The University of Pennsylvania Orthopaedic Journal
14: 5–14, 2001
© 2001 The University of Pennsylvania Orthopaedic Journal

Posterior Instability of the Glenohumeral Joint: Diagnosis and Management

DANIEL S. LAMAR, JR., M.D., GERALD R. WILLIAMS, JR., M.D., JOSEPH P. IANNOTTI, M.D., PH.D., AND MATTHEW L. RAMSEY, M.D.

Abstract: Posterior instability of the glenohumeral joint is being recognized with increasing frequency. The clinical manifestations of this problem most often begin insidiously, as the result of repetitive microtrauma and can be particularly disabling. Although many patients are able to reproduce their instability, diagnosis can be difficult. A detailed physical examination, plain radiographs, and appropriate advanced imaging studies can help identify the pathology. In the past, treatment has realized conflicting and often pessimistic results. These results were likely due to our incomplete understanding of the pathophysiology, the use of non-anatomic surgical strategies and an inconsistent classification system. While non-operative treatment remains the initial option for most clinical scenarios, surgical intervention is frequently necessary. Improvements in our understanding of the pathology through advanced diagnostics, along with improved arthroscopic technology and open surgical techniques have engendered renewed interest in the definitive surgical management of posterior instability.

Introduction

Posterior instability of the shoulder represents the symptoms resulting from excessive posterior glenohumeral translation. Both traumatic and atraumatic origins have been recognized, and diagnosis depends on a clinical history of instability, reproduction of symptoms during physical examination, and an appropriate diagnostic evaluation. Acute posterior dislocation is rare in comparison to its anterior counterpart, accounting for only 5% of all shoulder dislocations. Recurrent posterior subluxation is the most common form of posterior instability and is being recognized with increasing frequency [24,31,76]. Historically, the literature has been unclear regarding the distinction between recurrent posterior subluxation and posterior dislocation, impairing our ability to standardize diagnosis and management guidelines.

McLaughlin recognized the distinction between (locked) posterior dislocation and recurrent posterior subluxation [44]. Attempts to further classify recurrent posterior subluxation have utilized the same terminology as anterior instability, defining instability based on degree (subluxation, dislocation), origin (traumatic, atraumatic), direction (posterior, inferior, multidirectional), and volition (voluntary, involuntary). Describing recurrent posterior subluxation based on a traumatic or atraumatic basis or as voluntary or involuntary does not necessarily define the underlying pathology or assist in treatment decisions. An anatomically based classification of recurrent posterior subluxation, as opposed to the more traditional etiology based methods of classification, facilitates treatment by defining the pathologic process that produces the instability. The salient features of this anatomically based classification system are summarized in Table 1.

From the Penn Orthopaedic Institute, Presbyterian Medical Center, Philadelphia, PA.
Address correspondence to: Matthew L. Ramsey, M.D., Penn Orthopaedic Institute, Presbyterian Medical Center, One Cupp Pavilion, 39th and Market Streets, Philadelphia, PA 19104.

Acute posterior dislocation

Acute posterior dislocations account for approximately 5% of all dislocations. Direct trauma to the front of the shoulder, a posteriorly directed force on an adducted arm (fall on an outstretched hand), and indirect muscle forces (seizure and electrical shock) can all cause posterior dislocation.

Diagnosis

The importance of a thorough clinical assessment cannot be overstated given that despite the routine use of radiographic evaluation, nearly half of all posterior dislocations are missed. Patients with posterior dislocation typically present with more pain than patients with other types of dislocation and the arm is usually splinted at the side in adduction and internal rotation. The classic signs of posterior dislocation are limited external rotation, limited forward elevation, void in the anterior aspect of the shoulder with prominence of the coracoid process, and fullness of the posterior aspect of the shoulder. A thorough neurovascular examination both before and after shoulder reduction is essential.

Radiographic evaluation for a suspected posterior dislocation must include anteroposterior (AP), scapular lateral, and axillary views of the shoulder. These views are generally sufficient to diagnose the position of the humeral head and any associated bony lesions. The classic radiographic features of posterior dislocation include humeral head overlap with the glenoid rim on a true AP, an empty glenoid on the axillary view, fracture of the lesser tuberosity, and a reverse Hill-Sachs lesion. Computed tomography (CT) can be helpful when a satisfactory trauma series is difficult to obtain or interpret.

Table 1. Classification of posterior instability

Posterior Dislocation
Acute posterior dislocation
Chronic (locked) posterior dislocation
Recurrent posterior subluxation
Volitional
Psychogenic
"learned"
Dysplastic
Glenoid retroversion
Humeral head retrotorsion
Acquired
Soft tissue deficiency
Bony deficiency
Scapulothoracic dysfunction

Treatment

The management of an acute posterior dislocation requires care in order to avoid further damage to the humeral head. Forceful reduction attempts in the face of a locked dislocation risk fracturing the humeral head. Closed reduction can be attempted if the reverse Hill-Sachs lesion involves 40% or less of the humeral head. The reduction maneuver involves flexion to 90 degrees with gentle adduction to disimpact the humeral head from the glenoid rim. Lateral traction can assist in disimpaction, while gentle pressure on the humeral head guides the humeral head into the glenoid fossa. Once the humeral head clears the glenoid, the arm is externally rotated and brought down to the side. External rotation should not be attempted until the humeral head has cleared the glenoid rim. The arm is immobilized in a brace in slight abduction and neutral to slight external rotation for four to six weeks.

Volitional recurrent posterior subluxation

Voluntary recurrent posterior subluxation describes a group of patients with an underlying conscious or unconscious ability to subluxate their shoulder using abnormal patterns of muscular activity. In this group of patients there is no initial anatomic pathology in the glenohumeral joint. Over time, stretching of the glenohumeral ligaments can occur such that an involuntary component to the instability develops. Rowe identified these patients as habitual dislocators [60]. Habitual dislocators are distinguished from other patients with posterior subluxation by their willful desire to subluxate their shoulders (Fig. 1). Despite the best intentions of the treating physician, habitual dislocators will frustrate all treatment efforts (operative and non-operative) because of their abnormal psychological need to subluxate their shoulder [60]. The overwhelming pathologic process in this group of patients is psychological and treatment should be directed accordingly. Surgical intervention in this group is contraindicated.

A second group of patients can voluntarily reproduce their instability but have no underlying psychological need to do so. This is a learned behavior that over time may develop an involuntary component. It is this involuntary component that is bothersome to the patient and often initiates evaluation by a physician. Electromyographic evaluation of patients who can voluntarily subluxate their shoulders demonstrates selective inhibition of certain muscle groups, which results in an unbalanced force couple, leading to posterior subluxation. Activation of the deltoid and pectoralis major without opposition from the short posterior rotators was identified in several patients resulting in the humeral head being pushed posteriorly [60]. Conversely, Pande demonstrated unopposed activation of the posterior short rotators and posterior deltoid that in effect pulls the humeral head posteriorly [56]. Treatment of these patients should be based on the anatomic pathology responsible for the involuntary component of the problem.

Dysplastic recurrent posterior subluxation

Dysplastic bony architecture of the glenohumeral joint is another uncommon cause of recurrent posterior subluxation [16]. Localized posterior glenoid hypoplasia, increased glenoid retroversion, and increased humeral head retrotorsion are all potential causes of recurrent posterior subluxation. Recent investigations have documented a low incidence of abnormal bony architecture in patients with instability and have postulated that developmental bony deformities are rare causes of recurrent posterior subluxa-

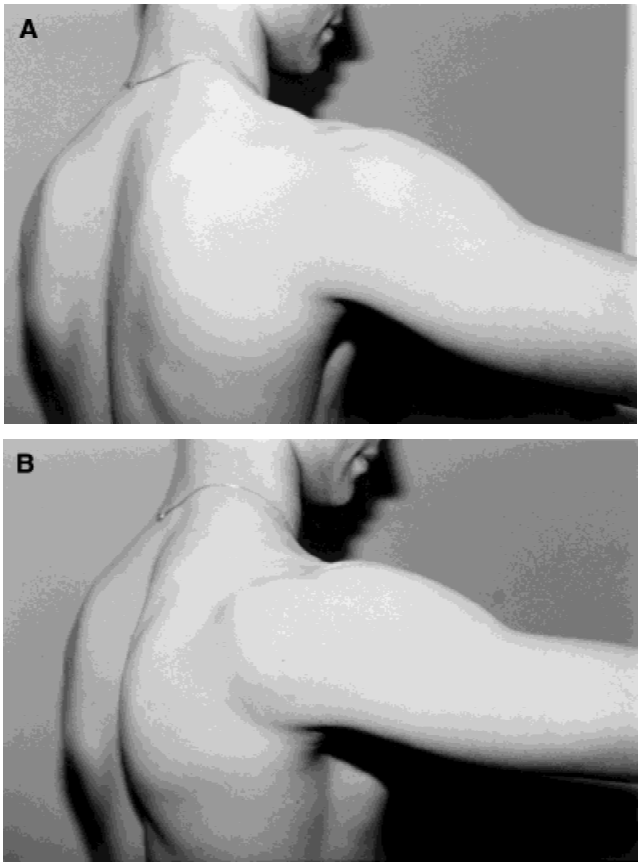

Fig. 1. Photograph of a patient before (**A**) and after (**B**) dislocating his shoulder posteriorly with asymmetric muscular contraction.

tion [25,59,78]. Edelson recently reported the incidence of posterior glenoid dysplasia in over 11,000 cadaveric specimens studied. As many as 35% of the specimens had deficiencies in the posterior-inferior aspect of the glenoid [16].

In the past, the theory that increased glenoid retroversion contributed to recurrent posterior subluxation was supported by radiographic techniques that indirectly measured glenoid retroversion and are now felt to be inaccurate [17]. The advent of computed tomography (CT) has allowed direct measurement of glenoid geometry and has renewed interest in this area. Recent studies based on CT scan assessment of glenoid version vary widely on the incidence of abnormal glenoid geometry and its contribution to instability. Gerber and Randelli found no correlation between altered glenoid version and instability [25,59]. Conversely, Hurley and Wirth have separately demonstrated increased glenoid retroversion and isolated posterior glenoid hypoplasia, respectively, in all patients with recurrent posterior subluxation reported in their series [34,79]. While the incidence of increased glenoid retroversion or hypoplasia in patients with recurrent posterior subluxation is confused by these conflicting reports, it is clear that in some cases it can contribute to recurrent posterior instability [78].

Acquired recurrent posterior subluxation

The largest group of patients with recurrent posterior subluxation acquires posterior instability as a result of repetitive microtrauma or from a single traumatic event. Since the etiology of posterior instability is not as crucial to treatment as the underlying pathologic lesion, we define acquired recurrent posterior subluxation based upon the anatomic lesion. Lesions of the labrum, rotator cuff musculature, and glenoid can contribute to recurrent posterior subluxation with the most consistent deficiency being redundancy of the posterior capsule.

The posterior capsule is thin, and along with the buttress provided by the posterior glenoid labrum, it serves as the primary static stabilizer to unidirectional posterior translation. Posterior labral tears have been described with recurrent posterior subluxation, however, they are generally degenerative tears rather than acute labral avulsions [9,22,30,32]. The relationship between the anterior soft tissues and posterior stability is referred to as the circle concept of capsuloligamentous stability, and a number of biomechanical studies have investigated this idea [47,54,63,64, 68,71,76]. Selective cutting of soft tissue structures thought to contribute to posterior stability has further defined the role of these anterior and posterior soft tissues to static posterior stability. Increased posterior translation has consistently been shown to require a lesion of the posterior capsule, particularly the posterior band of the inferior glenohumeral ligament [54]. Ovesen and Schwartz have demonstrated that isolated sectioning of the posterior rotator cuff musculature in the absence of a capsular lesion did not increase posterior translation [53,63]. A number of studies have found that the superior capsule (rotator interval capsule) plays an important role in posterior stability [28,63, 65]. Harryman and colleagues observed that sectioning the soft tissues of the rotator interval capsule increased posterior and inferior translation often to the point of dislocation. Imbrication of the rotator interval increases resistance to posterior and inferior translation [28].

A requisite for shoulder stability is that scapulothoracic and glenohumeral rhythm remain synchronous [12,75]. Dysfunction of scapulothoracic rhythm may compromise the stability of the glenohumeral joint [75]. The serratus anterior muscle plays a key role in scapulothoracic rhythm, and paralysis results in scapular winging with a subsequent loss of power in elevation that may influence glenohumeral stability [19]. Warner et al. used Moire topographical analysis to study patients with glenohumeral instability and demonstrated abnormal scapulothoracic mechanics compared to asymptomatic patients [75]. While no patient demonstrated severe scapular winging, the degree of scapulothoracic dysfunction was variable. In patients with glenohumeral instability and lesser degrees of scapulothoracic dysfunction, it is unclear whether instability is the result of altered scapulothoracic mechanics or the cause of it.

Diagnosis

The most important components in diagnosing recurrent posterior subluxation are a meticulous history and physical examination. Patients typically present with complaints of pain or a sensation of the shoulder "coming out" when the arm is placed in a provocative position. The provocative position is variable but usually includes some degree of flexion, adduction, and internal rotation. In the majority of patients, pain is limited to the episodes of subluxation. Persistent pain is unusual and may be associated with rotator cuff or biceps tendonitis and posterior capsule irritation [32,73]. Pain as a predominant complaint is more common in athletes and may indicate a predisposition to capsular or rotator cuff irritation with overuse during athletic activity [22,72,73]. A traumatic event initiating recurrent posterior subluxation is not typical. Most often patients cannot recall an initiating event. Over time, they begin to notice shoulder subluxation with certain activities and can identify risky positions.

The disability associated with posterior subluxation is variable and dependent upon the severity of the symptoms. As a general rule, activities of daily living and simple work activities are not limited by symptoms of recurrent posterior subluxation [30–32]. Participation in sports is generally more troublesome, often requiring modification or complete elimination of the activity. In more severe cases of posterior subluxation, activities of daily living and work may be interrupted [57].

Physical examination

The physical examination is directed at reproducing the patient's symptoms and defining the character of instability. Range of motion is generally normal in patients who have not had prior surgery. Occasionally internal and external

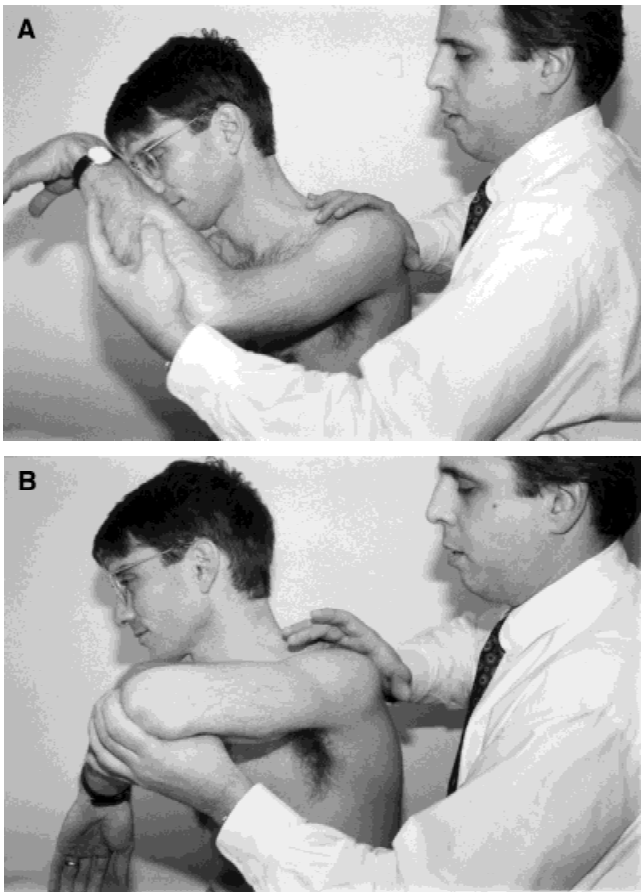

Fig. 2. Seated posterior stress test. The examiner stands to the side of the patient and stabilizes the scapula. With the arm in 90° of forward elevation in the plane of the scapula a posteriorly directed force is applied with the arm in (**A**) external rotation and (**B**) internal rotation. The degree of posterior translation in each position is assessed.

rotation is limited and often in athletes there is activity-specific loss of motion. Overhead throwing athletes may demonstrate increased external rotation with an associated mild loss of internal rotation [72,73]. It is critical to evaluate scapulothoracic function during the physical examination for scapular winging or disruption of normal scapulothoracic rhythm.

The majority of patients can demonstrate their subluxation [31]. Once the position of subluxation is demonstrated by the patient, symptoms of instability can usually be recreated by the physician. In patients who cannot demonstrate their instability, the diagnosis of recurrent posterior instability is more difficult. Testing for posterior subluxation should be performed with the patient in both the sitting and supine positions. It is important to realize that in a normal shoulder, the humeral head can subluxate posteriorly up to 50%, therefore comparison to the unaffected side is critical [27,50]. The posterior stress test is performed with the patient seated and the examiner to the affected side (Fig. 2). The scapula is stabilized while the opposite hand positions the arm in flexion, adduction, and internal rotation, applying a posterior-directed force. With coronal plane extension, the humeral head will relocate into the glenoid fossa. The relocation is sudden and can be felt by the patient and examiner. Most patients with recurrent posterior subluxation have a positive posterior stress test, however apprehension typical of anterior instability is unusual [30,31,57].

The load and shift test should be performed with the patient in the seated and supine positions [43]. In the seated position with the examiner behind the patient, the scapula is stabilized in order to minimize scapulothoracic motion. With the opposite hand, the humeral head is grasped and a centering force is applied. Anterior and posterior translation is assessed and compared to the opposite side. The supine load and shift is particularly useful and is performed with the arm held in 45 to 60 degrees of abduction with varying degrees of rotation from full external to full internal rotation (Fig. 3). Positive testing will result in a reproduction of the patient's symptoms of instability, pain, and crepitation. Side to side differences in the amount of internal rotation necessary to obliterate or minimize posterior translation is a clinical measure of residual pathologic capsular laxity.

Inferior translation is assessed by grasping the elbow with the arm at the side and applying an inferior-directed force. Attention to the region below the acromion will show an

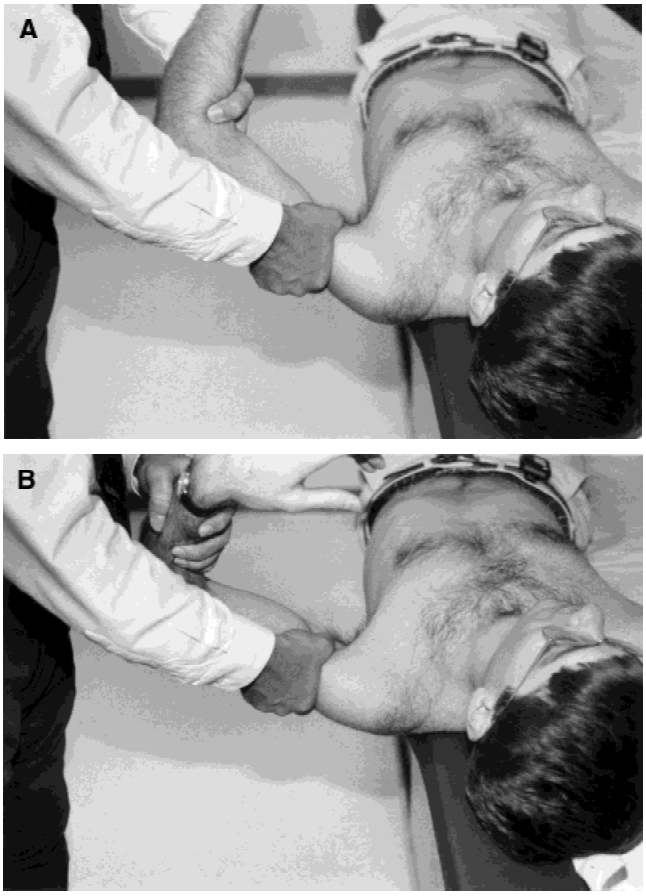

Fig. 3. Supine load and shift test. The patient is supine on the examining table. The arm is brought into approximately 90° of forward elevation in the plane of the scapula. A posteriorly directed force is applied to the humerus with the arm in varying degrees of rotation from (**A**) external rotation to (**B**) internal rotation.

indentation indicating a positive sulcus sign if inferior instability exists [43]. In most normal patients passive external rotation will cause a decrease in the sulcus sign indicating an intact rotator interval capsule (Fig. 4).

The zone of instability is most often posterior-inferior, but can also be straight posterior, or multidirectional. In cases where an isolated posterior component of instability exists, the posterior stress test and load and shift tests demonstrate subluxation at approximately 80 to 90 degrees of forward elevation. In the more common instance of a posterior-inferior instability, subluxation occurs with more forward elevation (110–120 degrees), and frequently a positive sulcus sign exists. Rotator interval capsular insufficiency may be an isolated process or a component of multidirectional laxity. In cases of multidirectional laxity with instability primarily manifested in the posterior-inferior zone, physical signs of generalized ligamentous laxity (i.e., hyperextension of the elbows, knees, metacarpal-phalangeal joints, etc.) are often present. Distinguishing between these cases of isolated posterior, posterior-inferior, and multidirectional laxity with a primarily posterior-inferior component is important in determining treatment options.

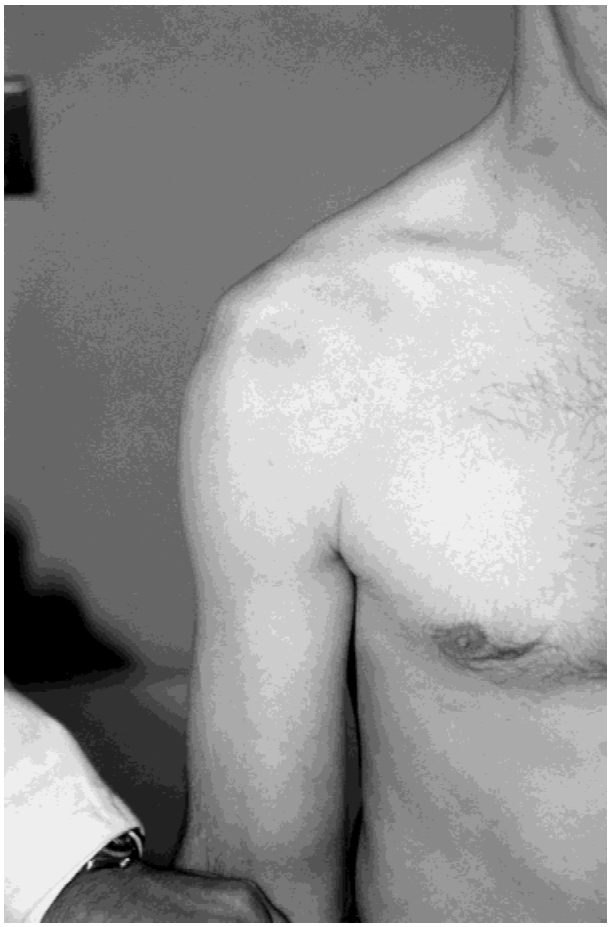

Fig. 4. Sulcus sign. The examiner is seated next to the patient. The forearm is grasped and an inferiorly directed force is applied to the arm in neutral glenohumeral rotation. Attention is directed to the region immediately inferior to the acromion. A positive sulcus sign (**A**) will reduce with external rotation of the shoulder in a patient with a competent rotator interval capsule (**B**).

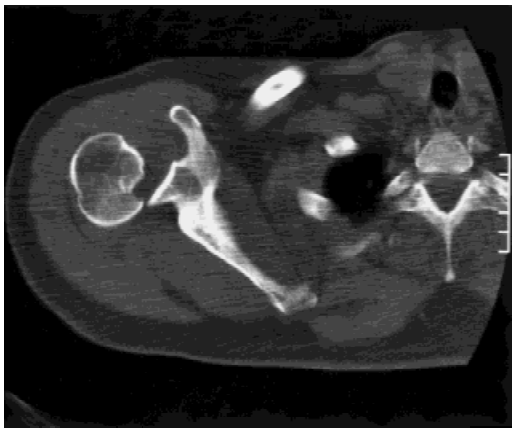

Fig. 5. CAT scan of a large reverse Hill-Sachs lesion.

Imaging studies

Routine radiographs of the shoulder should include the standard three view trauma series. Particular attention should be directed toward the axillary radiograph for evidence of calcification of the posterior capsule, fracture or erosion of the posterior glenoid, or a reverse Hill-Sachs defect (Fig. 5) [22,51,52]. Stress radiographs are generally not necessary.

Advanced imaging studies, although not routinely used, are considered when the specific pathologic lesion underlying recurrent posterior subluxation is unclear. Computed tomography (CT) excels in its ability to define bony detail. If plain radiographs suggest abnormalities of glenoid version, glenoid hypoplasia, or posterior glenoid erosion, CT is useful (Fig. 6) [25,59]. Magnetic resonance imaging (MRI) has improved our ability to assess soft tissue pathology about the shoulder. The advantages of MR imaging over plain radiographs and CT include a lack of exposure to ionizing radiation, excellent soft tissue resolution, and the ability to image in multiple planes. Numerous studies have shown MR imaging to be superior to other imaging studies at defining labral and capsuloligamentous pathology [26,33,35,36,38,66]. Magnetic resonance arthrography (MR arthrography) has been found to be even more sensitive at detecting labral pathology than MR imaging alone [10,20]. Distention of the joint with contrast affords better visualization of the glenoid labrum and glenohumeral ligaments (Fig. 7) [10].Advanced imaging techniques have evolved rapidly and afford better definition of intraarticular pathol ogy compared to standard radiographic techniques. How ever, the information obtained can be misleading if not considered in context with the history and physical examina tion. These studies should be used to confirm the presence of suspected, specific pathologic lesions rather than as a screening tool.

Treatment

A diagnostic work-up along with a treatment algorithm for recurrent posterior subluxation is summarized in Figure 8.

Non-operative

The recommended initial treatment by most authors for symptomatic recurrent posterior subluxation is non-

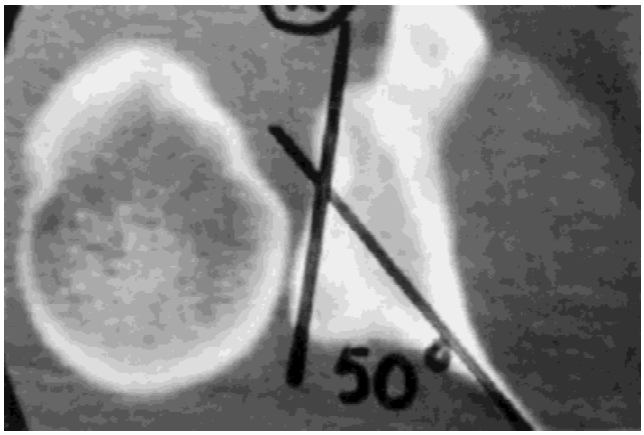

Fig. 6. CT scan of a patient with posterior glenoid hypoplasia with increased glenoid retroversion.

operative [7,22,31,42,52,55,57,73]. Non-operative treatment should include activity modification as well as a shoulder strengthening program for the dynamic muscular stabilizers to include the rotator cuff (especially infraspinatus and teres minor), posterior deltoid, and scapular stabilizing muscles. Activity modification is aimed at preventing further injury to the posterior capsule, labrum, and rotator cuff. Strengthening is accomplished through resisted external rotation exercises with rubber bands of increasing resistance and in time may progress to isokinetic exercises. It is important to balance the strengthening program with internal rotation exercises as well as periscapular strengthening in order to reestablish synchronous scapulohumeral rhythm [45].

Surgical options and rationale for surgical treatment

Patients who fail a prolonged trial of non-operative therapy and remain symptomatic should be considered for surgical stabilization directed at the underlying pathology. Numerous posterior procedures have been described for recurrent posterior subluxation. These procedures include posterior capsulorrhaphy with or without a bone block, posterior bone block alone, glenoid osteotomy, posterior infraspinatus capsular tenodesis, and posterior-inferior capsular shift [1,2,5,6,21,22,37,39,44,46,47,49,52,57,65]. Historically, the results of surgery have been poor with recurrence rates of up to 50% with significant complications [31,72]. An inadequate preoperative understanding of the clinically significant pathology contributed to these poor results. Recently, surgical techniques have been employed which anatomically correct the underlying pathology resulting in more encouraging results [3,22,31,72]. Consequently, the importance of accurately defining the pathologic lesion preoperatively cannot be overstated. The specific surgical techniques are determined by the underlying pathology in each particular case and are summarized in Table 2.

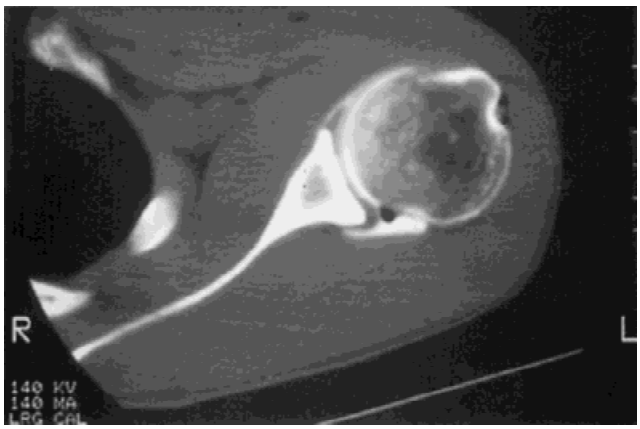

Fig. 7. CT arthrogram of the shoulder. Intra-articular contrast improves visualization of the glenoid labrum.

A patulous posterior capsule is commonly the primary pathologic lesion. If a reverse Bankart lesion coexists with a redundant posterior capsule, combined repair of the reverse Bankart lesion and posterior capsular plication is necessary. When posterior capsular redundancy or detachment is combined with erosion of the posterior glenoid rim or increased glenoid retroversion, a posterior capsular procedure is combined with posterior glenoid bone graft or posterior opening wedge osteotomy. Posterior bone block procedures may also be indicated for failed capsular plication, even if the glenoid architecture is normal.

It is critical to determine the direction of instability prior to surgery. If instability is only posterior, a procedure addressing the redundant posterior capsule is warranted. Posterior subluxation is often accompanied by varying degrees of inferior and multidirectional laxity. In cases of posterior-inferior subluxation with no anterior component and a functionally intact rotator interval, a posterior-inferior capsular shift from a posterior approach should be performed. In patients with multidirectional laxity, the surgical approach depends on the primary location of their symptoms and in the rare patient with both anterior and posterior symptoms, combined anterior and posterior capsular procedures are performed.

The importance of restoring scapulothoracic mechanics in controlling posterior instability is often understated. Scapular winging can be an important contributor to recurrent posterior subluxation. A pectoralis major transfer, alone or in combination with posterior capsulorrhaphy, may be indicated when posterior subluxation and a long thoracic nerve injury coexist [3,22,31,58,72]. Determining the need for posterior capsulorrhaphy in addition to the pectoralis major transfer is often difficult, and in most instances a pectoralis major muscle transfer alone is sufficient to correct the posterior instability.

In the presence of glenoid hypoplasia or increased glenoid retroversion, a glenoid osteotomy should be considered [65]. Humeral rotational osteotomy remains a controversial procedure and is only entertained when there is documented abnormal humeral retrotorsion [11,70].

Patients with a psychological cause for recurrent posterior subluxation should be managed with an exercise program combined with psychological counseling [60]. Surgery should only be considered after their underlying psychological problems are resolved and the patient still demonstrates symptomatic involuntary recurrent posterior

subluxation. Extreme caution is warranted in performing surgery in this group of patients. If any element of psychogenic posterior subluxation remains, surgery is doomed to failure.

Post-operative management after posterior shoulder surgery requires the use of a thoracobrachial orthosis. Prior to surgery patients are fitted for the orthosis with the arm positioned in slight abduction, posterior to the coronal plane of the body and in neutral to 10° of external rotation. The pre-fitted orthosis is applied in the operating room at the conclusion of the surgical procedure.

Immobilization is maintained for a 4–6 week period and is based on the degree of passive motion measured four weeks post-operatively. If the shoulder demonstrates an inability to internally rotate past the neutral position, the brace is discontinued at four weeks. The arm is then kept in a sling for an additional two weeks during which time the patient starts a gentle exercise program. Patients with generalized ligamentous laxity are generally treated with a brace for six weeks. Following the period of immobilization, the patient starts active assisted, supine forward flexion, external rotation, and internal rotation exercises. At 6–8 weeks cross body adduction, active range of motion, and light weight or theraband resistive exercises are started. Progressive stretching and strengthening begins at 12 weeks post-operatively. Total rehabilitation time is usually 16–24 weeks for primary capsulorrhaphy. Revision surgery and complex situations including scapulothoracic reconstruction may require longer time periods for rehabilitation.

Results

The lack of a universally accepted and applied classification system for posterior instability makes interpretation of treatment results presented in the literature difficult.

Table 2.

Procedure	Indication
Posterior capsulorrhaphy	Unidirectional posterior instability
Posterior labral repair	Unidirectional posterior instability with reverse Bankart lesion
Posterior bone block	Unidirectional posterior instability with posterior glenoid erosion or insufficiency Failed soft tissue procedure
Posterior opening wedge glenoid osteotomy	Posterior instability associated with increased glenoid retroversion or localized glenoid hypoplasia
Anterior inferior capsular shift with rotator interval plication	Posterior-inferior subluxation, with underlying multi-directional laxity

Success in treating recurrent posterior subluxation of the shoulder using a non-operative exercise program has been shown to be dependent on the amount of disability the patient is experiencing at the time of presentation. Fronek et al. reports a 63% success rate using non-operative measures in patients with moderate disability when performing strenuous activities at presentation [22]. Hurley et al. demonstrated similar improvement in 68% of the patients treated under a similar treatment protocol [34]. Success in both of these clinical investigations was defined as clinical improvement that satisfied the patient to the extent that no further treatment was required. These patients often demonstrate persistent posterior instability, however, the involuntary subluxations that prompted them to seek treatment are significantly improved. These studies combined with the mixed results of operative intervention warrant the inclusion of non-operative measures as an initial form of treatment in any algorithm.

At first glance, the results of the surgical treatment of recurrent posterior subluxation are discouraging. Some authors have even concluded that recurrent posterior subluxation should not be treated surgically [31]. A more detailed examination of the literature shows cause for guarded optimism. Historically, the confusion regarding the classification of posterior instability combined with a poor understanding of the underlying pathophysiology, and the routine use of surgical procedures that failed to address that pathology resulted in high surgical failure rates.

The surgical management of recurrent posterior subluxation can be divided into soft tissue and osseous procedures. Since the cause of posterior instability usually resides in the posterior soft tissue structures, repairs aimed at reinforcing these deficient structures are most commonly employed. Fronek and colleagues reported on 24 patients with isolated posterior subluxation treated with posterior capsulorrhaphy with a 91% success rate [22]. The capsular repair was reinforced by the infraspinatus tendon and a posterior bone block if the posterior soft tissues were deficient. Hawkins also favors utilization of the infraspinatus tendon to reinforce the capsular repair posteriorly and reports an 85% success rate [29,32]. In 1980, Neer and Foster introduced the inferior capsular shift in patients where there are inferior and posterior components to the instability [47]. Bigliani reported the early results with this procedure in 25 patients with recurrent posterior subluxation with 88% satisfactory results [2]. Pollock reported longer-term follow-up of this procedure with an overall satisfactory rate of 80% [57]. Excluding revision cases, the success rate improved to 96%, highlighting the importance of meticulous soft tissue repair at the index procedure.

While labral detachment from the posterior glenoid rim is rare in recurrent posterior subluxation, a number of authors have successfully treated recurrent posterior subluxation with an anatomic labral repair in the presence of a reverse Bankart lesion. Rowe and Yee performed reverse Bankart repairs on two patients with recurrent posterior subluxation with no recurrence of instability [61].

Several non-anatomic procedures have been previously

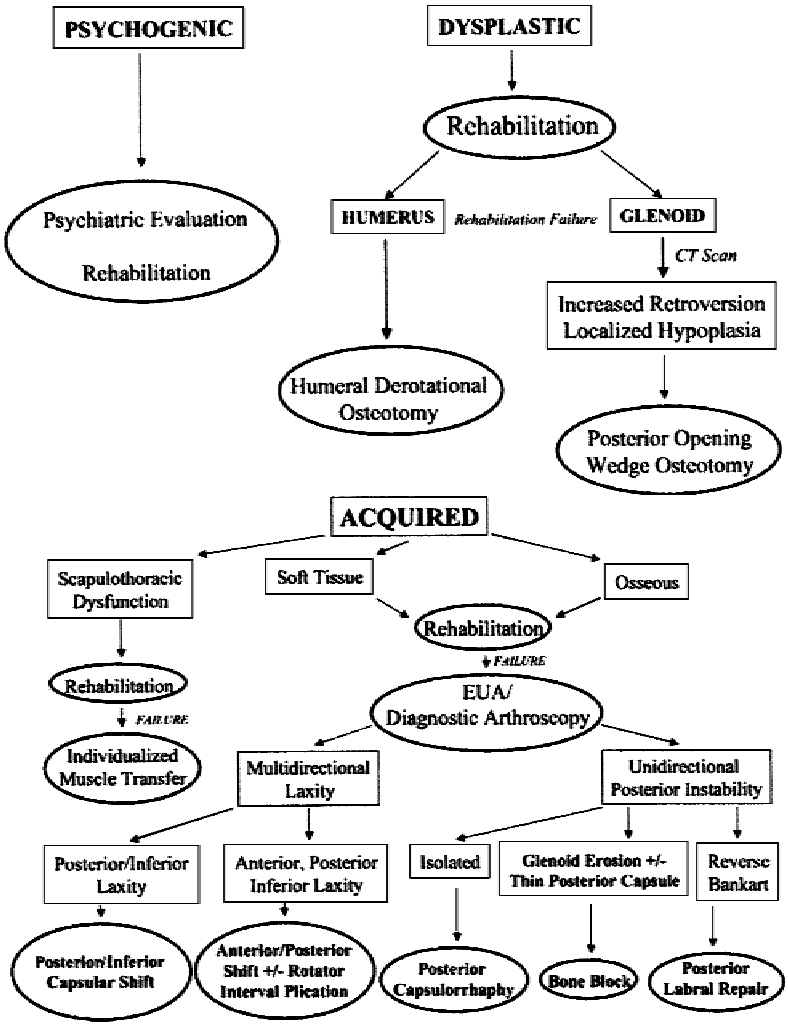

Fig. 8. University of Pennsylvania treatment algorithm for recurrent posterior subluxation. (**A**) Psychogenic and dysplastic recurrent posterior subluxation and (**B**) acquired posterior subluxation.

described with mixed results. Among these is the reverse Putti-Platt operation. Dugas et al. treated 18 patients with recurrent posterior subluxation with a reverse Putti-Platt procedure [15]. Satisfactory results were obtained in 16 of 17 patients available for follow-up. Six patients experienced mild loss of motion. Similarly, Hawkins reported good results in his patients undergoing this procedure [31]. Boyd and Sisk reported nine non-anatomic soft tissue repairs that were augmented by posterior transfer of the long head of the biceps tendon [5]. All patients reportedly did well without recurrence of instability.

The second group of procedures for recurrent posterior subluxation addresses bony pathology by either redirecting abnormal osseous anatomy (glenoid osteotomy or proximal humerus rotational osteotomy) or by augmenting deficient glenoid bone stock or incompetent posterior soft tissues (bone block procedures). Glenoid osteotomy (glenoplasty), first reported by Scott, is a posterior opening wedge osteotomy of the glenoid neck with interposition of bone graft, thereby redirecting the glenoid more anteriorly [65]. In his original report of three cases, one patient dislocated anteriorly in the early postoperative period and another had recurrent posterior subluxation. Norwood and Terry reported 19 patients with recurrent posterior subluxation from various causes treated by glenoid osteotomy [52]. Three patients (16%) continued to experience isolated posterior instability, 4 patients (21%) developed isolated anterior instability, and 2 patients (12%) had multidirectional instability in the postoperative period. English and Macnab advocated an anatomic approach to recurrent posterior subluxation [18]. The surgical results of eight patients were reviewed. All demonstrated increased glenoid retroversion on preoperative radiographs. Four of eight patients were treated with posterior glenoid osteotomy without recurrence. More recently, glenoid osteotomy has been successfully employed in patients with localized posterior glenoid hypoplasia and recurrent posterior subluxation [79].

In spite of these results, the role of posterior glenoid opening wedge osteotomy in the treatment of recurrent posterior subluxation is open to many criticisms. Gerber has documented coracoid impingement following posterior glenoid osteotomy [25]. There has been a spectrum of anterior instability reported following glenoid osteotomy that ranges from coracoid impingement to anterior dislocation [52,65]. Furthermore, posterior glenoid osteotomy is a technically demanding procedure with the potential for significant complications. Hawkins reported a 41% complication rate with this procedure including subsequent glenohumeral

arthritis in two patients [31]. Finally, earlier studies that justify glenoid osteotomy based on radiographic evidence of increased glenoid retroversion may have overstated this problem [17]. The ability to document increased glenoid retroversion by plain radiographs has been questioned. Currently, CT scan is the most accurate method of determining glenoid version.

Increased proximal humeral retrotorsion has been implicated as a cause for recurrent posterior subluxation. Rotational osteotomy has been used to treat recurrent posterior subluxation based on the assumption that increased humeral retrotorsion contributes to posterior instability. The relationship between humeral retrotorsion and posterior instability has not been established. By limiting internal rotation through proximal humeral rotational osteotomy, it was theorized that posterior instability would subside. Surin reported 12 cases of recurrent posterior instability treated with external rotation osteotomy of the proximal humerus [70]. One patient had pain after osteotomy that was attributed to anterior impingement. A second patient developed recurrent instability. The majority of patients had significant restriction of external rotation postoperatively.

Another group of bony procedures act to buttress the posterior glenoid with bone graft from the iliac crest or spine of the scapula. Several authors have reported the use of posterior bone block procedures for recurrent posterior instability [1,37]. Ahlgren and colleagues treated five patients with a posterior bone block procedure [1]. Two of five patients had normal shoulders postoperatively while three demonstrated varying degrees of recurrent posterior instability. More commonly, posterior bone block procedures are combined with a posterior capsulorrhaphy or performed for failed posterior soft tissue procedures [37,46,74].

Conclusions

Recurrent posterior subluxation is less common than anterior subluxation, but is being diagnosed with increasing frequency. The treatment of recurrent posterior subluxation is dependent upon the underlying pathology. An anatomically based classification system facilitates the diagnosis and an appropriate treatment plan. The earlier literature on surgical treatment has yielded inconsistent results, but more recently, improved recognition of the underlying pathology coupled with more anatomic surgical approaches has resulted in consistently improved results.

References

1. Ahlgren S, Hedlund T, Nistor L. Idiopathic posterior instability of the shoulder joint: Results of operation with posterior bone graft. *Acta Orthop Scand* 49:600–3, 1978.
2. Bigliani LU, Endrizzi DP, McIlveen SJ, Flatow EL, Dalsey RM. Operative management of posterior instability. *Ortho Trans* 13:232, 1989.
3. Bigliani LU, Pollock RG, Endrizzi DP, McIlveen SJ, Flatow EL: Surgical repair of posterior instability of the shoulder: Long term results. 9th combined Meeting of the Orthopaedic Associations of the English-Speaking World, Toronto, 1992.
4. Bowen MK, Warren RF, Altchek DW, O'Brien SJ. Posterior subluxation of the glenohumeral joint treated by posterior stabilization. *Ortho Trans* 15:764, 1991.
5. Boyd HB, Sisk TD. Recurrent Posterior Dislocation of the Shoulder. *J Bone Joint Surg* 54A:779–86, 1972.
6. Brewer B, Wubben RC, Carrera GF. Excessive retroversion of the glenoid cavity. A cause of non-traumatic posterior instability of the shoulder. *J Bone Joint Surg* 68A:724–31, 1986.
7. Burkhead W, Ca R. Treatment of instability of the shoulder with an exercise program. *J Bone Joint Surgery* 74A:890–6, 1992.
8. Callaghan JJ, McNeisk LM, Dehaven JP, Savory CG. A prospective comparison study of double contrast CT, Arthrography, and Arthroscopy of the shoulder. *Am J Sports Med* 16:13, 1988.
9. Caspari RB, Geissler WD. Arthroscopic manifestations of shoulder subluxation and dislocation. *Clin Orthop* 291:54–66, 1993.
10. Chandnani VP, Yeager TD, DeBerardino T, et al. Glenoid labral tears: Prospective evaluation with MR imaging, MR arthrography, and CT arthrography. *Am J Radiology* 161:1229–35, 1993.
11. Chaudhuri GK, Sengupta A, Saha AK. Rotation osteotomy of the shaft of the humerus for recurrent dislocation of the shoulder: anterior and posterior. *Acta Orthop Scand* 45:193–8, 1974.
12. Codman EA: Rupture of the supraspinatus tendon and other lesions in or about the subacromial bursa. In: *The Shoulder*. Boston, MA: Thomas Todd; 1934.
13. Cofield RH, Nessler JP, Weinstabl R. Diagnosis of shoulder instability by examination under anesthesia. *Clinical Orthop* 45–53, 1993.
14. Cooper, A. On the dislocation of the os humeri upon the dorsum scapula, and upon fractures near the shoulder joint. *Guy's Hosp Rep* 4:265–84, 1839.
15. Dugas RW, Scerpella TA, Clancy WG. Surgical treatment of symptomatic posterior shoulder instability. *Ortho Trans* 14:245, 1990.
16. Edelson JG. Localized glenoid hypoplasia. *Clin Orthop* 321:189–95, 1995.
17. Engebretsen L, Craig EV. Radiologic features of shoulder instability. *Clinical Orthop* 291:29–44, 1993.
18. English E, Macnab I. Recurrent posterior dislocation of the shoulder. *Canadian J Surg* 17:147–51, 1974.
19. Fery A: Results of treatment of anterior serratus paralysis. In: Post M, Morrey BF, Hawkins RJ, editors. *Surgery of the Shoulder*. St. Louis, MO: Mosby Year Book; p 325–9, 1990.
20. Flannigan B, Kursunoglu-Brahme S, Snyder S, et al. MR arthrography of the shoulder: comparison with conventional MR imaging. *Am J Radiology* 155:829–32, 1990.
21. Fried A. Habitual posterior dislocation of the shoulder joint: A case report on 5 operated cases. *Acta Orthop Scand* 18:329, 1949.
22. Fronek J, Warren RF, Bowen M. Posterior subluxation of the glenohumeral joint. *J Bone Joint Surg* 71A:205–16, 1989.
23. Galinat BJ, Howell SJ, Kraft TA. The glenoid-posterior acromial angle: An accurate method of evaluating glenoid version. *Ortho Trans* 12:727, 1988.
24. Gerber C, Ganz R. Clinical assessment of instability of the shoulder. With special reference to anterior and posterior drawer tests. *J Bone Joint Surg* 66B:551–6, 1984.
25. Gerber C, Ganz R, Vinh T.S. Glenoplasty for recurrent posterior shoulder instability. An anatomic reappraisal. *Clin Ortho & Rel Res* 70–9, 1987.
26. Gross ML, Seeger LL, Smith JB. Magnetic resonance imaging of the glenoid labrum. *Am J Sports Med* 18;229–34, 1990.
27. Harryman DT, Sildes JA, Clark JM, McQuade KJ, Gibb TD, Matsen FA. Translation of the humeral head on the glenoid with passive glenohumeral motion. *J Bone Joint Surg* 72A:1334–43, 1990.
28. Harryman DT, Sidles JA, Harris SL, Matsen FA. The role of the rotator interval capsule in passive motion and stability of the shoulder. *J Bone Joint Surg* 74A:53–66, 1992.
29. Hawkins RJ, Bell RH, Lippitt SB: Instability. In: Hawkins RJ, Bell RH, Lippitt SB, editors. *Atlas of Shoulder Surgery.* St. Louis: Mosby; p 47–113, 1996.
30. Hawkins RJ, Bell RM. Posterior instability of the shoulder. *Instr Course Lect* 211–15, 1989.

31. Hawkins RJ, Koppert G, Johnston G. Recurrent posterior instability (subluxation) of the shoulder. *J Bone Joint Surg* 66A:169–74, 1984.
32. Hawkins RJ, McCormack RG. Posterior shoulder instability. *Orthopedics* 11:101–8, 1988.
33. Huber H, Gerber C. Voluntary subluxation of the shoulder in children. A long-term follow-up study of 36 shoulders. *J Bone Joint Surg* 76B: 118–22, 1994.
34. Hurley JA, Anderson TE, Dear W, Andrish JT, Bergfeld JA, Weiker GG. Posterior shoulder instability. Surgical versus conservative results with evaluation of glenoid version. *Am J Sports Med* 20:396–400, 1992.
35. Iannotti JP, Zlatkin MB, Esterhai JL, et al. Magnetic resonance imaging of the shoulder: sensitivity, specificity and predictive value. *J Bone Joint Surg* 73A:17–29, 1991.
36. Jahnke AH Jr, Petersen SA, Neumann C, Steinbach L, Morgan F. A prospective comparison of computerized arthrotomography and magnetic resonance imaging of the glenohumeral joint. *Am J Sports Med* 20:695–700, 1992.
37. Jones V. Recurrent posterior dislocation of the shoulder: Report of a case treated by posterior bone block. *J Bone Joint Surg* 40B:203–7, 1958.
38. Kieft GJ, Bloem JL, Rozing PM, et al. MR imaging of recurrent anterior dislocation of the shoulder: Comparison with CT arthrography. *Am J Radiology* 150:1083–7, 1988.
39. Kretzler HH. Scapular osteotomy for posterior shoulder dislocation. *J Bone Joint Surg* 56A:197, 1974.
40. Laumann U: Kinesiology of the shoulder joint. In: Kobel R, editor. *Shoulder Replacement*. Berlin: Springer-Verlag; 1987.
41. Liou JTS, AJ W, Totty WG, Brown JJ. The normal shoulder: Common variations the simulate pathologic conditions at MR imaging. *Radiology* 186:435–41, 1993.
42. Matsen FA III: Glenohumeral instability. In: Evarts CM, editor. *Surgery of the Musculoskeletal System*. Vol. 2. New York: Churchill Livingstone; p 349–75, 1983.
43. Matsen FA, Thomas SC, Rockwood CA, Wirth MA: Glenohumeral instability. In: Rockwood CA, Matsen FA, editors. *The Shoulder*. Philadelphia, PA: WB Saunders; p 611–754, 1998.
44. McLaughlin HL. Follow-up notes on articles previously published in the journal: Posterior dislocation of the shoulder. *J Bone Joint Surg* 44A:1477, 1972.
45. Moseley JB Jr, Jobe FW, Pink M, Perry J, Tibone J. EMG analysis of the scapular muscles during a shoulder rehabilitation program. *Am J Sports Med* 20:128–34, 1992.
46. Mowery CA, Garfin SR, Booth RE, Rothman RH. Recurrent posterior dislocation of the shoulder: Treatment using a bone block. *J Bone Joint Surg* 67A:777–81, 1985.
47. Neer CSII, Foster CS. Inferior capsular shift for involuntary inferior and multidirectional instability of the shoulder. *J Bone Joint Surg* 62A:897–908, 1980.
48. Neumann CH, Peterson SA, Jahnke AH. MR imaging of the labral-capsular complex: Normal variations. *Am J Radiology* 157:1015–21, 1991.
49. Nobel W. Posterior traumatic dislocation of the shoulder. *J Bone Joint Surg* 44A:523–38, 1962.
50. Norris TR: C-arm fluoroscopic evaluation under anesthesia for glenohumeral subluxation. In: Bateman JE, Welsh RP, editors. *Surgery of the Shoulder*. Philadelphia, PA: BC Becker, Inc; p 22–5, 1984.
51. Norris TR. Diagnostic techniques for shoulder instability. *Instr Course Lect* 34:239–57, 1985.
52. Norwood LA, Terry G. Shoulder posterior subluxation. *Am J Sports Med* 12:25–30, 1984.
53. Ovesen J, Nielsen S. Anterior and posterior shoulder instability: A cadaver study. *Acta Orthop Scand* 57:324–27, 1986.
54. Ovesen J, Söjbjerg JO. Posterior shoulder dislocation: Muscle and capsular lesions in cadaver experiments. *Acta Orthop Scand* 57:535–36, 1986.
55. Pagnani MJ, Galinat BJ, Warren RF: Glenohumeral instability. In: DeLee J, Drez D, editors. *Orthopaedic Sports Medicine: Principles and Practice.* Vol. 1. Philadelphia, PA: W.B. Saunders; p 580–622, 1994.
56. Pande P, Hawkins R, Peat M. Electromyography in voluntary posterior instability of the shoulder. *Am J Sports Med* 17:644–648, 1989.
57. Pollock RG, Bigliani LU. Recurrent posterior shoulder instability. Diagnosis and treatment. *Clin Orthop* 291:85–96, 1993.
58. Post M. Pectoralis major transfer for winging of the scapula. *J Shoulder Elbow Surg* 4:1–9, 1995.
59. Randelli M, Gambrioli PL. Glenohumeral osteometry by computed tomography in normal and unstable shoulders. *Clin Orthop* 208:151–56, 1986.
60. Rowe CR, Pierce DS, Clark JG. Voluntary dislocation of the shoulder. A preliminary report on a clinical, electromyographic, and psychiatric study of 26 patients. *J Bone Joint Surg* 55A:445–60, 1973.
61. Rowe CR, Yee LBK. A posterior approach to the shoulder joint. *J Bone Joint Surg* 26:580–4, 1944.
62. Schutte JP, Lafayette LA, Hawkins RJ, Chris A. The use of computerized tomography in determining humeral retroversion. *Orthop Trans* 12:727, 1988.
63. Schwartz E, Warren RF, O'Brien SJ, Fronek J. Posterior shoulder instability. *Orthop Clin North Am* 18:409–19, 1987.
64. Schwartz RE, O'Brien SJ, Warren RF, Torzilli PA. Capsular restraints to anterior-posterior motion of the shoulder. *Orthop Trans* 12:727, 1988.
65. Scott DJJ. Treatment of recurrent posterior dislocations of the shoulder by glenoplasty. *J Bone Joint Surg* 49A:471–6, 1967.
66. Seeger LL, Ruskowski JT, Bassett LW, et al. MR imaging of the normal shoulder: Anatomic correlation. *Am J Radiology* 148:83–92, 1987.
67. Shaffer BS, Conway J, Jobe FW, Kvitne RS, Tibone JE. Infraspinatus muscle-splitting incision in posterior shoulder surgery. An anatomic and electromyographic study. *Am J Sports Med* 22:113–20, 1994.
68. Speer KP, Deng X, Borrero S, Torzilli PA, Altchek DA, Warren RF. Biomechanical evaluation of a simulated Bankart lesion. *J Bone Joint Surg* 76A:1819–26, 1994.
69. Stoller DW: *Magnetic Resonance Imaging in Orthopaedics and Sports Medicine.* Philadelphia, PA: Lippincott-Raven; p 511–633, 1997.
70. Surin V, Blader S, Markhede G, Sundholm K. Rotational osteotomy of the humerus for posterior instability of the shoulder. *J Bone Joint Surg* 72A:181–6, 1990.
71. Terry GC, Hammon D, France P, Norwood LA. The stabilizing function of passive shoulder restraints. *Am J Sports Med* 19:26–34, 1991.
72. Tibone J, Ting A. Capsulorrhaphy with a staple for recurrent posterior subluxation of the shoulder. *J Bone Joint Surg* 72:999–1002, 1990.
73. Tibone JE, Bradley JP. The treatment of posterior subluxation in athletes. *Clin Orthop* 291:124–37, 1993.
74. Toumey JW, Hammon D, France P, Norwood LA. Posterior recurrent dislocation of the shoulder treated by capsulorrhaphy and iliac bone block. *Lahey Clin Bull* 5:197–201, 1948.
75. Warner JJP, Micheli LJ, Arslanian LE, Kennedy J, Kennedy R. Scapulothoracic motion in normal shoulders and shoulders with glenohumeral instability and impingement syndrome. *Clin Orthop* 285:191–9, 1992.
76. Warren RF, Kornblatt IB, Marchand R. Static factors affecting posterior shoulder stability. *Orthop Trans* 8:89, 1984;.
77. Wirth MA, Butters KP, Rockwood CA Jr. The posterior deltoid-splitting approach to the shoulder. *Clin Orthop* 296:92–8, 1993.
78. Wirth MA, Lyons FR, Rockwood CA Jr. Hypoplasia of the glenoid. A review of sixteen patients. *J Bone Joint Surg* 75A:1175–84, 1993.
79. Wirth MA, Seltzer DG, Rockwood CA Jr. Recurrent posterior glenohumeral dislocation associated with increased retroversion of the glenoid. A case report. *Clin Orthop* 308:98–101, 1994.