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Chapter 9

Multidirectional and Posterior Shoulder Instability

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Multidirectional Instability

Neer and Foster first described the diagnosis and management of MDI, based on their study of patients with inferior subluxation or dislocation and associated anteroposterior subluxation or dislocation.[1] They identified three types of MDI: anteroinferior dislocation and posterior subluxation, posteroinferior dislocation and anterior subluxation, and dislocation in all three directions. Neer and Foster distinguished MDI from the more commonly recognized unidirectional anteroposterior instability, emphasizing the often-subtle presentation of MDI and the pitfalls of treating it with a traditional unidirectional repair. They proposed using an inferior capsular shift, and this procedure has had good intermediate-term results.

Definition and Classification

Despite progress in shoulder instability research, the understanding of MDI is still incomplete. There is no standard definition in the literature, and patient symptoms are variable. Therefore, research outcomes must be carefully interpreted with regard to the study's definition of MDI.

Instability is defined either as pathologic joint translation that causes symptoms or as inability to keep the humeral head centered within the glenoid cavity during active motion. The term is reserved for symptomatic shoulders and specifically for those in which the sensation of the humeral head translating on the glenoid is present. Instability is frequently associated with pain or discomfort. Laxity is defined as translation in a particular direction or rotation. A person who has significant laxity may have no symptoms. MDI can be defined as shoulder laxity that is global (anterior, posterior, and inferior) and concurrently produces symptoms inferiorly and in at least one other direction. The symptoms are usually experienced in the end ranges of glenohumeral motion and may limit the patient's activities of daily living. Extremes of motion are avoided because of significant apprehension.

Etiology

MDI can have a congenital or acquired etiology. Congenital, or primary, MDI occurs in patients with an inherited ligamentous laxity; some of these patients have a collagen disorder such as Ehlers-Danlos syndrome or Marfan syndrome. These patients tend to develop MDI at a relatively young age and are less likely to be successfully treated with surgery. Frequently they have a positive family history. In contrast, acquired, or secondary, MDI results from repetitive microtrauma to the shoulder. It is commonly seen in swimmers, weight lifters, gymnasts, and athletes who participate in throwing or racquet sports. These patients may have some underlying hyperlaxity. A previously asymptomatic individual may become symptomatic after a single traumatic event, which can be relatively minor.

The etiology of MDI is believed to be multifactorial, and the current theories focus on anatomic, biochemical, and neuromuscular abnormalities. In a normal shoulder, glenohumeral stability is maintained by an intricate balance of static and dynamic mechanisms. The capsuloligamentous restraints in the shoulder function as checkreins to impart stability at the extremes of motion. In the midrange of glenohumeral motion, the precise centering of the humeral head on the glenoid is enhanced by the rotator cuff muscles through a mechanism known as concavity-compression. Labral detachment can compromise concavity-compression, because the labrum normally deepens the glenoid by 50% and contributes 20% of the stability ratio in the inferior and posteroinferior directions.[2] Recurrent instability can lead to muscle imbalance of the dynamic stabilizers, which can also compromise effective concavity-compression.

A deficiency of the rotator interval tissue and a redundant inferior capsular pouch are the two anatomic

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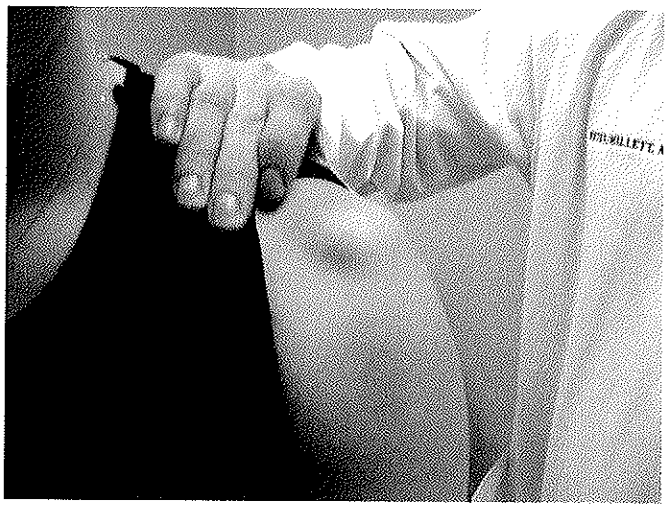

Figure 1 Squaring of the shoulder, indicating the presence of inferior instability.

lesions associated with MDI. Cadaver studies have demonstrated the importance of the rotator interval and inferior capsular pouch in resisting inferior-posterior displacement of the humeral head. In MDI patients, the rotator interval tissue was found to have a cleft or to be insubstantial.[3,4] Defects in the interval can disrupt the normal negative intra-articular pressure and thereby contribute to instability.[5] The superior glenohumeral ligament is the primary restraint to inferior subluxation when the arm is in adduction.[6] The inferior glenohumeral ligament is the primary restraint to inferior translation when the arm is progressively abducted; during humeral rotation, it also imparts anteroposterior stability. Thus, a capacious inferior pouch can contribute to instability in the inferior, anterior, and posterior directions. The inferior glenohumeral ligament, a hammock-like structure with anchors on both sides of the glenoid, contributes to the stability of the glenohumeral joint.

Basic science studies have examined the type and quantity of collagen in patients with MDI. Collagen fibril diameter and cross-linking are directly related to tensile strength. Capsular tissue from patients with MDI or unidirectional instability was found to have more stable and reducible collagen cross-links, greater mean collagen fibril diameter, higher cysteine concentration, and higher density of elastin, compared with tissue from unaffected individuals.[7] It is unknown whether these differences represent a predisposition to laxity or a response to repetitive stretching injuries. Skin samples from patients with MDI were found to have a significantly smaller mean collagen fibril diameter than samples from patients with unidirectional instability.[7] This finding suggests the presence of an underlying connective tissue abnormality.

Neuromuscular abnormalities have been investigated as a factor in MDI. The theory that MDI has an underlying neuromuscular cause is supported by several research findings. Many patients have equal or greater laxity in the contralateral, asymptomatic shoulder.[8] Symptoms occur in the midrange of glenohumeral motion, in which capsuloligamentous restraints remain lax.[9] Patients have altered glenohumeral and scapulothoracic rhythm.[9] Mechanoreceptors were identified in the shoulder joint capsule of patients with MDI, as well as proprioceptive deficits that can be reversed by surgical stabilization.[10,11] In patients with MDI and laxity, electromyography revealed abnormalities in the deltoid muscle, rather than in the rotator cuff muscles.[12] Another study found significant differences between the activation parameters of the supraspinatus, infraspinatus, posterior deltoid, and pectoralis major muscles of patients with MDI and those of control subjects.[13]

Clinical Evaluation

History

A patient with MDI may have a variety of symptoms, including pain, instability, weakness, paresthesias, fatigue, popping, clicking, grinding, dead arm syndrome, and difficulty in throwing, lifting, or sleeping. The patient should be asked about the positions that provoke symptoms, because this information can help in defining the primary direction of instability. Discomfort and traction paresthesias are usually associated with inferior instability. A patient with anterior instability will report difficulty in reaching overhead or sleeping with the arms overhead. MDI should be differentiated from traumatic anterior instability, which has a poor prognosis with nonsurgical treatment. Symptoms that occur with shoulder forward flexion can indicate posterior instability. In addition, patients should be asked whether the subluxation or dislocation has a voluntary component.

The incidence of MDI is greatest in the second and third decades of life, and most patients are younger than 35 years. There is apparently no gender difference. Preadolescent patients are more likely to have voluntary subluxation; they usually do not have pain.[8] Surgical intervention is probably not advisable for a preadolescent patient, because MDI in this age group has a favorable natural history.

Physical Examination

A complete shoulder examination should be performed, including an evaluation of the cervical spine and the scapulothoracic articulation. The shoulder should be visually inspected for asymmetry, scapular winging, atrophy, and the presence of previous incisions. Squaring of the shoulder is a common finding in inferior instability, because the normally round contour of the deltoid is lost as the humeral head is inferiorly subluxated (Figure 1). The acromioclavicular, sternoclavicular, and scapulothoracic joints are often overlooked as possible

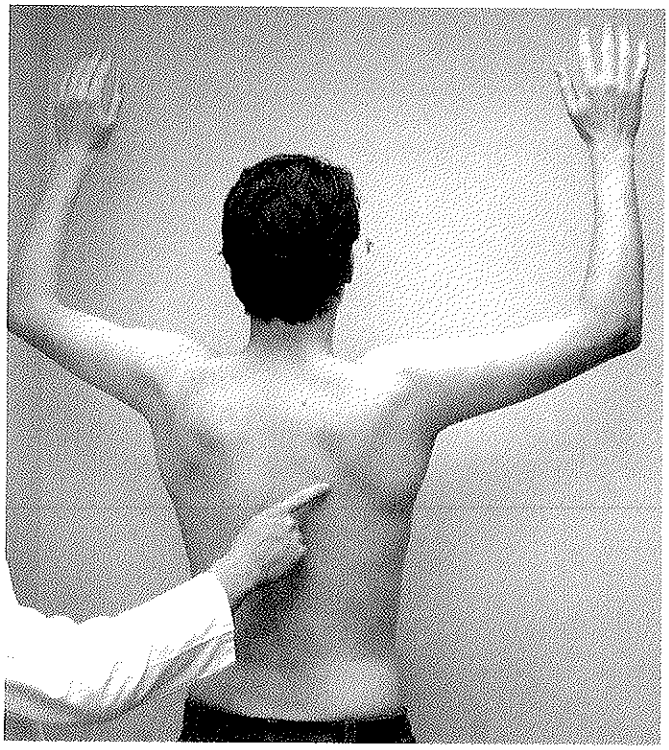

Figure 2 Scapulohumeral examination, showing the normal position of the scapula.

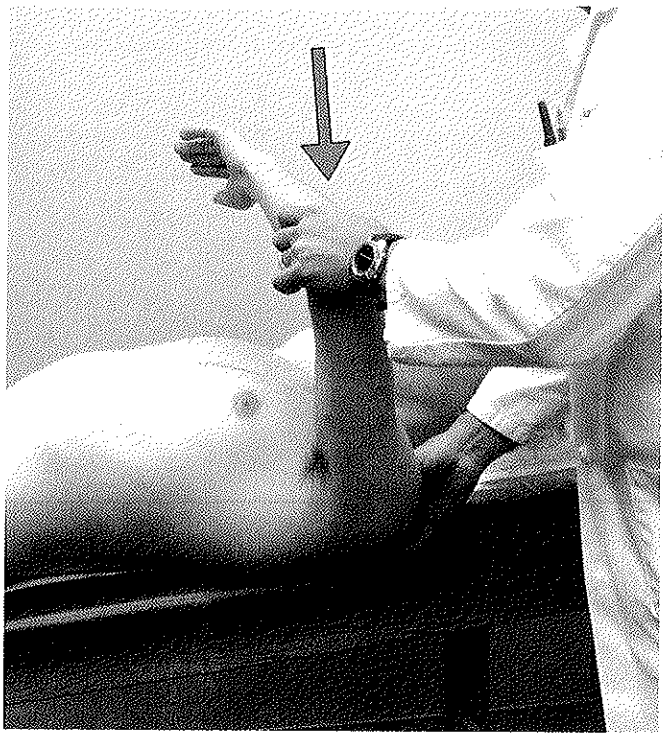

Figure 3 The posterior stress test.

sources of the patient's symptoms. These joints can generate mechanical symptoms that may be misinterpreted as originating from the glenohumeral joint.

Generalized ligamentous laxity should be assessed by evaluating the contralateral shoulder, the elbows, and the knees and by testing the patient's ability to oppose the thumb to the forearm. Signs of generalized ligamentous laxity have been reported in 45% to 75% of patients undergoing surgery for MDI.[1,9,14] Sulcus testing should be performed and quantified as the distance from the greater tuberosity to the acromion. A value greater than 2 cm is considered pathognomonic for MDI, although pain and symptoms of inferior instability must also be present for the diagnosis to be conclusive. If the sulcus distance does not decrease as the arm is externally rotated, the shoulder should be considered to have a pathologic defect in the rotator interval.

Scapulohumeral rhythm and scapulothoracic mechanics should be assessed to exclude the possibility of scapular winging, which can be confused with posterior instability (Figure 2). Compensatory scapular winging can occur; it dynamically increases bony stability by anteverting the glenoid. A thorough neurologic examination and appropriate neurologic testing should be performed.

Most patients with MDI have increased passive range of motion in the shoulder joint, which is not considered pathologic unless accompanied by symptoms. The redundancy of the inferior capsule can be assessed by holding the arm in 90° of abduction, with the forearm in neutral rotation and with a downward force applied to the lateral brachium. In the hyperabduction test, passive range of abduction greater than 105° is associated with laxity in the inferior glenohumeral ligament.[15] Anteroposterior instability can be assessed using the load-and-shift test and modified load-and-shift test. The apprehension, relocation, posterior stress, and jerk tests can be used to further isolate anteroposterior instability.[16] Impingement tests are commonly positive in patients with MDI. A distinction should be made between maneuvers that cause pain and those that cause apprehension.

The posterior stress, jerk, load-and-shift, and modified load-and-shift tests are performed specifically to identify posterior instability. The posterior stress test is performed with the patient supine. The arm is flexed to 90° and internally rotated. An axial load is applied to the humerus with one hand, with the other hand applied to the back of the shoulder (Figure 3). A positive test results in palpable or observable subluxation of the humeral head over the glenoid rim.

The jerk test is performed with the patient sitting upright; the arm is flexed 90° and internally rotated, and the elbow is flexed 90°. An axial load is applied to the arm, with the other arm supporting the posterior shoulder, and the arm is then extended. If the test is positive, the glenohumeral joint will be reduced from a posteriorly subluxated or dislocated position with a jerk.

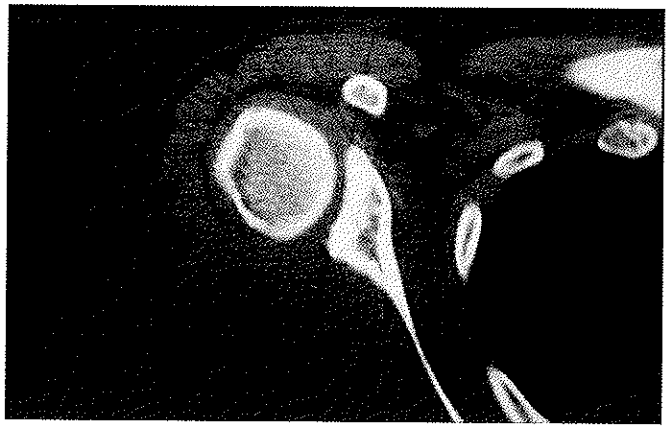

Figure 4 MRI showing glenoid dysplasia.

The load-and-shift test is performed with the patient sitting upright, with the arm at the side. The humeral head and proximal humerus are grasped and compressed into the glenoid fossa; anteroposterior stress is applied, and the degree of translation is graded. A 50% displacement of the humeral head is considered the upper limit of the normal range.

The modified load-and-shift test is performed with the patient supine and the affected shoulder positioned at the edge of the examination table. The shoulder is placed in neutral rotation in the scapular plane. Axial force is applied at the elbow to concentrically reduce the humeral head in the glenoid fossa. Anteroposterior force is applied to the proximal humerus in varying degrees of rotation and elevation, and the degree of translation is graded. The load-and-shift and modified load-and-shift tests are typically graded as follows: grade 0, minimal translation; grade 1, translation of the humeral head to the glenoid rim; grade 2, translation of the humeral head over the glenoid rim, with spontaneous reduction; grade 3, translation of the humeral head over the glenoid rim, without spontaneous reduction.

The load-and-shift, sulcus, and provocative tests were found to be reliable in detecting instability,[17] and positive sulcus sign and load-and-shift tests were found to have a high positive predictive value for MDI.[18]

Imaging Studies

Plain radiography is required for any patient suspected of having shoulder instability. The standard series includes the AP, true AP, and axillary or West Point views. Osseous pathology is occasionally present, and therefore it is important to look for a humeral head defect (Hill-Sachs or reverse Hill-Sachs lesion), traumatic glenoid change (bony Bankart lesion, erosion, or rounding), or inherent bony abnormality such as glenoid dysplasia or retroversion (Figure 4). CT should be used if examination of plain radiographs suggests the presence of a bony abnormality. CT arthrography can accurately assess capsulolabral detachment. In many centers, MRI is used rather than CT, although it does not provide as much bony detail. The use of CT or MRI may not be warranted in the absence of a suspected bony abnormality or traumatic history.

Nonsurgical Treatment

MDI should first be treated using nonsurgical methods. Physical therapy programs for shoulder rehabilitation focus on strengthening and retraining the rotator cuff muscles, as well as strengthening the periscapular musculature. The goal is to restore effective concavity-compression and normal muscle-firing patterns and to improve proprioceptive function. In most patients, positive results appear within the first 3 months. An 80% success rate was found after nonsurgical treatment of patients with MDI.[14] In 57 patients at an average 8-year follow-up after rehabilitation for MDI, 19 (33%) had a poor result.[19]

Surgical Treatment

Surgical treatment should be considered for a patient whose MDI has not responded to nonsurgical treatment. The open inferior capsular shift procedure is intended to reduce capsular volume and thicken and overlap capsule on the side of greatest instability. This procedure can be performed through an anterior, posterior, or combined surgical approach. In using a posterior approach, it is important to recognize that the posterior capsule is thin and pliable in comparison with the robust anterior capsule. The shift can be humeral or glenoid based, according to the surgeon's preference. At an average 8.3-year follow-up of 34 shoulders, the inferior capsular shift procedure was found to have a satisfactory outcome in 88% of shoulders, with recurrent instability in 26%; radiographs revealed a posterolateral defect in the humeral head in 9 of 14 shoulders treated with a posterior or combined surgical approach.[20] A cadaver study found decreased joint volume after inferior capsular shift.[21]

As shoulder arthroscopy has evolved during the past decade, the frequency of arthroscopic treatment of MDI has also increased. High rates of failure were initially reported, but recent studies have found results equal to or better than those of open procedures. In current arthroscopic techniques, multiple portals and suture anchors are used to reduce capsular volume and recreate the capsulolabral construct.[22] Twenty-five patients having no history of earlier shoulder surgery were treated for MDI with an arthroscopic capsular shift using a transglenoid technique; 88% had a satisfactory result (using the Neer rating) at an average 5-year follow-up.[23] Another study found a successful result in 44 of 47 patients (94%) at 2- to 5-year follow-up after an arthroscopic capsular shift using suture anchors; 85% had returned to their desired level of sports activity.[24]

Thermal capsulorrhaphy was briefly popular for treating MDI and capsular laxity. The procedure is easy to perform and was based on valid basic science findings. However, there was no solid clinical evidence to support its popularity. The most popular thermal capsulorrhaphy techniques used laser or radiofrequency heat technology. Collagen shrinkage was found to occur at temperatures between 65°C and 75°C. The amount of shrinkage depended on the length of exposure and the cross-linking of the exposed tissue.[25] Cell death begins when the temperature of the tissue reaches 45°C and concludes at 55°C to 60°C. The biologic response to this injury varies but is characterized by fibroplasia and angiogenesis. In some in vitro studies, anteroposterior laxity increased after thermal treatment;[26] other studies found decreased capsular volume and glenohumeral translation.[27] The clinical results of thermal capsulorrhaphy for MDI were comparable to the results of cadaver studies. Success rates from 40% to 100% were reported. At 2-year follow-up, thermal capsulorrhaphy had failed in 9 of 19 shoulders with MDI (47%); during revision surgery, 3 patients (33%) were found to have a capsular deficiency.[28] Another study found that 37% of patients with recurrent anterior dislocation or MDI had an unsatisfactory result at 2- to 5-year follow-up after thermal capsulorrhaphy.[29]

Although the role of thermal capsulorrhaphy in the treatment of MDI has yet to be determined, it is important to note that this technique is not recommended because of its high failure rate and the risk of complications such as thermal necrosis and axillary neuritis.

Posterior Instability

Posterior instability is an uncommon condition, affecting only 2% to 5% of all patients with shoulder instability. It can occur in isolation or as a component of MDI; unidirectional posterior instability is more common than posterior instability associated with MDI. Trauma is believed to be the underlying cause in approximately half of patients with posterior instability. The majority of patients are athletes who participate in activities that cause repetitive stress to the posterior capsule.

Definition and Classification

Posterior shoulder instability can be classified by direction, degree, cause, or volition. The distinction between posterior laxity and posterior instability is important. Patients with posterior instability frequently have pain. The degree of instability can range from mild subluxation to frank dislocation, although recurrent posterior subluxation is the most common form.

Posterior instability is most commonly caused by acquired trauma, which can be a single traumatic event that occurs when the shoulder is in an at-risk position (flexion, adduction, or internal rotation) or as the culmination of multiple smaller traumatic episodes.[16] The presence of posterior instability without trauma should alert the physician to the possibility of an underlying collagen disease or bony abnormality. Surgical intervention for such patients should be approached with caution.

Determining the volitional aspect of posterior instability is important. Involuntary posterior instability typically results from a traumatic event and most commonly is manifested as mild subluxation. The patient cannot control the symptoms. Voluntary posterior instability occurs when the patient is able to willfully dislocate or subluxate the shoulder. These patients have voluntary muscular and positional patterns, as well as an underlying muscular imbalance that allows posterior subluxation or dislocation. A patient with voluntary posterior instability is usually considered a poor surgical candidate, although a patient with voluntary positional posterior instability and no underlying psychiatric or secondary gain issues may respond well to surgical intervention. Such a patient has instability when the arm is flexed and adducted. Although able to voluntarily produce the instability, the patient prefers to avoid doing so.

Clinical Evaluation

History

A fall or blow to the arm in an at-risk position can result in a posterior labral detachment (reverse Bankart lesion). Repetitive stress on the posterior capsule during sports or another activity can lead to acquired posterior subluxation. Patients commonly report pain and weakness, and other instability symptoms may also be present. Overhead athletes often describe insidious pain occurring late in their sports activity, when muscle fatigue and dynamic stability are compromised. Direction, frequency, severity, mechanical symptoms, and volition should be investigated as part of the patient history.

Physical Examination

Physical examination findings are often more subtle in a patient with posterior instability than in a patient with anterior instability or MDI. The active and passive ranges of motion are usually normal and symmetric. Posterior joint line tenderness may be present with palpation, and crepitus may occur as the arm is internally rotated. Strength testing usually reveals symmetry, although rare patients have a posterior rotator cuff deficiency or nerve injury with external rotation weakness. Atrophy of the posterior rotator cuff musculature may be apparent. Skin dimpling along the posteromedial border of the deltoid also has been associated with recurrent posterior shoulder dislocation.[30]

Generalized ligamentous laxity should be assessed by evaluating the contralateral shoulder, the elbows, and

the knees, and by testing the patient's ability to oppose the thumb to the forearm. The evaluation of a patient with suspected posterior instability should include sulcus testing, assessment of scapulohumeral rhythm and compensatory scapular winging, and a thorough neurologic examination. Specific posterior instability tests include the posterior stress test, jerk test, load-and-shift test, and modified load-and-shift test.

Imaging Studies

Standard plain radiographs of the shoulder should be obtained. CT or MRI can be used in assessing glenoid morphology and version. CT arthrography provides the most useful information for assessing bony anatomy and articular orientation.

Nonsurgical Treatment

Patients with posterior instability had a 63% to 91% success rate after nonsurgical treatment, with no limitations in activities of daily living and only moderate limitations in sports activities.[31,32] The goal of physical therapy is to strengthen the dynamic muscular stabilizers to compensate for damaged or deficient static stabilizers. The exercises should focus on strengthening the posterior deltoid, the external rotators, and the periscapular muscles, and they should be augmented with activity modification and biofeedback.

Surgical Treatment

The open posteroinferior capsular shift procedure is often used to address soft-tissue abnormalities resulting in posterior instability. The joint can be inspected, any posterior labral injury can be repaired, and the capsular shift and repair can be performed through a posterior approach.[31,33] An 85% success rate was reported at an average 7-year follow-up after open posterior capsulorrhaphy.[33] An 80% success rate was reported at an average 5-year follow-up, and the success rate increased to 96% when revision procedures were excluded.[34] In 44 shoulders (41 patients) treated with primary shoulder stabilization using an open posterior capsular shift, 8 shoulders (19%) had a recurrence of posterior instability at 1.8-year to 22.5-year follow-up; 84% of patients were satisfied with the current status of their shoulder.[35] However, satisfaction and outcome scores were significantly poorer in shoulders found to have a chondral defect at the time of stabilization and in patients older than 37 years at the time of surgery.

A patient with a posterior Bankart lesion is an ideal candidate for either an open posteroinferior capsular shift or arthroscopic treatment. The relative contraindications for an arthroscopic procedure include an unsuccessful earlier arthroscopic stabilization, humeral avulsion of the glenohumeral ligament, or gross bidirectional instability or MDI from generalized laxity

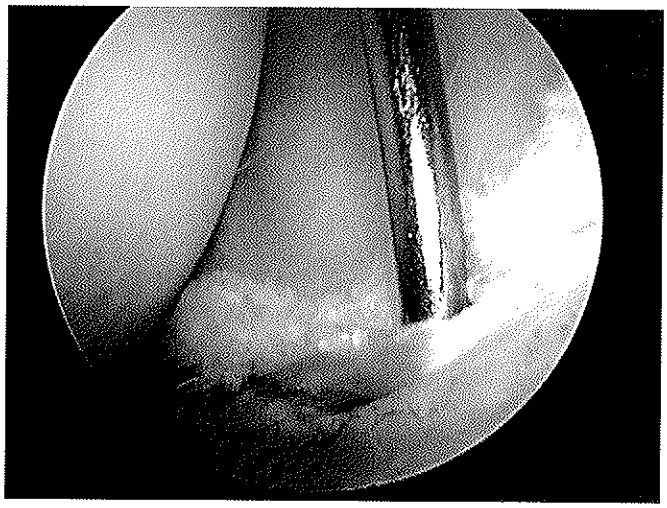

Figure 5 Arthroscopic view of a posterior Bankart lesion.

caused by a condition such as Ehlers-Danlos syndrome or Marfan syndrome. However, patients with multidirectional laxity that is symptomatic only in a posterior direction respond well to arthroscopic stabilization. Glenoid erosion and excessive glenoid retroversion are absolute contraindications for an arthroscopic procedure. Arthroscopic treatment can successfully address capsulolabral injury and capsular redundancy, and rotator interval closure can provide additional inferior stability. With the patient in the beach chair position, an accessory portal placed through the midportion of the rotator cuff allows easy and complete viewing of the posterior glenohumeral joint and does not require the use of traction.[36] This portal provides a superior-to-inferior view of the posterior glenoid rim and capsule and allows the use of standard anterior and posterior portals for posterior Bankart repair (Figure 5)

The posteroinferior labral lesion associated with posterior instability has recently received increased attention. Four types have been identified: type I, an incomplete detachment; type II, a marginal crack (Kim Lesion); type III, a chondrolabral erosion; and type IV, a flap tear.[37-39]

Arthroscopic posterior capsular plication for unidirectional posterior instability was successful in 16 of 17 patients; 11 returned to their preinjury level of function.[40] After arthroscopic posteroinferior capsulolabral augmentation, 35 of 41 patients noted improvement.[41] The success rate was 92% in 27 shoulders repaired arthroscopically; 55% of the patients were American football players.[42] After arthroscopic stabilization, 26 of 27 patients who had recurrent posterior instability were able to return to their sport with little or no limitation. Of 33 patients who underwent successful arthroscopic treatment of posterior shoulder instability, those with voluntary dislocation and earlier surgery had poorer

outcomes.[43] At a minimum 2-year follow-up of 100 shoulders, the mean American Shoulder and Elbow Surgeons (ASES) Shoulder Index score improved from 50.36 to 85.66 ($P < 0.001$), and stability, pain, and function significantly improved, based on standardized subjective scales ($P < 0.001$).[44] Contact athletes did not have significantly different outcomes from the entire group on any measure. Overall, 89% of patients were able to return to their sport, and 67% were able to return to their earlier level.

Thermal shrinkage was formerly used to decrease a patulous posterior capsule. The clinical result was unpredictable, and the reported failure rates were unacceptably high, ranging from 4% to 60%.[29,45] In addition, capsular necrosis and rupture were reported.[46] For these reasons, the technique is not recommended.

Summary

Posterior instability and MDI are diagnosed based on the patient's history, a thorough physical examination, and appropriate imaging. Posterior instability is particularly challenging to diagnose and treat, because the condition is uncommon and its symptoms are often subtle. Patients with MDI, voluntary instability, or a bony defect require careful assessment to discover the cause of the instability. Most patients respond to a well-designed rehabilitation program; for those who require surgical treatment, predictable results can be achieved using either an open or arthroscopic technique. The use of thermal capsulorrhaphy should be avoided.

Annotated References

1. Neer CS II, Foster CR: Inferior capsular shift for involuntary inferior and multidirectional instability of the shoulder: A preliminary report. *J Bone Joint Surg Am* 1980;62:897-908.

2. Howell SM, Galinat BJ: The glenoid-labral socket: A constrained articular surface. *Clin Orthop Relat Res* 1989;243:122-125.

3. Harryman DT II, Sidles JA, Harris SL, Matsen FA III: The role of the rotator interval capsule in passive motion and stability of the shoulder. *J Bone Joint Surg Am* 1992;74:53-66.

4. Warner JJ, Deng XH, Warren RF, Torzilli PA: Static capsuloligamentous restraints to superior-inferior translation of the glenohumeral joint. *Am J Sports Med* 1992;20:675-685.

5. Kumar VP, Balasubramaniam P: The role of atmospheric pressure in stabilising the shoulder: An experimental study. *J Bone Joint Surg Br* 1985;67:719-721.

6. Turkel SJ, Panio MW, Marshall JL, Girgis FG: Stabilizing mechanisms preventing anterior dislocation of the glenohumeral joint. *J Bone Joint Surg Am* 1981;63:1208-1217.

7. Rodeo SA, Suzuki K, Yamauchi M, Bhargava M, Warren RF: Analysis of collagen and elastic fibers in shoulder capsule in patients with shoulder instability. *Am J Sports Med* 1998;26:634-643.

8. Self, EB Jr: Clinical guidelines for shoulder pain, in Norris TR (ed): *Orthopaedic Knowledge Update: Shoulder and Elbow 2*. Rosemont, IL, American Academy of Orthopaedic Surgeons, 2002, pp 443-467.

   This is a review of clinical guidelines for evaluation and management of shoulder pain.

9. Schenk TJ, Brems JJ: Multidirectional instability of the shoulder: Pathophysiology, diagnosis, and management. *J Am Acad Orthop Surg* 1998;6:65-72.

10. Lephart SM, Myers JB, Bradley JP, Fu FH: Shoulder proprioception and function following thermal capsulorrhaphy. *Arthroscopy* 2002;18:770-778.

    Twenty patients with anterior, anteroinferior, or multidirectional glenohumeral instability were treated with thermal capsulorrhaphy. At a 6- to 24-month follow-up, no appreciable deleterious effect on proprioception or function was found. Level of evidence: IV.

11. Lephart SM, Borsa PA, Fu FH: Proprioception of the shoulder in health, unstable, and surgically repaired shoulders. *J Shoulder Elbow Surg* 1994;3:371-380.

12. Morris AD, Kemp GJ, Frostick SP: Shoulder electromyography in multidirectional instability. *J Shoulder Elbow Surg* 2004;13:24-29.

    Patients with MDI had activity patterns of the anterior, middle, and posterior deltoid different from individuals with normal shoulders. Activity patterns of the rotator cuff were similar in both groups. Inefficiency of the dynamic stabilizers of the glenohumeral joint may be involved in MDI. Level of evidence: IV.

13. Barden JM, Balyk R, Raso VJ, Moreau M, Bagnall K: Atypical shoulder muscle activation in multidirectional instability. *Clin Neurophysiol* 2005;116:1846-1857.

    MDI is associated with atypical patterns of muscle activity that occur even when highly constrained movements are used to elicit the activity. Glenohumeral hyperlaxity and dysfunctional neuromuscular control of the rotator cuff are contributing factors to the pathoetiology of MDI. Level of evidence: IV.

14. Burkhead WZ Jr, Rockwood CA Jr: Treatment of instability of the shoulder with an exercise program. *J Bone Joint Surg Am* 1992;74:890-896.

15. Gagey OJ, Gagey N: The hyperabduction test. *J Bone Joint Surg Br* 2001;83:69-74.

    In patients with instability, 85% had a range of passive abduction of more than 105°, compared with 90° in the contralateral shoulder. A range of passive abduction of more than 105° is associated with lengthening and laxity of the inferior glenohumeral ligament. Level of evidence: IV.

16. Millett PJ, Clavert P, Hatch GF III, Warner JJ: Recurrent posterior shoulder instability. *J Am Acad Orthop Surg* 2006;14:464-476.

    The current understanding, diagnosis, and treatment of posterior and multidirectional instability of the shoulder are reviewed.

17. Tzannes A, Paxinos A, Callanan M, Murrell GA: An assessment of the interexaminer reliability of tests for shoulder instability. *J Shoulder Elbow Surg* 2004;13:18-23.

    The load-and-shift, sulcus, and provocative tests (apprehension, augmentation, relocation, and release) are reliable clinical tests for instability in symptomatic patients, if care is taken with respect to arm positioning and if the apprehension test is positive. Level of evidence: III.

18. Tzannes A, Murrell GA: Clinical examination of the unstable shoulder. *Sports Med* 2002;32:447-457.

    A sulcus 2 cm or larger appearing beneath the acromion while the arm is pulled inferiorly is predictive of MDI (likelihood ratio, 9). A positive load-and-shift test is extremely predictive for instability (likelihood ratio, >80). Level of evidence: V.

19. Misamore GW, Sallay PI, Didelot W: A longitudinal study of patients with multidirectional instability of the shoulder with seven- to ten-year follow-up. *J Shoulder Elbow Surg* 2005;14:466-470.

In young, athletic patients, the response to nonsurgical treatment of MDI was relatively poor. Only 17 of 57 patients had a satisfactory outcome after 7 to 10 years of nonsurgical management. Level of evidence: II.

20. Hamada K, Fukuda H, Nakajima T, Yamada N: The inferior capsular shift operation for instability of the shoulder: Long-term results in 34 shoulders. *J Bone Joint Surg Br* 1999;81:218-225.

21. Yamamoto N, Itoi E, Tuoheti Y, et al: The effect of the inferior capsular shift on shoulder intra-articular pressure: A cadaveric study. *Am J Sports Med* 2006;34:939-944.

In seven fresh-frozen cadaver shoulders, the inferior capsular shift procedure decreased joint volume and increased responsiveness of intra-articular pressure to downward loading.

22. Cohen SB, Wiley W, Goradia VK, Pearson S, Miller MD: Anterior capsulorrhaphy: An in vitro comparison of volume reduction. Arthroscopic plication versus open capsular shift. *Arthroscopy* 2005; 21:659-664.

In this in vitro anatomic comparison of procedures, open lateral capsular shift resulted in significantly greater volume reduction than arthroscopic plication. The amount of volume reduction required to eliminate instability caused by capsular laxity remains unknown. Level of evidence: IV.

23. Treacy SH, Savoie FH III, Field LD: Arthroscopic treatment of multidirectional instability. *J Shoulder Elbow Surg* 1999;8:345-350.

24. Gartsman GM, Roddey TS, Hammerman SM: Arthroscopic treatment of multidirectional glenohumeral instability: 2- to 5-year follow-up. *Arthroscopy* 2001;17:236-243.

Of 47 patients treated arthroscopically for shoulder instability, 94% (44 patients) had a good or excellent outcome at final follow-up; 85% (22 of 26 patients) had returned to their desired level of sports activity. Level of evidence: IV.

25. Hayashi K, Markel MD: Thermal capsulorrhaphy treatment of shoulder instability: Basic science. *Clin Orthop Relat Res* 2001;390: 59-72.

26. Wolf RS, Zheng N, Iero J, Weichel D: The effects of thermal capsulorrhaphy and rotator interval closure on multidirectional laxity in the glenohumeral joint: A cadaveric biomechanical study. *Arthroscopy* 2004;20:1044-1049.

The effects of thermal capsulorrhaphy and rotator interval closure on glenohumeral laxity were examined in 10 cadaver shoulders. Statistical analysis revealed that rotator interval closure significantly decreased laxity. Thermal capsulorrhaphy did not increase anterior and posterior translation but did decrease inferior translation.

27. Victoroff BN, Deutsch A, Protomastro P, Barber JE, Davy DT: The effect of radiofrequency thermal capsulorrhaphy on glenohumeral translation, rotation, and volume. *J Shoulder Elbow Surg* 2004;13:138-145.

This cadaver study evaluated changes in resistance to translational forces, rotation, and joint volume in radiofrequency thermal capsulorrhaphy. Significant reduction in glenohumeral translation and volume with only a small loss of rotation was found.

28. Miniaci A, McBirnie J: Thermal capsular shrinkage for treatment of multidirectional instability of the shoulder. *J Bone Joint Surg Am* 2003;85:2283-2287.

Nineteen patients with MDI were treated with thermal capsular shrinkage. The failure rate was substantial. Recurrence of instability occurred in 47%; stiffness, in 26%; and neurological symptoms, in 21%. Level of evidence: IV.

29. D'Alessandro DF, Bradley JP, Fleischli JE, Connor PM: Prospective evaluation of thermal capsulorrhaphy for shoulder instability: Indications and results, two- to five-year follow-up. *Am J Sports Med* 2004;32:21-33.

This nonrandomized prospective study evaluated the indications for and results of thermal capsulorrhaphy in 84 shoulders. At an average 38-month follow-up, the overall results were excellent in 33 (39%), satisfactory in 20 (24%), and unsatisfactory in 31 (37%).

30. Williams RJ III, Strickland S, Cohen M, Altchek DW, Warren RF: Arthroscopic repair for traumatic posterior shoulder instability. *Am J Sports Med* 2003;31:203-209.

This is a retrospective review of the arthroscopic repair of 27 patients with a posterior Bankart lesion. All patients had a detached capsulolabral complex. At an average 5.1-year follow-up, 25 patients reported no pain or instability. Level of evidence: IV.

31. Fronek J, Warren RF, Bowen M: Posterior subluxation of the glenohumeral joint. *J Bone Joint Surg Am* 1989;71:205-216.

32. 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* 1992; 20:396-400.

33. Hawkins RJ, Koppert G, Johnston G: Recurrent posterior instability (subluxation) of the shoulder. *J Bone Joint Surg Am* 1984;66: 169-174.

34. Pollock RG, Bigliani LU: Recurrent posterior shoulder instability: Diagnosis and treatment. *Clin Orthop Relat Res* 1993;291:85-96.

35. Wolf BR, Strickland S, Williams RJ, Allen AA, Altchek DW, Warren RF: Open posterior stabilization for recurrent posterior glenohumeral instability. *J Shoulder Elbow Surg* 2005;14:157-164.

This retrospective study evaluated clinical and radiographic outcomes after open posterior stabilization. Open posterior shift was found to be reliable in shoulders with a grade III posterior instability, particularly with a sulcus of grade II or higher. Level of evidence: IV.

36. Costouros JG, Clavert P, Warner, JJ: Trans-cuff portal for arthroscopic posterior capsulorrhaphy. *Arthroscopy* 2006;22:A9-A16.

Five patients underwent arthroscopic repair of posterior shoulder instability using the trans–rotator cuff portal. The mean ASES score improved from 53 (±15) to 87 (±8). All patients had complete resolution of pain and instability at an average 24-month follow-up.

37. Kim SH, Park JS, Jeong WK, Shin SK: The Kim test: A novel test for posteroinferior labral lesion of the shoulder: A comparison to the jerk test. *Am J Sports Med* 2005;33:1188-1192.

The Kim test, a novel diagnostic test for detecting a posteroinferior labral lesion of the shoulder, was compared with the jerk test in 172 painful shoulders. The Kim test was more sensitive in detecting a predominantly inferior labral lesion. Level of evidence: I.

38. Kim SH, Ha KI, Yoo JC, Noh KC: Kim's lesion: An incomplete and concealed avulsion of the posteroinferior labrum in posterior or multidirectional posteroinferior instability of the shoulder. *Arthroscopy* 2004;20:712-720.

This study clinically described the Kim's lesion, which is an incomplete avulsion of the posteroinferior labrum concealed by ap-

parently intact superficial portion. Failing to address this lesion surgically may result in persistent posterior instability. Level of evidence: IV.

39. Kim SH, Kim HK, Sun JI, Park JS, Oh I: Arthroscopic capsulolabroplasty for posteroinferior multidirectional instability of the shoulder. *Am J Sports Med* 2004;32:594-607.

Thirty-one patients with symptomatic posteroinferior MDI had labral lesions, including retroversion of the posteroinferior labrum, which were previously unrecognized. Restoration of the labral buttress and capsular tension by arthroscopic capsulolabroplasty successfully stabilized the shoulders.

40. Wolf EM, Eakin CL: Arthroscopic capsular plication for posterior shoulder instability. *Arthroscopy* 1998;14:153-163.

41. Antoniou J, Duckworth DT, Harryman DT II: Capsulolabral augmentation for the management of posteroinferior instability of the shoulder. *J Bone Joint Surg Am* 2000;82:1220-1230.

42. Kim SH, Ha KI, Park JH, et al: Arthroscopic posterior labral repair and capsular shift for traumatic unidirectional recurrent posterior subluxation of the shoulder. *J Bone Joint Surg Am* 2003;85:1479-1487.

In all 27 patients who underwent arthroscopic posterior labral repair and capsular shift, shoulder function improved as measured using the University of California Los Angeles, ASES, and Rowe scores at a mean 39-month follow-up. All patients returned to their sports activity with little or no limitation. Level of evidence: IV.

43. Provencher MT, Bell SJ, Menzel KA, Mologne TS: Arthroscopic treatment of posterior shoulder instability: Results in 33 patients. *Am J Sports Med* 2005;33:1463-1471.

At a mean 39.1-month follow-up of 33 patients who underwent posterior arthroscopic shoulder stabilization, the mean ASES score was 94.6. Patients with voluntary instability or prior surgery had lower outcome scores. Level of evidence: IV.

44. Bradley JP, Baker CL III, Kline AJ, Armfield DR, Chhabra A: Arthroscopic capsulolabral reconstruction for posterior instability of the shoulder: A prospective study of 100 shoulders. *Am J Sports Med* 2006;34:1061-1071.

At a 27-month follow-up of 91 athletes with unidirectional posterior shoulder instability after arthroscopic treatment, the mean ASES score improved from 50.36 to 85.66. Stability, pain, and function significantly improved, and 89% were able to return to their sports activity. Level of evidence: II.

45. Lyons TR, Griffith PL, Savoie FH III, Field LD: Laser-assisted capsulorrhaphy for multidirectional instability of the shoulder. *Arthroscopy* 2001;17:25-30.

Laser-assisted capsulorrhaphy was performed on 27 shoulders (26 patients). At a minimum 2-year follow-up, 96% of shoulders remained stable and asymptomatic, and 86% of athletes had returned to their previous level of activity. In 12%, the rating was unsatisfactory.

46. 	Wong KL, Williams GR: Complications of thermal capsulorrhaphy of the shoulder. *J Bone Joint Surg Am* 2001;83-A(suppl 2, pt 2):151-155.

A survey of 378 surgeons on their experience with thermal treatment for shoulder instability revealed recurrent instability rates after laser, monopolar, and bipolar radiofrequency capsulorrhaphy of 8.4%, 8.3%, and 7.1%, respectively. Postsurgical axillary neuropathy affected 1.4% of patients.