where many of the techniques of arthroscopic staple capsulorraphy or transglenoid labral repair positioned the labrum, so one should expect a relatively high failure rate with this approach.

In contrast, the open Bankart repair as described by Rowe[9] and by Thomas and Matsen[14] used transosseous tunnels for suture that exited onto the face of the glenoid and automatically lateralized the capsulolabral repair. Suture anchor techniques, both open[15] and arthroscopic,[6-8,10-12] that lateralize the labrum have also been shown to give excellent results.

There has been much debate recently in orthopaedic circles about open versus arthroscopic Bankart repairs and whether the arthroscopic repairs are inherently

weaker for some reason, such as having less inflammatory response with fewer "spot-welds" (suture anchor fixation points). We do not believe that there is any evidence of inherent weakness in arthroscopic repairs. One need only look at the results of the open Dutoit staple capsulorraphy to realize that the issue is not whether the operation is open or arthroscopic. The Dutoit procedure[16] medialized the capsulolabral complex to create a surgically produced ALPSA lesion. Not surprisingly, long-term follow-up of this open stabilization procedure showed an unacceptable recurrence rate of 22%.[17] This recurrence rate is in the same range as that of the arthroscopic staple capsulorraphy, which Johnson reported to be 21%.[18] In this case, the problem was not that the procedure was done arthroscopically, but that the repair was medialized. Granted, the untoward effects of medializing the repair had not yet been discovered when either of these clinical series was performed. However, the fact remains that a bad operation is a bad operation whether it is performed arthroscopically or open.

For a straightforward Bankart lesion, current reports indicate that either open or arthroscopic Bankart repair can give equally good results.[6,12,14,15] On the other hand, an operation whose principles are ill conceived (e.g., medialization of the capsuloligamentous complex) will have poor results whether it is performed open or arthroscopic.

Recurrence Related to Glenohumeral Bone Defects

In our series of arthroscopic Bankart repairs, we defined a significant glenoid bone defect as one in which the arthroscopic appearance of the glenoid when viewed from a superior-to-inferior perspective was that of an inverted pear (see Video). The geometry of the inverted-pear glenoid is the reverse of the normal pear-shaped glenoid. This pathologic bone-deficient variation has a smaller diameter below the midglenoid notch than above it, and this configuration is obvious when viewed arthroscopically.

On the humeral side, we defined a significant bone defect to be an engaging Hill-Sachs lesion (see Video). These defects were Hill-Sachs lesions that we could see arthroscopically that engaged the anterior rim of the glenoid when the arm was brought into a position of athletic function (a position that we defined as 90° abduction combined with external rotation in the range between 0° and 135°).

With the above definition of significant glenohumeral bone defects, all 194 of our patients fell clearly into 1 of 2 categories: those with significant bone defects and those without them. When we compared the recurrence rates for these 2 groups of patients, there was a striking difference: the recurrence rate for the standard arthroscopic Bankart repair without a significant bone defect (4% recurrence) was equal to that of the gold standard open repair, whereas the recurrence rate for the arthroscopic Bankart repair in the face of a significant bone defect was an unacceptable 67%. Further subdividing the patients with major bone defects into glenoid and humeral defects, we found that those with an inverted-pear glenoid had a 61% (11 of 18) recurrence rate and that those with an engaging Hill-Sachs lesion had a 100% (3 of 3) recurrence rate.

How can we explain the difference in behavior and prognosis in these shoulders with significant bone defects? The answer lies in the reconfigured geometry of the injured shoulder with a traumatic humeral or glenoid defect, as we shall discuss.

The Humeral Side: The Hill-Sachs Lesion as a Cause of Articular-Arc Deficit

The shoulders with large Hill-Sachs lesions that were rated as failures did not redislocate. In fact, they probably did not even resubluxate. What these patients complained of was a catching or popping sensation with the shoulder in the abducted and externally rotated position. They had enough apprehension in that position that they voluntarily curtailed overhead activities. Despite the fact that these patients were probably not true subluxators, they were classified as failures because their symptoms of instability recurred.

One of us (S.S.B.) had an opportunity to perform a second-look arthroscopy on 1 of these patients[19] and found that the Bankart lesion had healed. Dynamic arthroscopic examination of the shoulder as it went into abduction and external rotation revealed the geometric etiology of the symptoms: there was an articular-arc deficit on the humeral side with an engaging Hill-Sachs lesion (Fig 1). That is, with the arm in abduction of 90°, if the shoulder was externally rotated more than 30°, the Hill-Sachs lesion would engage the anterior corner of the glenoid, and the patient would sense that engagement as a popping or catching sensation. (See Case Report in this issue: Burkhart and Danaceau. Articular arc-length mismatch as a cause of failed Bankart repair.[19])

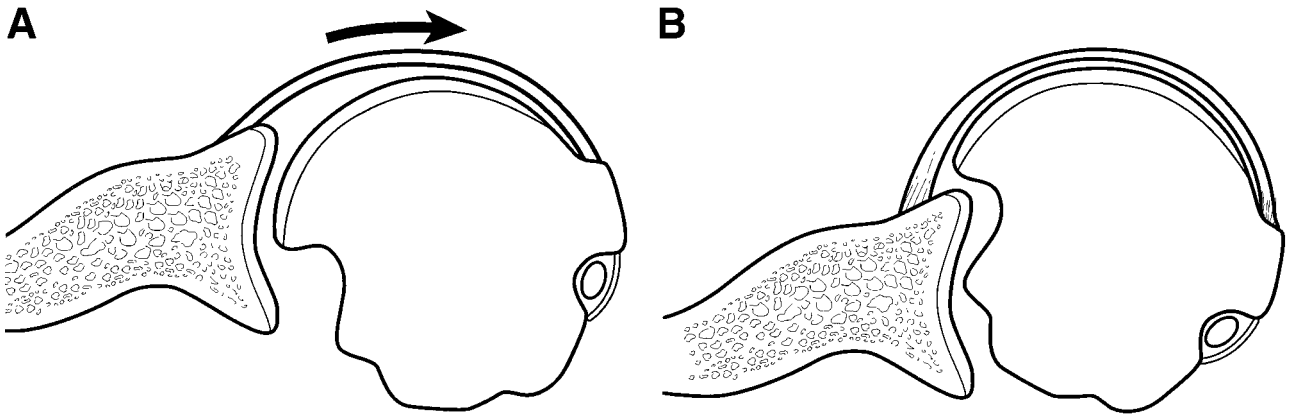

FIGURE 1. This large Hill-Sachs lesion involves a large portion of the humeral articular surface. In this case, even without a Bankart lesion, the Hill-Sachs lesion can engage the anterior corner of the glenoid, causing symptoms similar to subluxation. We call this an articular arc deficit.

Engaging Versus Nonengaging Hill-Sachs Lesions

This brings up the issue of engaging versus nonengaging Hill-Sachs lesions. We have not seen this topic discussed previously in the literature, but we feel it is very important in predicting the success of arthroscopic Bankart repairs. We define an engaging Hill-Sachs lesion as one that presents the long axis of its defect parallel to the anterior glenoid with the shoulder in a functional position of abduction and external rotation, so that the Hill-Sachs lesion engages the corner of the glenoid (Fig 2). A nonengaging Hill-Sachs lesion is one that presents the long axis of its defect at a diagonal, nonparallel angle to the anterior glenoid with the shoulder in a functional position of abduction and external rotation (Fig 3), or one in which the "engagement point" occurs with the arm in a nonfunctional position of shoulder extension or of low shoulder abduction ($<70°$ abduction). Because this first type of nonengaging Hill-Sachs lesion passes diagonally across the anterior glenoid with external rotation, there is continual contact of the articular surfaces and nonengagement of the Hill-Sachs lesion by the anterior glenoid. Such shoulders are reasonable candidates for arthroscopic Bankart repair because they do not have a functional articular-arc deficit.

Obviously, for every Hill-Sachs lesion, there is a position of the shoulder at which the humeral bone defect will engage the anterior glenoid. The symptoms are greatest if the engagement occurs with the shoulder in a functional position, which typically involves a combination of flexion, abduction, and external rotation. However, we have found that many Hill-Sachs lesions engage only when the shoulder is in some degree of extension, which is a nonfunctional position for everything except throwing a baseball, or in abduction of less than 70°, which is also a nonfunctional position. Therefore, we define this second group of Hill-Sachs lesions as nonengaging.

The orientation of the Hill-Sachs lesion is determined solely by the position of the humeral head relative to the glenoid when it becomes indented by the glenoid. This can occur with the shoulder in any degree of abduction or with the arm at the side and is not necessarily the degree of abduction in which the shoulder dislocated. For example, the shoulder may dislocate with the arm at 90° of abduction, and then assume a position of 0° abduction after the dislocation. Hence, the Hill-Sachs lesion that becomes indented with the arm at the side with some extension of the shoulder will be located more vertically and superiorly than the lesion that indents with the shoulder abducted and externally rotated. This former lesion (the Hill-Sachs that becomes indented with the arm at the side) is generally a nonengaging lesion.

Once we recognize an engaging Hill-Sachs lesion with an articular-arc deficit, we must not only repair the Bankart lesion (if there is one), but we must also keep the Hill-Sachs lesion from engaging the anterior glenoid. This can be done in 1 of 3 ways:

First, the surgeon can restrict external rotation enough that the lesion will not engage (Fig 4). We are of the opinion that this can be accomplished most predictably by an open capsular shift procedure.[1] We

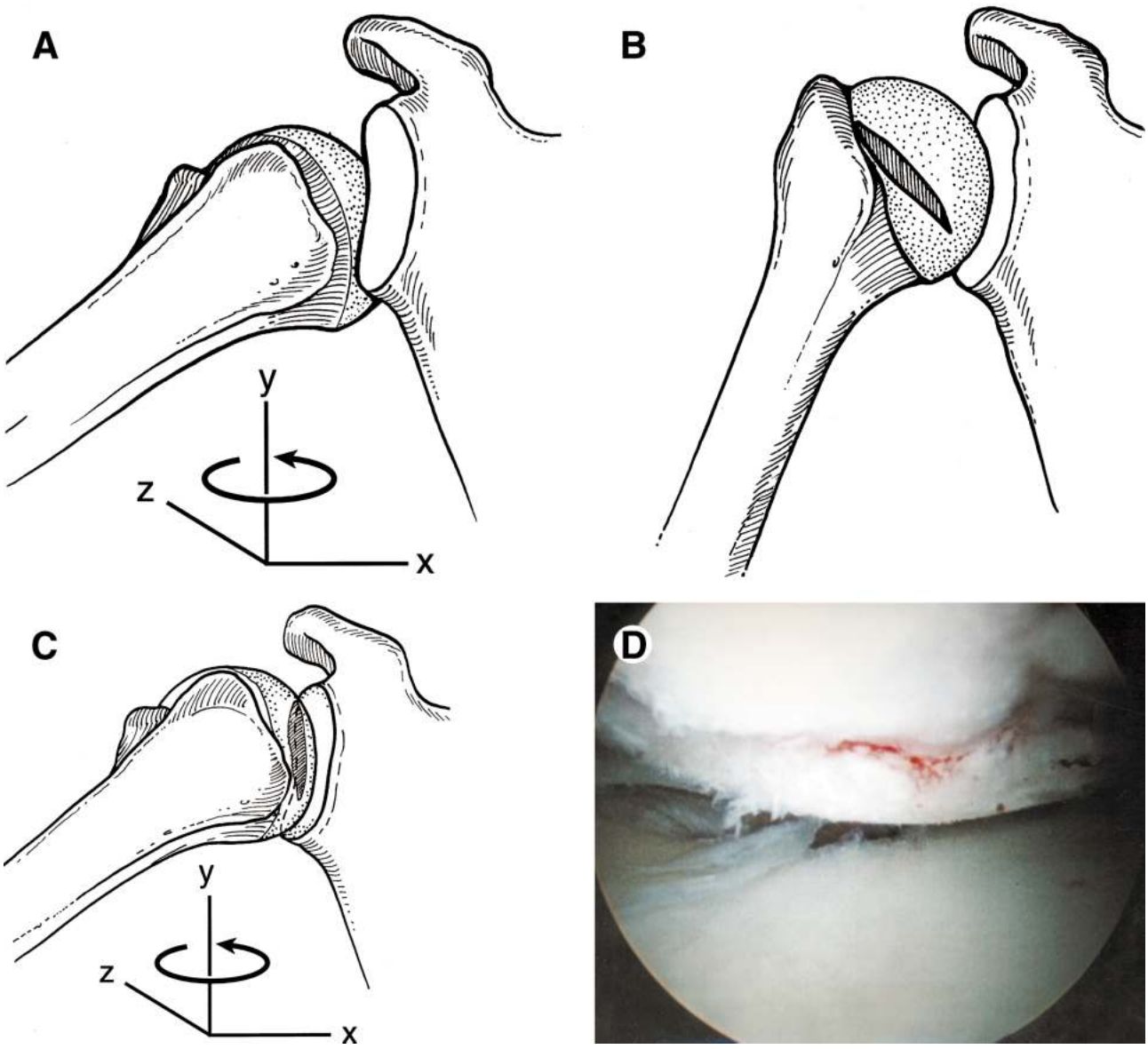

FIGURE 2. Engaging Hill-Sachs lesion. In a functional position of abduction and external rotation, the long axis of the Hill-Sachs lesion is parallel to the glenoid and engages its anterior corner. (A) Creation of lesion with arm in abduction and external rotation. (B) Orientation of Hill-Sachs lesion. (C) Engagement of Hill-Sachs lesion in functional position of abduction and external rotation. (D) Arthroscopic photograph of an engaging Hill-Sachs lesion in a right shoulder as it approaches its engagement position with the anterior glenoid rim.

do not believe that arthroscopic capsular plication or thermal capsulorraphy can predictably restrict external rotation to the extent that may be required, so we recommend open capsular shift in this situation.

Second, the surgeon may choose not to restrict external rotation, but instead to lengthen the articular arc of the humerus and fill in the Hill-Sachs defect so that it can no longer engage. This can be performed most precisely by a size-matched humeral osteoarticular allograft, but it can also be accomplished with a corticocancellous iliac graft.[20] We recommend this procedure in patients in whom it is important to maintain external rotation, such as overhead athletes, or in patients who fail a capsular

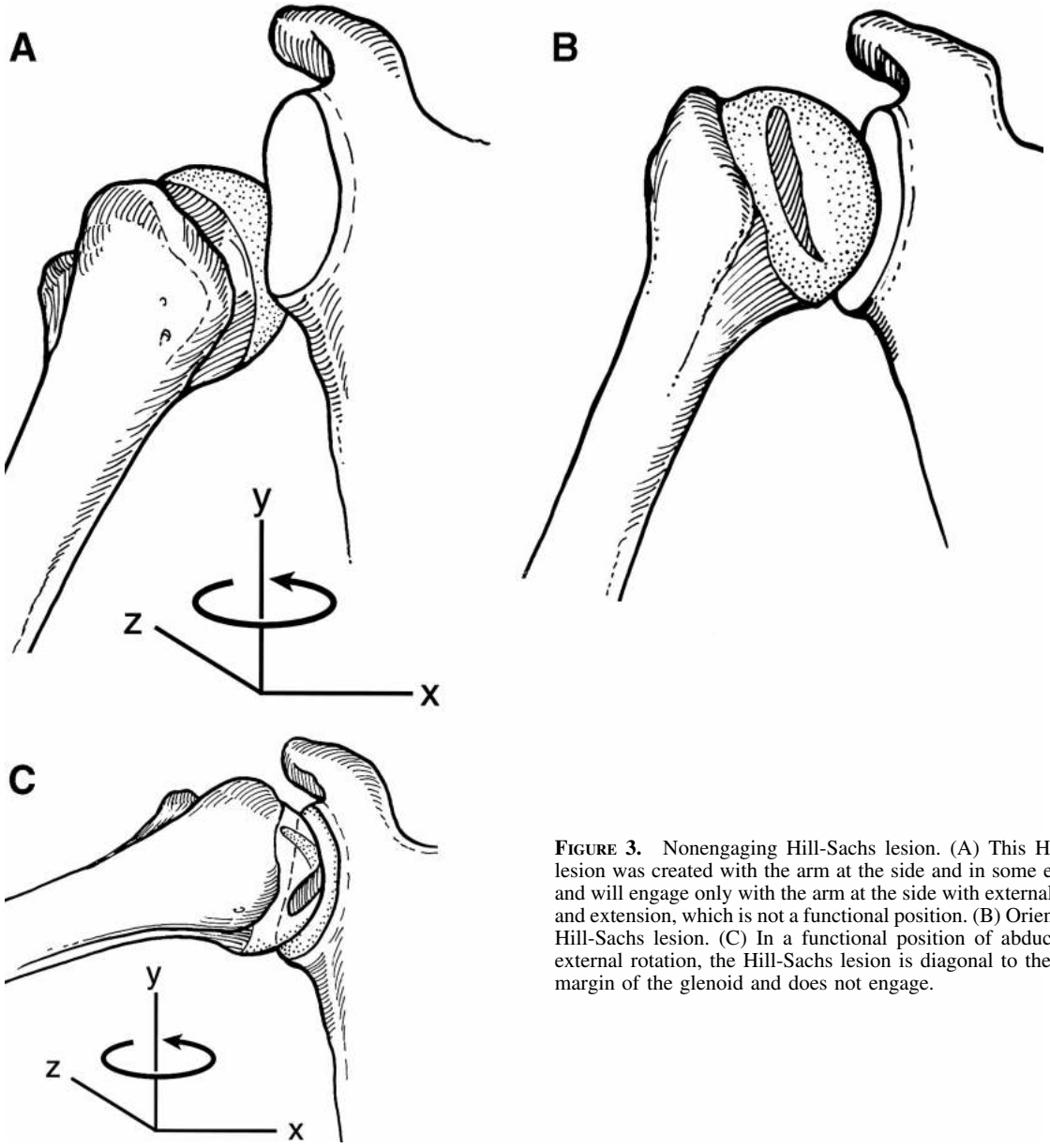

FIGURE 3. Nonengaging Hill-Sachs lesion. (A) This Hill-Sachs lesion was created with the arm at the side and in some extension and will engage only with the arm at the side with external rotation and extension, which is not a functional position. (B) Orientation of Hill-Sachs lesion. (C) In a functional position of abduction and external rotation, the Hill-Sachs lesion is diagonal to the anterior margin of the glenoid and does not engage.

shift procedure and begin to engage the Hill-Sachs lesion again.

Third, the surgeon may try to prevent the Hill-Sachs lesion from engaging by performing a rotational proximal humeral osteotomy[21] and internally rotating the articular surface of the humerus. If the articular surface is adequately rotated, the Hill-Sachs lesion will not engage. This is a formidable operation with significant potential morbidity, so we recommend it only if all other treatment options have failed.

The Glenoid Side: The Inverted-Pear Configuration Caused by Bony Bankart or Impression Bankart Lesions Resulting in Containment Failure

All of our failures that were associated with bony Bankart lesions had anterior-inferior glenoid defects that were large enough to significantly narrow the inferior half of the glenoid. Ordinarily, the glenoid, when viewed *en face*, has the shape of a pear, with the lower half being significantly wider than the upper

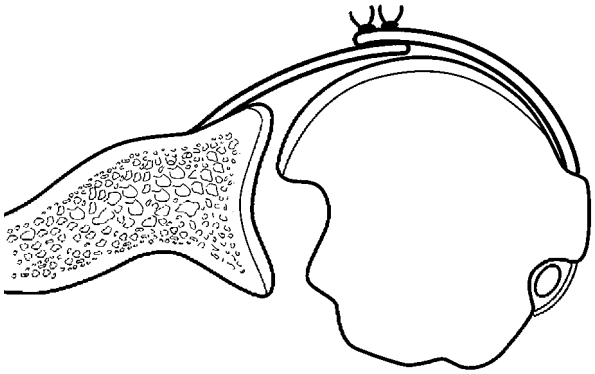

FIGURE 4. Capsular plication can restrict external rotation enough so that the Hill-Sachs lesion will not engage the anterior glenoid.

half (Fig 5A). With a large bony Bankart lesion, or even a Bankart lesion without an associated bone fragment but with a significant impression (compression) defect, the shape of the glenoid changes to that of an inverted pear, where the top half of the glenoid is wider than the lower half (Figs 5B and C). This inverted pear is immediately recognizable when the surgeon views through an anterosuperior portal and looks down inferiorly on the glenoid. Bigliani et al.[22] reported a 12% recurrence rate in patients with glenoid rim fractures who had undergone Bankart repair. They recommended coracoid transfer if the glenoid rim fracture comprised 25% of the anterior-posterior diameter of the glenoid. They did not specify the type of coracoid transfer (Bristow *v* the much larger cora-

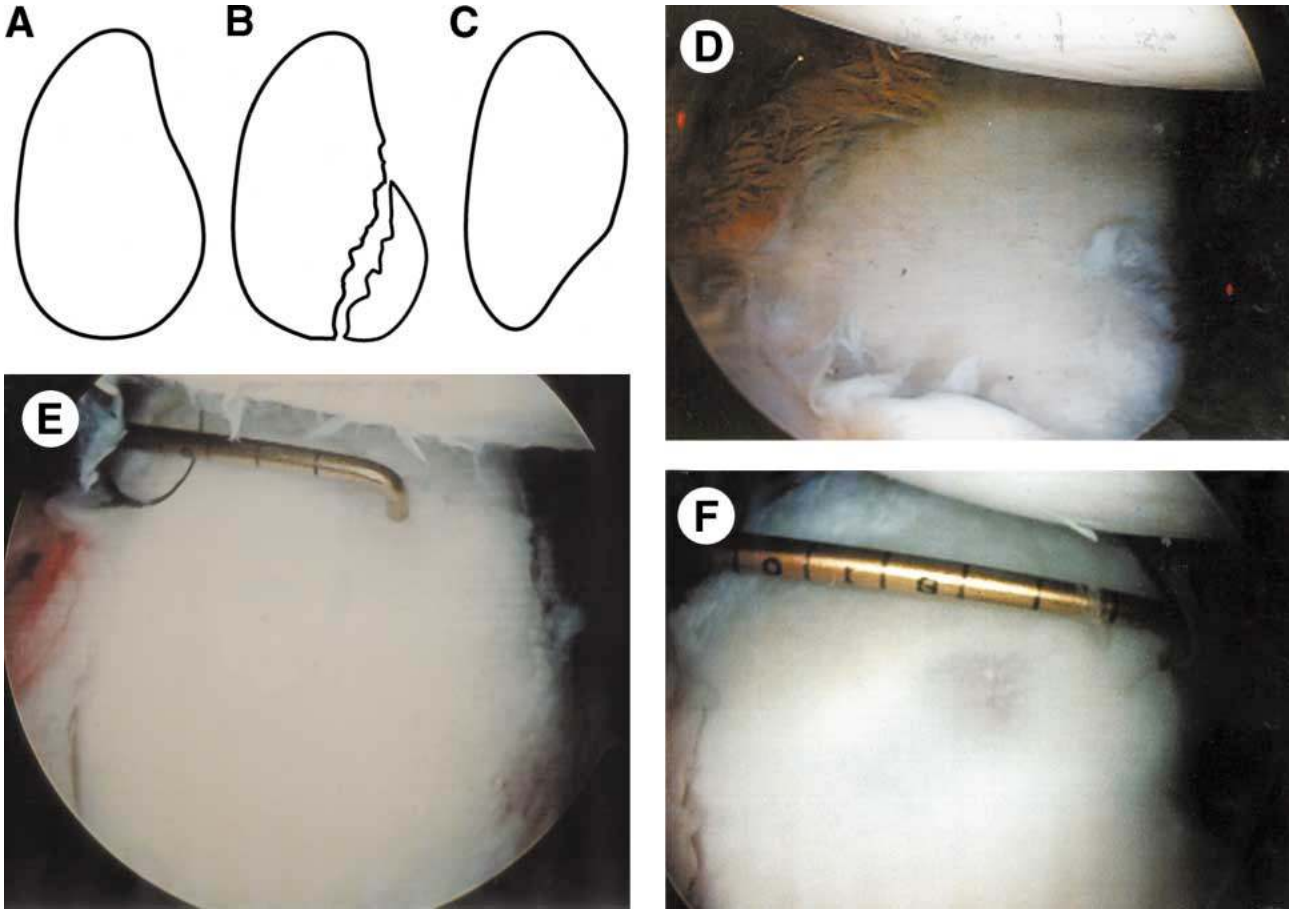

FIGURE 5. (A) The normal shape of the glenoid is that of a pear, larger below than above. (B) A bony Bankart lesion can create an inverted-pear configuration. (C) A compression Bankart lesion can also create an inverted pear. (D) Arthroscopic view of an inverted-pear glenoid (left shoulder) as viewed from an anterosuperior portal. Note how the inferior glenoid (at top) narrows to an apex that is much narrower than the superior glenoid (at bottom). (E) Left shoulder viewed through an anterosuperior portal. The tip of the hook probe rests on the bare spot of the glenoid, and the 3-mm laser marks on the probe indicate a 12-mm distance from the bare spot to the posterior glenoid rim. (F) The probe is placed just inferior to the bare spot, showing that the distance from the bare spot to the anterior glenoid rim is 6 mm, suggesting a 6-mm bone loss from compression of the anterior glenoid. This represents a 25% reduction in the diameter of the inferior glenoid.

coid graft of the Latarjet procedure) that they recommended.

Impression Bankart lesions result from compression of the anterior glenoid and have no bony fragment to indicate the amount of bone that has been lost from the articular arc. We are in the process of completing an anatomic study that appears to confirm that the bare spot of the normal glenoid lies equidistant between the anterior and posterior rims of the glenoid and, thus, is a good reference point for estimating the percentage of glenoid bone loss from compression (Figs 5E and F).

In the case of bony Bankart lesions, if one excises the bony Bankart fragment and repairs the capsulolabral complex to the remaining glenoid despite an inverted-pear configuration, the repair may seem secure. However, the glenoid bone loss can create a serious containment problem for the shoulder.

Containment of the humeral head by the glenoid is a result of 2 geometric variables. The first is the "deepening effect" of a wider glenoid due to the longer arc of its concave surface (Fig 6A). This serves to "deepen the dish" of the glenoid. Loss of part of the glenoid surface will cause the glenoid "dish" to be shallower and, therefore, less resistant to shear forces that might tend to cause a dislocation (Fig 6B).

The second geometric variable affecting containment of the humeral head is the arc length of the glenoid. Axial humeral forces are resisted by the glenoid until the direction of the force vector passes beyond the edge of the glenoid (Fig 7). At that point, such forces are concentrated at the bone-ligament interface and can cause a Bankart lesion. With anterior-inferior glenoid bone deficiency, the safe zone, or arc through which the glenoid resists axial forces, may be much smaller than in a normal joint (Figs 8 and 9), so that the glenohumeral ligaments are presented with a load that would normally be resisted by the bony buttress of the glenoid. No wonder the inverted-pear configuration predisposes to ligament disruption and recurrent dislocation. Our series confirms the predilection of the inverted-pear configuration for recurrent dislocation. Of 18 shoulders with the inverted-pear

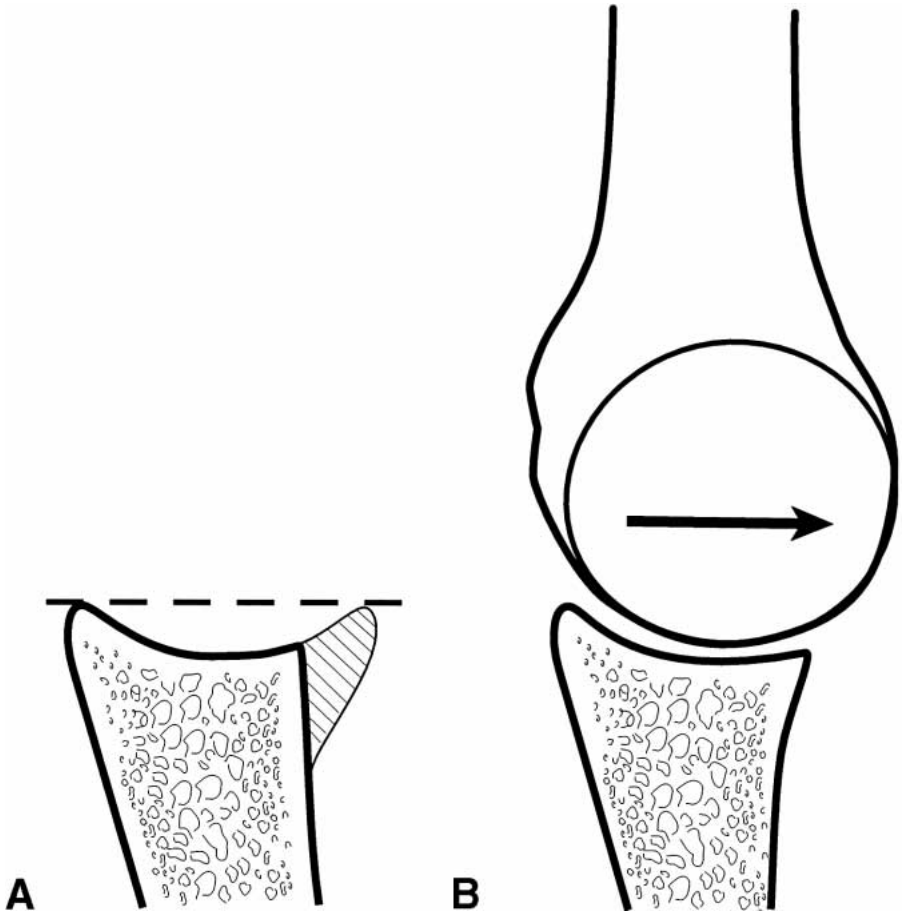

FIGURE 6. (A) The anterior glenoid rim serves to "deepen the dish" of the glenoid and acts as a buttress to resist dislocation. (B) A shoulder with a bony Bankart lesion has a shallower "dish" anteriorly with less resistance to shear forces.

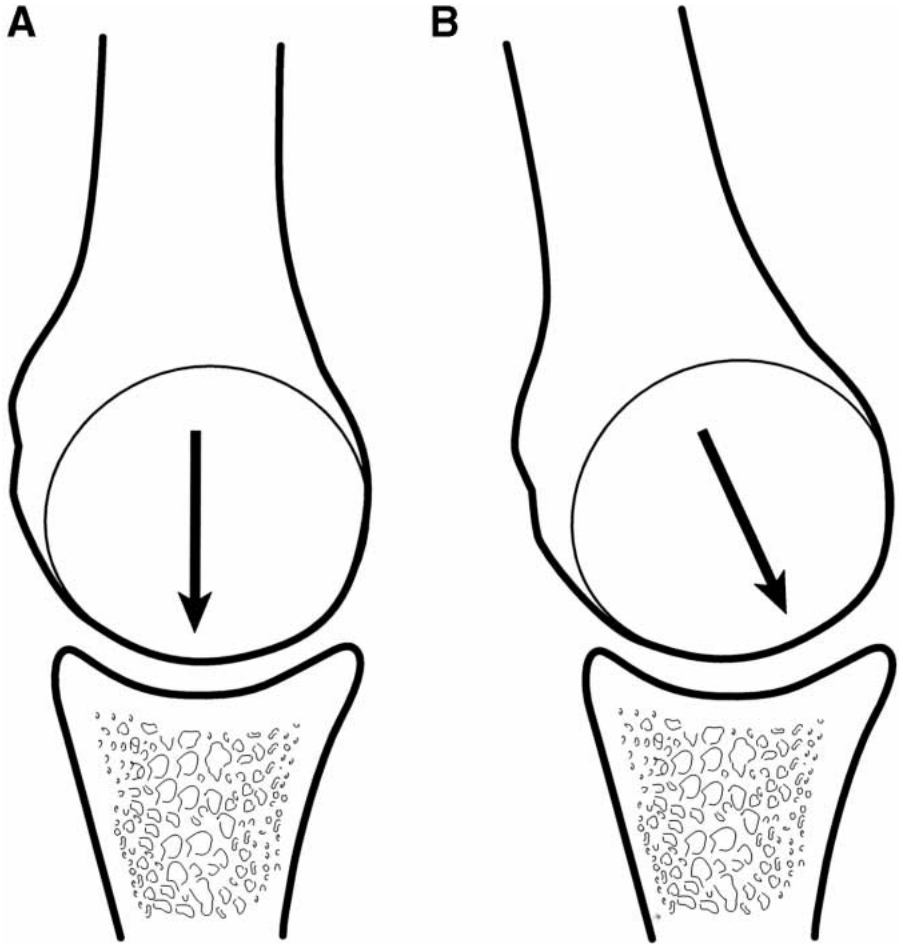

FIGURE 7. Effect of glenoid arc length. The glenoid resists axial forces at a variety of angles from the humerus throughout the glenoid arc length (A and B). Force vectors that pass beyond the edge of the glenoid can create Bankart lesions. Loss of a part of the glenoid articular surface (e.g., a bony Bankart lesion) shortens the arc through which the glenoid can resist humeral forces.

glenoid, 11 redislocated after arthroscopic Bankart repair, for a 61% recurrence rate.

Correcting the Inverted Pear: The Latarjet Procedure

The most logical solution to the problem of anterior glenoid bone deficiency is to rebuild this bony anterior buttress with a bone graft. However, free iliac grafts to the glenoid may not reliably hold up and are prone to resorption. The Bristow[23] procedure has too small a graft surface to create a bony buttress or to enlarge the arc length of the glenoid, and it primarily depends on the sling effect of the conjoined tendon as the shoulder is brought into abduction and external rotation.

Our preference is to reconstruct the anterior-inferior glenoid by means of the Latarjet procedure.[2,3] This operation differs from the Bristow procedure in that a much larger piece of bone (about 2 to 3 cm in length) is used to create a more formidable arc length extension of the anterior-inferior glenoid. The procedure is performed as follows:

As a final check of the anterior rim before committing to an osteotomy of the coracoid, the glenoid is exposed. This exposure is accomplished by detaching the upper half of the subscapularis and dissecting it off the capsule before capsular incision. The inferior portion of the capsule must be dissected from the deep surface of the lower subscapularis (Fig 10). If this is not done, the inferior part of the capsule will adhere to the subscapularis, and the surgeon will have difficulty repairing it later to the corner of the remaining glenoid. The anterior capsuloligamentous complex is dissected medially as far as possible before transecting the capsular sleeve to be sure there is sufficient length to the capsule to later repair to the glenoid rim (Figs 11A and B). This step may also be performed after coracoid osteotomy when there is better visualization. The pectoralis minor is dissected from the coracoid

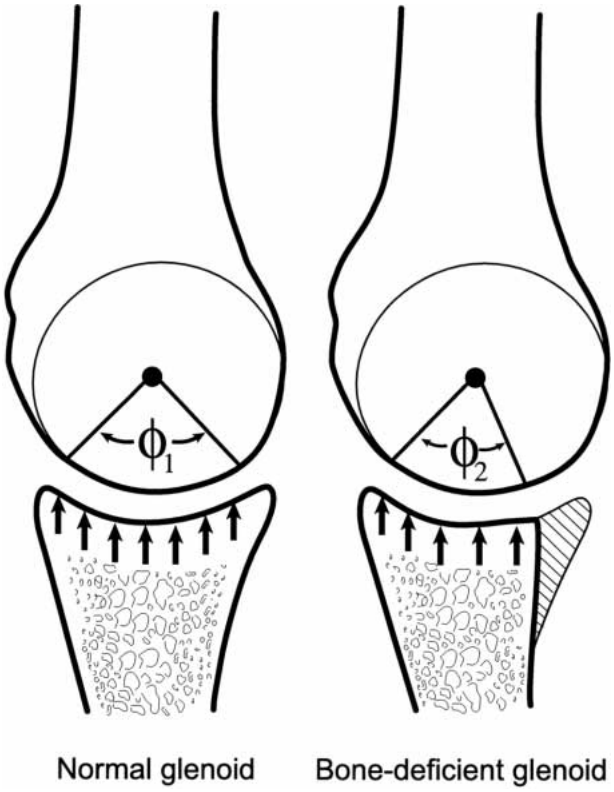

FIGURE 8. Glenoid bone loss shortens the "safe arc" through which the glenoid can resist axial forces. $Ø_2$ (bone-deficient condition) is less than $Ø_1$ (normal glenoid).

leaving a sliver of bone with the tendon (Figs 12 and 13), then the coracoid is osteotomized just proximal to the angle (elbow) of the coracoid, from medial to lateral, using an osteotome or angled saw (Fig 14). The osteotomy should traverse the coracoid between the pectoralis minor insertion and the attachment of the coracoclavicular ligaments. The coracoclavicular ligaments must be left intact so as not to destabilize the acromioclavicular joint. The osteotomy may be done with an osteotome, but an angled saw is particularly helpful in cutting the thick superolateral cortex of the coracoid.

The conjoined tendon is left attached to the coracoid graft because this provides some blood supply to the coracoid and makes this a vascularized graft; the transferred coracoid graft can continue to serve as a stable attachment point for the conjoined tendon. The coracoid graft is rotated about its long axis to obtain the best fit against the anterior glenoid, and then a high-speed burr is used to contour the graft for an exact fit to the anterior glenoid. At this point, if it has not already been done, the capsule is dissected from the glenoid as far medially as possible before incising it for later repair. It is often easier to dissect the capsule from the glenoid after the coracoid has been osteotomized. Suture anchors are then placed along the glenoid at the 3, 4, and 5 o'clock positions for later capsulolabral repair after the coracoid graft is in place (Fig 15). The coracoid graft is fixed to the glenoid with 2 bicortical screws (Fig 16). Then the capsule is repaired to the glenoid by means of the previously placed suture anchors (Fig 17A and B). In this way, the graft becomes an extra-articular platform to extend the safe arc through which the glenoid can resist axial forces (Fig 18). It is important to note that the graft is not intended to be a bone block (Fig 19). The detached pectoralis minor may be repaired to the base of the coracoid with a suture anchor (per J.F.DeB.) or simply approximated to the adjacent soft tissues (per S.S.B.). Finally, the upper half of the subscapularis muscle is repaired over the bone graft, with the conjoined tendon penetrating anteriorly between the upper and lower halves of the subscapularis (Fig 20).

Allain et al.[3] recently reported a high rate of late osteoarthritis following the Latarjet procedure (58% of 52 shoulders). This was a long-term follow-up study (average, 14.3 years), and the majority of patients with osteoarthritis had only grade I findings. The factors that these authors identified as predisposing toward osteoarthritis were (1) coracoid graft placement too far laterally and (2) coexistent rotator cuff tear at the time of the original injury. One must wonder whether the late osteoarthritis is due at least partially to the fact that these individuals had sustained intra-articular fractures predisposing them to a higher risk of late degenerative change. Interestingly, none of the 52 shoulders had redislocated at an average of 14.3 years postoperatively, testifying to the stability of this construct.

In patients who have both an inverted-pear glenoid and an engaging Hill-Sachs lesion, we have found that the Latarjet procedure alone is usually adequate to treat this combined bone deficiency. In this case, the bone graft corrects the glenoid deficiency so that it can resist axial forces across an expanded glenoid diameter, and the graft also lengthens the glenoid articular arc to prevent the Hill-Sachs lesion from engaging.

Contact Athletes

Contact athletes represented a significant segment of our study population. There were 96 rugby players and 5 American football players, for a total of 101 contact athletes. The authors have approximately

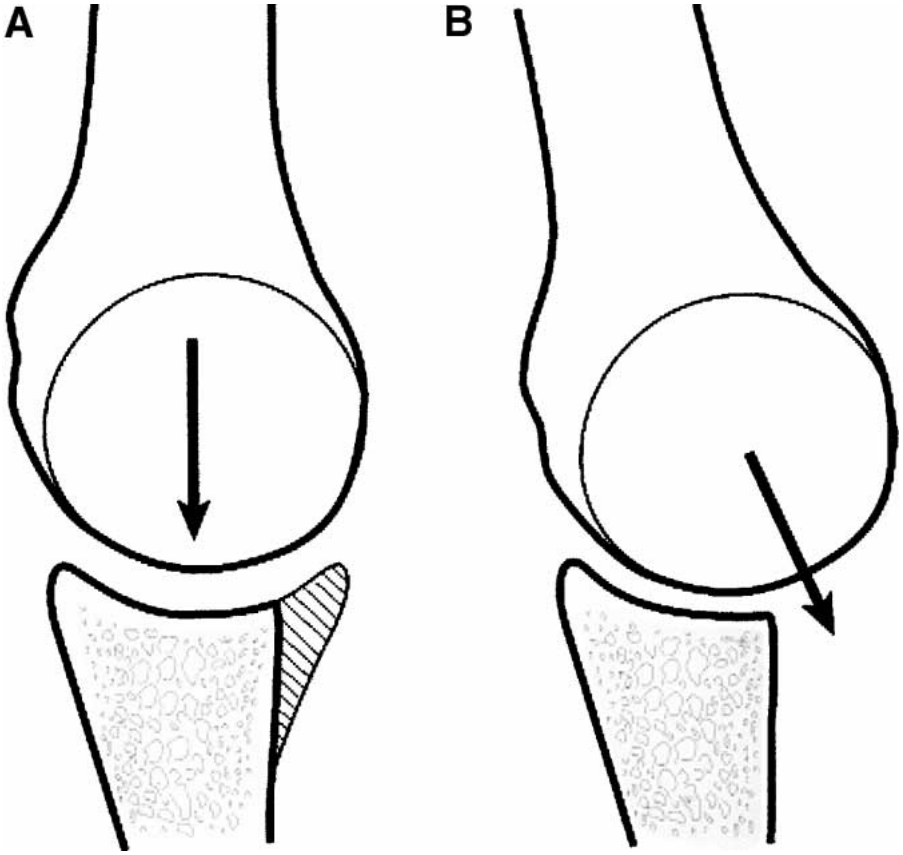

FIGURE 9. (A) Axial force from the humerus applied centrally on the glenoid will not create a Bankart lesion or failure of a soft tissue Bankart repair. (B) If an axial force is applied through a point beyond the edge of the deficient glenoid, failure of a soft-tissue Bankart repair is likely because the load must be borne by the soft tissues.

equal percentages of their practices devoted to sports medicine, yet the rugby players represented 63% of the South African patients in this study whereas the American football players comprised only 12% of the American patients. We realize that there may be some subtle demographic reasons for the disparity in the percentage of contact athletes operated on by the 2 authors (e.g., referral patterns to each surgeon). Even so, with the rugby players representing such a significantly higher percentage of the total than the American football players, we have concluded that playing rugby produces a higher incidence of anterior dislocation than American football. Additionally, the rugby players had a much higher incidence of significant bone defects that the American football players (9.4% for rugby *v* 0% for American football), implying that there must be a different mechanism of injury between the 2 groups. In this regard, we believe that a unique strength of this study is the fact that it has allowed us to compare 2 different categories of collision athletes and search for specific factors that might explain the differences in outcome between the 2 groups of athletes.

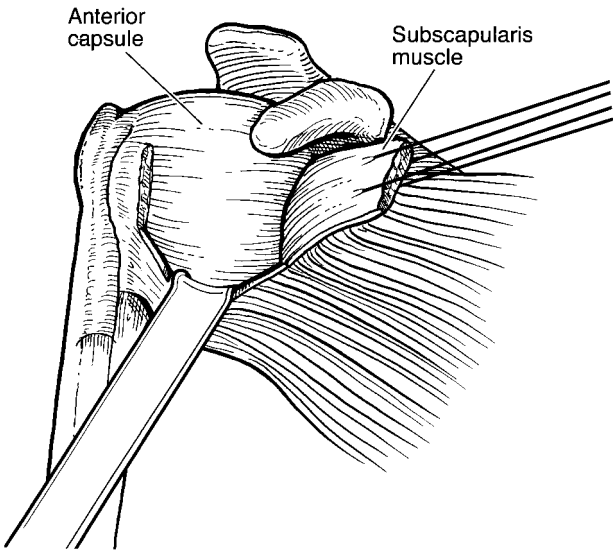

FIGURE 10. Management of the subscapularis: detatch the superior half of the tendon, then develop a plane between the inferior half of the subscapularis and the capsule.

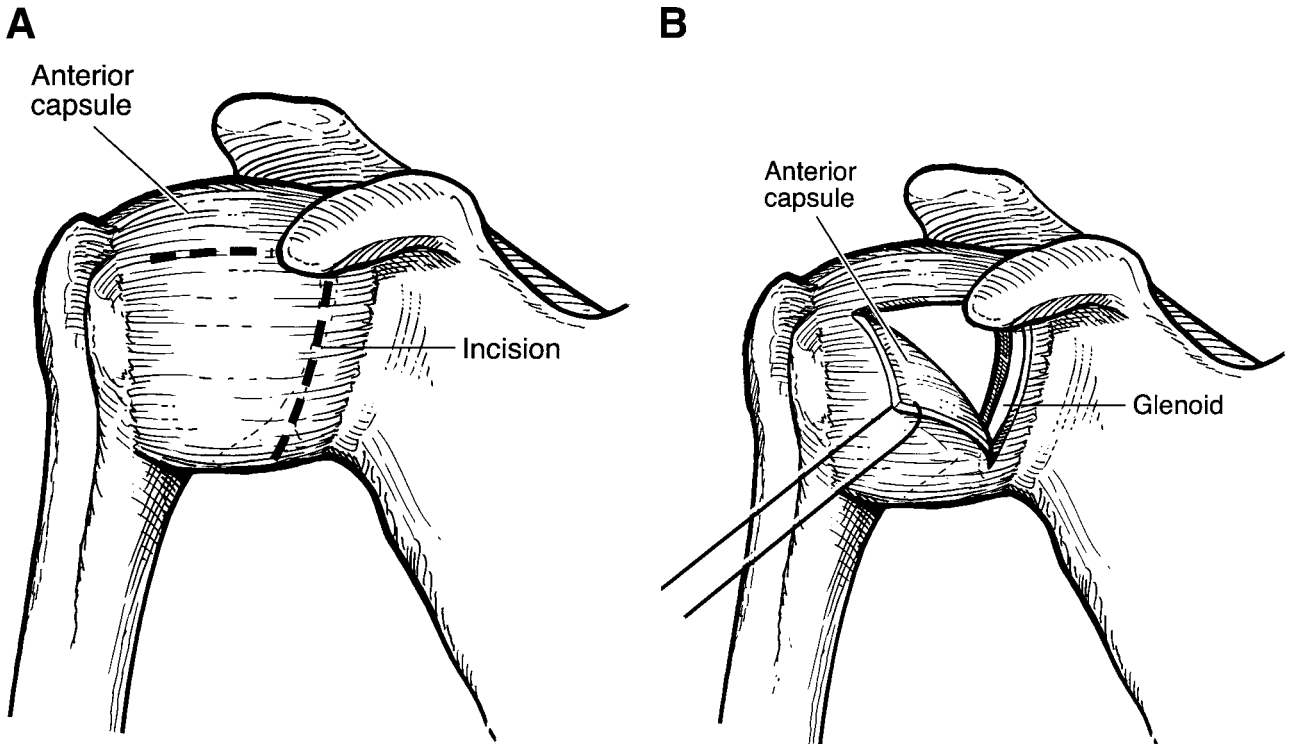

FIGURE 11. (A) Outline of capsulotomy. (B) Dissect the capsule medially as far as possible before detaching it from the glenoid neck to preserve as much capsular length as possible for later reattachment.

In examining the mechanism of injury, one must look at the differences in shoulder position at skill positions between the 2 sports. In American football, the running backs generally have their arms held close to their bodies and seldom sustain dislocations. Many of the dislocations occur in defensive players making arm tackles or sustaining awkward falls to the ground with the arm in abduction and external rotation. On the other hand, rugby players who carry the ball in one

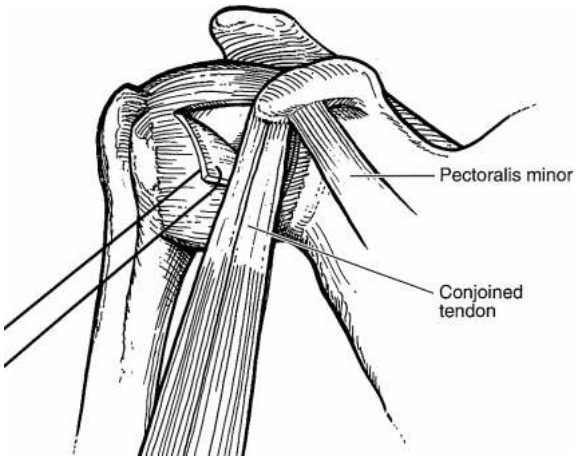

FIGURE 12. Tendon attachments onto the coracoid before coracoid osteotomy.

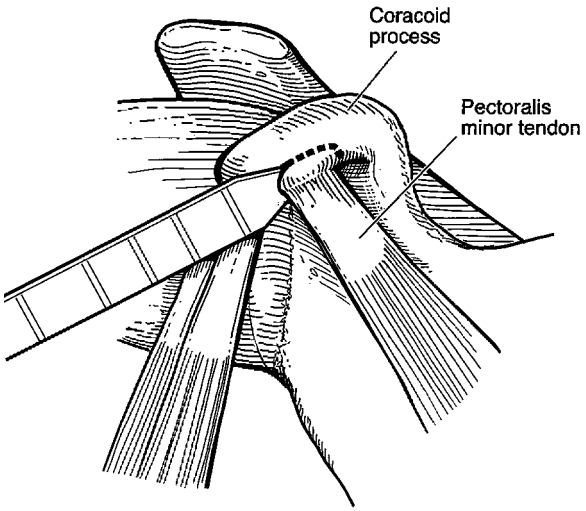

FIGURE 13. Pectoralis minor (insertion at dotted line) attachment is removed from the coracoid so that the osteotomy can be made proximal to the "elbow" of the coracoid.

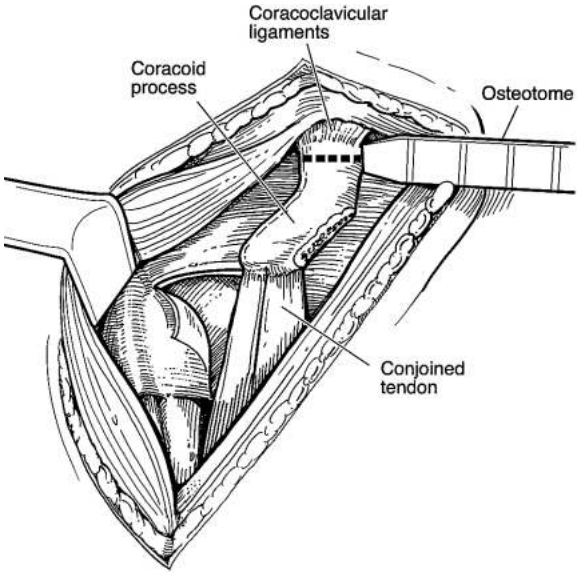

FIGURE 14. Osteotomy of coracoid. Note that the osteotomy is made proximal to the "elbow" of the coracoid. The osteotomy must not angle too steeply toward the glenoid, or it may create an intra-articular glenoid fracture. From a technical standpoint, it is much easier to make the osteotomy at the proper angle if the surgeon removes the medial retractor and allows the osteotome itself to rest on the skin edge and act as its own retractor.

arm will generally "stiff-arm" the opposition with the other arm. The arm used to stiff-arm the defenders is the one that is typically dislocated, and this occurs with an axially directed force while the shoulder is in about 70° of abduction and 30° of extension. In this position, the vector of the axially directed force is brought to bear on the anterior glenoid rim, causing an intra-articular fracture (bony Bankart lesion) rather than soft tissue disruption from bone. In contrast, the American football injuries are generally rotational injuries to the shoulder in which obliquely directed forces applied to the hand or arm produce significant moments and torsional effects at the shoulder that tend to peel the glenohumeral ligaments from the anterior glenoid.

Comparing the rate of failure of arthroscopic Bankart repairs among the categories of contact athletes, the most striking finding is the vastly higher failure rate for those with significant bone defects (89% recurrence rate) compared with those without significant bone defects (6.5% recurrence rate). The American football players, none of whom had any significant bone defects, had no recurrences. This experience with contact athletes suggests that the conventional wisdom of making repairs on all contact athletes by open means is overly simplistic. Based on the athletes studied in this series, our data indicate that those athletes without significant bone defects who have arthroscopic Bankart repairs are not at any greater risk than the noncontact athletes for failure of the repair ($P = .156$).

The group of contact athletes particularly at risk of recurrence is the group with significant bone defects, which in our study was comprised mainly of rugby players. In this group, we recommend the Latarjet reconstruction for inverted-pear glenoid defects, and we recommend the open capsular shift procedure for patients with engaging Hill-Sachs lesions.

The Folly of Overconstraining the Soft Tissues

Based on our experience with this group of patients, we do not understand the current emphasis on greater soft tissue constraint to "improve" the results of arthroscopic Bankart repairs. The methods may vary (thermal capsular shrinkage, adjunctive capsular plication, rotator interval closure), but the goal of these adjunctive procedures is the same—to reduce the recurrence rate by limiting the extremes of motion.

Baker et al.[24] have arthroscopically observed intrasubstance capsular tearing without Bankart lesions in some first-time dislocators. In addition, Bigliani et al.[25] have reported tensile failure of the inferior glenohumeral ligament in which ligament disruption is preceded by ligament stretching, and they suggest the possibility of residual laxity due to plastic deformation resulting from stretching. However, to our knowledge, there are no clinical or basic science studies proving

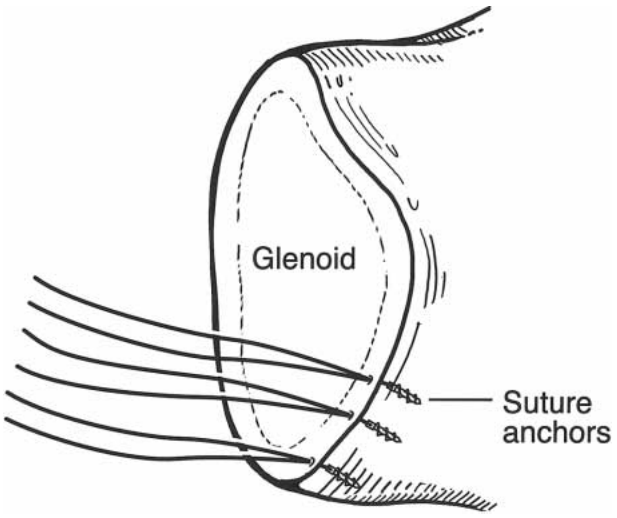

FIGURE 15. Suture anchors are placed at the 3, 4, and 5 o'clock positions for later reattachment of the capsulolabral complex.

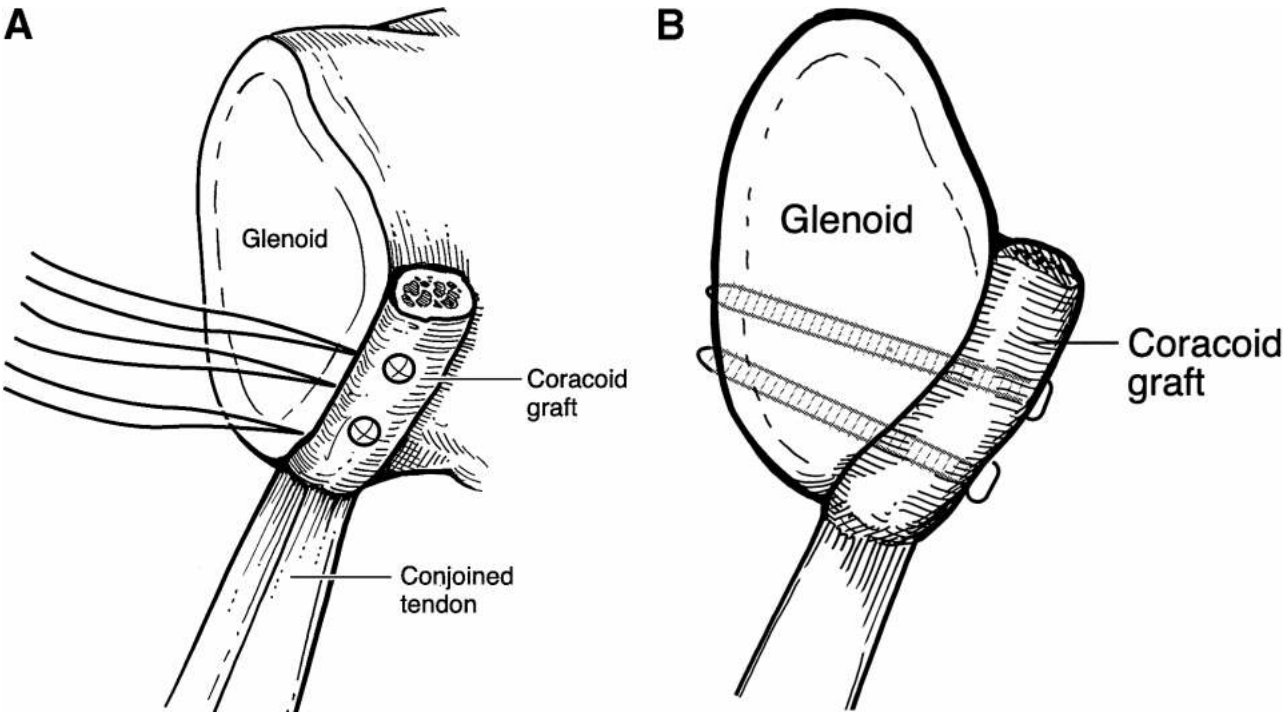

FIGURE 16. (A) Coracoid graft is fixed to glenoid with 2 bicortical screws. If need be, the graft can be contoured with a power burr to fit the curve of the anterior-inferior glenoid. (B) Note how the coracoid graft restores the pear shape of the glenoid by widening its inferior diameter.

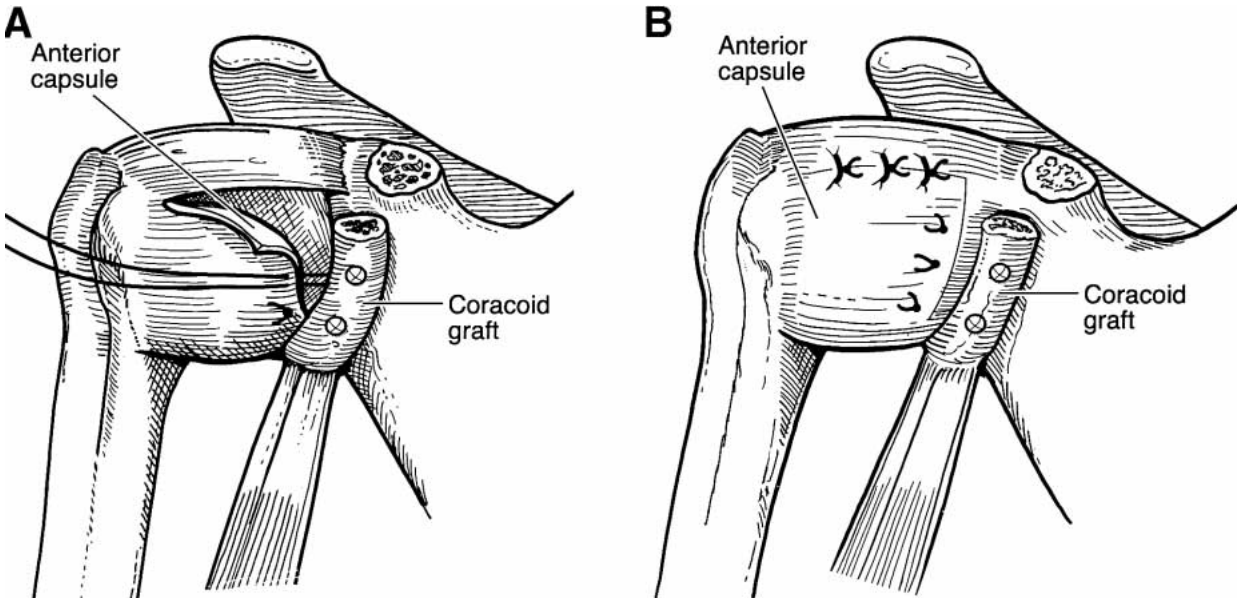

FIGURE 17. (A) The capsule is repaired to the glenoid by means of the previously placed suture anchors. (B) Completed capsular repair with graft in place. Note that the coracoid graft is extra-articular.

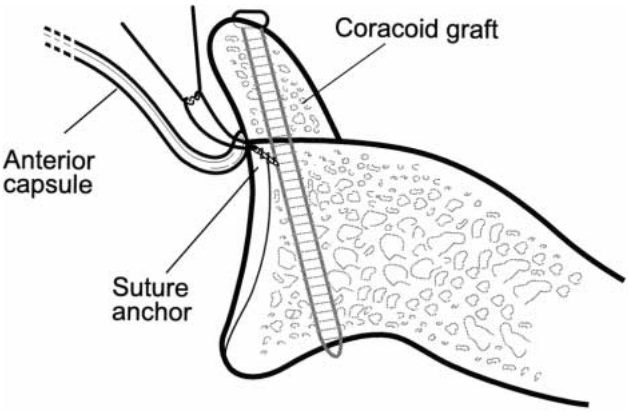

FIGURE 18. The graft is placed so that it becomes an extra-articular platform that acts as an extension of the articular arc of the glenoid.

that plastic deformation ever occurs in patients, much less that it occurs with any regularity.

We are concerned that the current emphasis on maximizing and even overtightening soft tissue constraints is diverting attention from what we believe is the real culprit in the failure of arthroscopic Bankart repairs—the significant bone defect. We must not penalize patients without bone defects by overconstraining and overtightening their capsular tissues, potentially causing permanent loss of motion, when we should be focusing our efforts on restoring bone stability to the relatively small subgroup with significant bone defects.

The problems with the failure of arthroscopic Bankart repair is not that there is some inherent mechanical inferiority of the arthroscopic repair in comparison with the open repair. The problem is that surgeons have not developed realistic criteria for patient selection based on mechanical support requirements. Engineers would have done this long ago. Why haven't surgeons?

CONCLUSIONS

1. Arthroscopic Bankart repairs produce results equal to open Bankart repairs if there are no significant structural bone deficits (large engaging Hill-Sachs lesions or large bony Bankart lesions).
2. Patients with significant bone deficits as defined in this study are not candidates for arthroscopic Bankart repair.
3. A Hill-Sachs lesion that engages the anterior glenoid rim in a functional position of combined flexion-abduction–external rotation on dynamic arthroscopic examination is a contraindication to arthroscopic repair. Such lesions demand either an open capsular shift procedure (to restrict external rotation and thereby prevent engagement of the Hill-Sachs lesion), or else creation of additional articular arc length by means of a bone graft to the Hill-Sachs lesion. Rotational osteotomy to shift the remaining articular cartilage to a position that effectively creates a more favor-

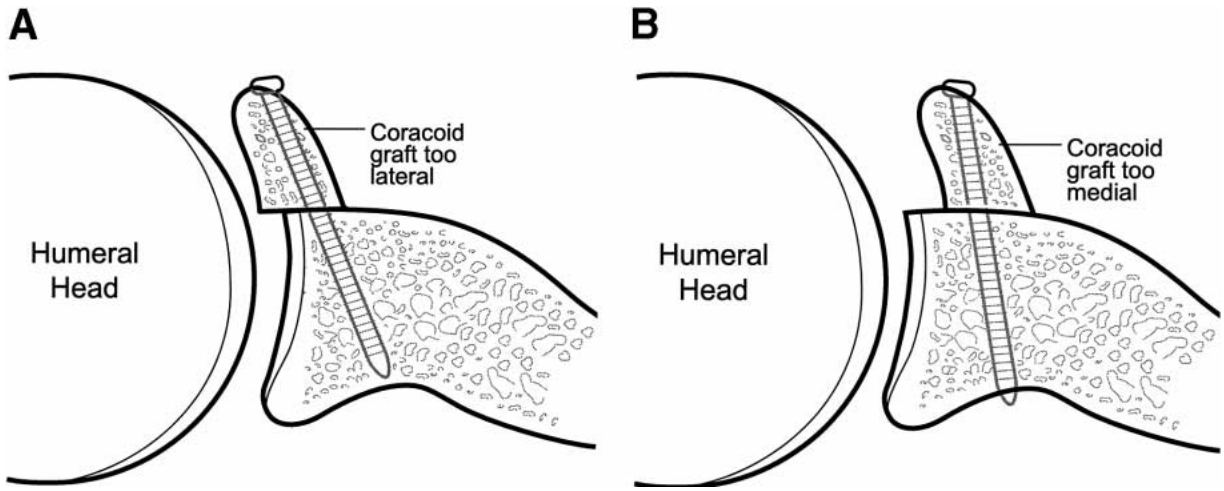

FIGURE 19. Incorrect placement of coracoid bone graft. (A) The graft must not be placed so that it protrudes laterally to the joint surface and acts as a bone block. Such placement produces a high incidence of late osteoarthritis. (B) Conversely, the surgeon must also avoid medial placement of the graft, as this may predispose to recurrent dislocation.

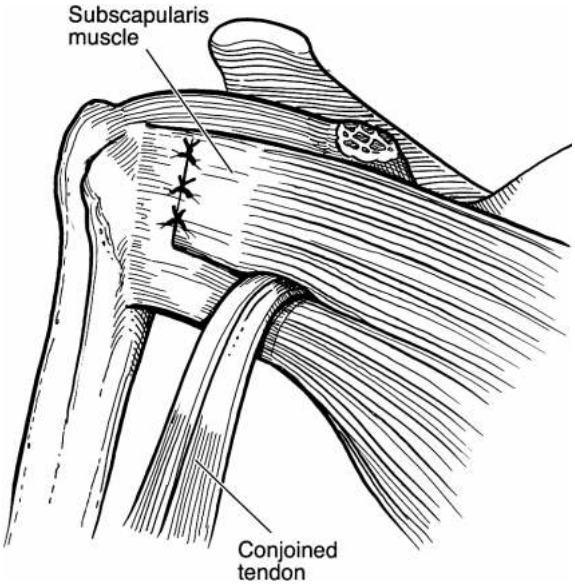

FIGURE 20. The upper half of subscapularis is repaired to its tendon stump. The repaired muscle spans the coracoid bone graft, and the attached conjoined tendon exits anteriorly between the upper and lower subscapularis.

able articular arc would be a final possibility, if all other surgical options had failed.

4. A nonengaging Hill-Sachs lesion, even if it is large, may be successfully treated by arthroscopic repair of the associated Bankart lesion. Such lesions may engage in the nonfunctional position of shoulder extension, or in low ranges (<70°) of shoulder abduction, but will not engage during functional activities.
5. A bony Bankart lesion that narrows the inferior half of the glenoid to a width that is less than that of the superior half of the glenoid (the inverted-pear configuration) is a contraindication to arthroscopic repair. Such lesions demand a bone graft to the anterior-inferior glenoid, and our preference is to use the vascularized coracoid graft of the Latarjet procedure. We find the inverted-pear configuration easier to identify than the 25% glenoid loss advised by Bigliani et al.[22] as a criterion for coracoid transfer.
6. Rugby players seem to be at risk of having significant bone deficiencies in association with anterior dislocation (9 of 21 major bone deficiencies in this series occurred in rugby players). We believe that arthroscopic Bankart repairs are reasonable in contact athletes without bone deficiency because their failure rate is no greater than that of the overall group. However, contact athletes with bone deficiency require open surgery aimed at their specific anatomic deficiencies.
7. This study does not address HAGL lesions.[26] Nonetheless, the surgeon treating contact athletes must be aware that these lesions may be fairly common in contact athletes.[27] Because there is no bone defect with the HAGL lesion, arthroscopic repair is reasonable, although difficult, and our recommendation is for anatomic repair, either open or arthroscopic.

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