Multidirectional Instability of the Shoulder: Pathophysiology, Diagnosis, and Management

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Abstract

Multidirectional instability of the shoulder is a complex entity. Relatively few series of patients with this condition have been reported. Affected patients have global (anterior, inferior, and posterior) excessive laxity of the glenohumeral joint capsule and a rotator interval capsule defect. The onset of symptoms is frequently related to atraumatic events. The chief complaint is more often related to pain than to instability per se. Symptoms are mostly experienced within the midrange of glenohumeral motion. Because the contralateral shoulder is often equally lax and asymptomatic, it appears that factors in addition to excessive capsular laxity play a pathophysiologic role. These factors may include subtle losses of strength and/or neuromotor coordination of the rotator cuff and scapular stabilizing muscles, defective proprioceptive responses, and the absence of a limited joint volume. Most patients can be successfully treated nonoperatively with a specific exercise program. If a 6-month trial of nonoperative management fails, the patient is a candidate for surgical reconstruction. The most time-honored procedure is an open inferior capsular shift, which corrects excessive global laxity of the capsule and the rotator interval defect.

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The first series of patients with multidirectional instability (MDI) of the shoulder was reported by Neer and Foster in 1980.[1] Patients suffered recurrent instability and pain. On clinical examination, the shoulder could be dislocated inferiorly and subluxated or dislocated anteriorly and posteriorly. They reported specifically on patients with MDI who did not respond to a program of strengthening exercises and then were treated surgically with an inferior capsular shift. A large, redundant inferior capsule was identified intraoperatively in all cases. The surgical procedure, designed by Neer, simultaneously eliminates excessive anterior, inferior, and posterior capsular laxity. The surgical technique also includes imbrication of the rotator interval capsule.

When discussing clinical aspects of MDI, it is imperative to distinguish between the terms "laxity" and "instability." "Laxity" objectively describes the extent to which the humeral head can be translated on the glenoid. "Instability" is an abnormal increase in glenohumeral translation that causes symptoms (subluxation or dislocation).[2] An asymptomatic shoulder that can be subluxated or dislocated in three directions on manual testing is described as having certain grades of laxity in three directions, but not MDI.

In our experience, patients with MDI possess two key clinical features. First, most symptoms are experienced in the midrange positions of glenohumeral motion, such as during activities of daily living. These symptoms are usually incapacitating enough that patients tend to avoid the extremes of glenohumeral motion. Second, the physical examination demonstrates the ability to dislocate or subluxate the glenohumeral joint in three directions (anteriorly, inferiorly, and posteriorly) with concurrent reproduction of symptoms in one or more of these directions.[1] Both features are thought to be necessary for a diagnosis of MDI and are useful in distinguishing MDI from other types of instability.

Classification

Classification of glenohumeral instability takes into consideration the frequency, direction, degree,

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and etiology of the instability and the possibility of voluntary causation of instability. Thomas and Matsen[3] commented that most patients with recurrent instability can be classified into traumatic and atraumatic groups. The characteristics of each group can be remembered with use of the mnemonic devices "TUBS" and "AMBRII," which have been derived as follows: Instability related to a Traumatic event presents as a Unidirectional instability problem, usually involves a Bankart lesion, and frequently requires Surgery to achieve stability. Instability that arises Atraumatically occurs in patients prone to Multidirectional instability who have Bilateral excessive laxity; this instability usually responds to a Rehabilitation program that emphasizes strengthening of the rotator cuff, but when operative intervention is undertaken, it must tighten the Inferior capsule and the rotator Interval capsule.

Neer and Foster[1] reported that the initial dislocation in their 36 patients with MDI occurred with varying degrees of injury: minor injury in 7 patients, moderate injury in 21 patients, and severe injury in 8 patients. Therefore, Neer[4] cautioned against a purely atraumatic concept of MDI because such thinking could lead to misdiagnosis.

Etiology

The etiologic factors of MDI include global shoulder laxity and precipitating events ranging from the atraumatic to the traumatic.[4]

Shoulder laxity can be congenital, acquired, or both.[4] In patients with congenitally lax shoulders, generalized ligamentous laxity is manifested in both shoulders and in other joints. Some patients are thought to acquire isolated shoulder laxity through the cumulative effect of repetitive use involving extremes of glenohumeral motion. Acquired laxity has been noted to occur in competitive athletes (specifically, gymnasts, weight lifters, and butterfly and backstroke swimmers) and in manual laborers.

There are a variety of events related to the conversion of a functionally stable, ligamentously lax shoulder to one with MDI. Precipitating events tend to be relatively atraumatic, in contrast to the magnitude of injury sustained by patients with traumatic unidirectional instability.[1,5,6] The history of onset is often related to a trivial or mild injury, a moderate injury (of insufficient violence to cause tearing of ligaments), a period of overuse or fatigue, or even disuse. Sometimes a precipitating event cannot be identified.

A relatively atraumatic onset of instability strongly suggests MDI. However, an episode of significant trauma can be a factor in a shoulder with excessive laxity. In the literature, athletes with lax shoulders constitute the majority of such patients.[7,8] In addition to MDI, these patients are occasionally found to have Bankart lesions. Neer[4] has warned that when there is a history of an initial significant traumatic event, MDI can be mistaken for traumatic unidirectional instability. If a unidirectional instability repair that tightens only one side of the capsule is performed, the shoulder could subluxate in a fixed position in the opposite direction. Failure to achieve stability and arthritis of instability are possible consequences.[4]

Pathophysiology

The anatomic "lesion" found in MDI is a large, patulous inferior capsular pouch that extends both anteriorly and posteriorly in varying degrees, creating a global increase in capsular volume. In our clinical operative experience, the rotator interval capsule in MDI is universally characterized by a defect that appears as an obvious broad cleft or as insubstantial, attenuated tissue. Experiments in cadaveric specimens involving selective division of glenohumeral capsuloligamentous structures have demonstrated that the inferior capsule and the rotator interval capsule act as restraints to inferior glenohumeral translation depending on arm position.[9,10] The inferior capsule resists inferior translation increasingly with progressive arm abduction to 90 degrees. The rotator interval capsule resists inferior translation with the arm at the side.

Because the contralateral shoulder often possesses equal laxity but remains asymptomatic, the pathophysiology of MDI seems to require factors beyond excessive capsuloligamentous laxity. The relative contribution of those factors remains controversial.

Lippitt et al[11] demonstrated that rotator cuff forces play an important role in glenohumeral stability by compressing the humeral head on the saucerlike, minimally constraining glenoid; this action is called concavity compression. The stabilizing effect of concavity compression was shown to depend on the integrity of the labrum, which deepens the glenoid socket, and the magnitude of the compressive force. Evidence suggests that concavity compression also depends on coordination of a balanced, dynamic force exerted by the rotator cuff tendons.[12] Concavity compression appears to be an especially important stabilizing mechanism during the midrange of glenohumeral motion, when the capsuloligamentous structures are slack.[11]

The glenoid is positioned by scapulothoracic motion to act as a

stable platform for the humeral head during active arm abduction.[13] Intuitively, it appears that maintaining the glenoid platform perpendicular to the direction of the net humeral force will optimize osseous contributions to glenohumeral stability as well as the mechanics of concavity compression. The importance of concavity compression and glenoid positioning may be reflected in the clinical experience that many MDI patients respond to a rehabilitative exercise program directed at improving strength and neuromotor coordination of the rotator cuff and scapular musculature.[5,14]

It is possible that known proprioceptive receptors in the glenohumeral joint capsule, in addition to providing joint-position sense, reflexively modulate rotator cuff forces during arm use to promote shoulder stability.[15,16] Patients with recurrent traumatic anterior instability appear to have deficits in joint-position sense compared with normal controls.[16] Although not proved scientifically, a defect in proprioception may be a component of the pathophysiology of MDI.

The presence of synovial fluid within the finite volume of the glenohumeral joint contributes to the formation of passive stabilizing articular adhesion-cohesion forces.[17] Also of importance is that an intact glenohumeral joint possesses negative intra-articular pressure.[18] These factors combine to create a stabilizing vacuum effect when inferior translation is imparted to the glenohumeral joint. Experimentally, when a cadaveric specimen is dissected free of muscle, the humeral head remains located, but when an aperture is made in the capsule, the humeral head demonstrates increased inferior translation.[18] The increased capsular volume in MDI and/or the presence of a true cleft in the rotator interval capsule that causes the glenohumeral joint to become "unsealed" may reduce the effectiveness of these codependent passive restraints.

One plausible hypothesis is that the provocation of MDI occurs when the system of dynamic restraint is overwhelmed, such as when the arm is unexpectedly manipulated or is fatigued due to repetitive use. The event, whether causing an identifiable episode of instability or not, results in pain and initiates a self-perpetuating cycle of increasing symptoms. When the painful shoulder is protected, muscular weakness and subtle losses of refined neuromotor coordination are thought to ensue. Disuse deconditions the dynamic restraints against glenohumeral instability, which are critical to stability in lax shoulders. With further use of a deconditioned shoulder, the patient is more prone to experiencing painful episodes of occult or frank instability, which can promote further disuse.

History

Most patients in whom MDI is diagnosed are young adults in their third decade (range, teenage to middle age). The occurrence of bilateral instability is not infrequent; in two published series,[1,5] surgery was performed bilaterally in 11% and 13% of patients, respectively. In our experience, an identified event of dislocation is not always present in the history of onset, although if a dislocation occurs, the vast majority of patients achieve a reduction on their own.

Symptoms associated with MDI are pain, varying degrees of instability, and transient neurologic symptoms in the affected extremity. The combination of these symptoms can vary considerably from patient to patient. Hawkins et al[6] have reported that the primary complaint in most patients is pain. Symptoms are most often experienced during common daily activities and tend to be easily provoked. As a result, MDI patients are often more functionally incapacitated than patients with other types of instability.

Activity-related complaints range from painful recurrent dislocations to pain without perceived episodes of instability. Between these extremes are pain associated with only a sense of shoulder "looseness" or a feeling that the shoulder begins to slip out of joint. Many patients comment on the presence of a diffuse, achy background level of constant pain. Some patients experience recurrent, transient episodes of numbness, tingling, and weakness in the affected extremity. Others have almost exclusively neurologic symptoms.

When recurrent subluxations or dislocations are apparent in the history, it is important to determine the frequency of occurrence, the amount of force involved in their causation, and the usual efforts needed to achieve a reduction. Patients tend to recount many episodes of instability related to low-demand activities and remark on the ability to effect an easy self-reduction. Specific activities and arm positions that cause symptoms should be sought in all cases, as they suggest directions of instability. For example, identifying whether carrying objects at the side causes symptoms is important because this suggests the inferior component of instability universal to MDI. It is also important to know whether recurrent dislocations occur during sleep, which represents the end stage of shoulder decompensation; in our experience, patients in whom this occurs tend to be less responsive to nonoperative forms of management.

The clinician must explore issues of voluntary control over dislocations. For patients with underlying emotional problems who purposefully cause instability events, both nonoperative and operative management will fail until the underlying emotional problems are resolved.[19] Another subset of patients who can voluntarily demonstrate a dislocation have no underlying emotional problems; these patients tend to respond to nonoperative management.

Given the varied presentations, it is not surprising that patients with MDI tend to have been seen by many physicians, have had many tests, and have been given many diagnoses. Common misdiagnoses include unidirectional instability, impingement, cervical disk disease, brachial plexitis, and thoracic outlet syndrome. The diagnosis of MDI should be entertained in the case of any young patient referred after a failed shoulder surgery, especially an instability repair.

Physical Examination

A diagnosis of MDI can be arrived at only after a careful physical examination. Because of the variable histories of MDI patients, findings on physical examination may be what first initiates the clinician's suspicion of the condition.

The patient should be inspected for muscular atrophy from both the front and the back. The normal round contour of the deltoid may instead have a squared appearance owing to inferior subluxation in the relaxed patient. Scapular mechanics should be observed during both active and resisted arcs of motion to detect altered scapular rhythm.

Because of the referred pain patterns associated with cervical spine disease, an examination of cervical ranges of motion is important in all patients seeking care for a shoulder problem. Provocation of symptoms distal to the neck should be carefully investigated and interpreted.

It is important to evaluate for signs of generalized ligamentous laxity because such signs have been reported in 45% to 75% of patients who have undergone surgery for MDI.[1,5,8] These signs include elbow hyperextension (Fig. 1), metacarpophalangeal joint hyperextension, genu recurvatum, patellar subluxation, and the ability of the abducted thumb to reach the ipsilateral forearm (thumb-to-forearm test). Clinicians must recognize generalized ligamentous laxity secondary to known connective tissue disorders, such as Ehlers-Danlos syndrome and Marfan syndrome, because to our knowledge patients with these conditions have never had successful results with soft-tissue instability repairs.[20] Patients with MDI often have an excessive passive range of glenohumeral motion.

Patient confidence and relaxation will be gained if instability tests are performed first on the asymptomatic shoulder. When performing these tests, one must recall that laxity is not instability; there is a wide spectrum of normal when assessing degrees of translation, and reproduction of symptoms is critically important. It is not uncommon to have to repeat the instability tests during several office visits because of muscle guarding. An examination under anesthesia at the time of a surgical procedure can provide a more accurate appreciation of the degree of translation.

Inferior laxity is assessed first by applying inferior traction with the arm at the side (sulcus test). This examination reflects the integrity of the rotator interval capsule.[10] In a positive test, an inferior translation of at least 1 to 2 cm occurs with the simultaneous appearance of an anterior soft-tissue dimple just beneath the acromion (sulcus sign). Occasionally, this maneuver will provoke neurologic symptoms in the affected extremity. A similar examination is performed with the arm abducted to 90 degrees and an inferior translational force being applied to the superior proximal humerus. A positive test in this position reflects redundancy of the inferior capsule.[9] Because of inadequate muscle relaxation, it is not uncommon for tests of the asymptomatic shoulder to appear more positive; nevertheless, this can be a pertinent finding supportive of a diagnosis of MDI.

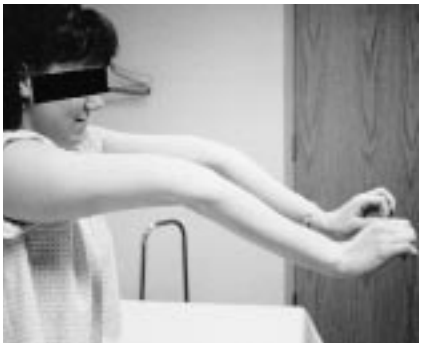

Fig. 1 The patient with MDI often has hyperextension of the elbows.

In the supine position, the patient is assessed for anterior and posterior instability with use of the load-and-shift test.[21] The shoulder is placed slightly off the edge of the examination table and is held in approximately 20 degrees of abduction in the plane of the scapula. The examiner gently grasps the proximal humerus and applies a slightly compressive load to center the humeral head on the glenoid while the free hand supports the elbow. Anterior and posterior translational forces are then applied at the proximal humerus in the plane of the gle-

noid surface. With maintenance of the slightly compressive force, the humeral head will begin to move medially when its center has translated beyond the edge of the glenoid rim. This sudden change in direction can usually be palpated by the examiner during the dislocating and/or relocating phases of translation. The extent of laxity (i.e., whether the shoulder can be subluxated or dislocated) is determined by the magnitude of the translation. It is advantageous to perform this examination in varying degrees of abduction and external rotation to effect different degrees of tension within the capsular ligaments. Normal degrees of posterior laxity allow the center of the humeral head to be translated up to half the width of the glenoid fossa, which patients with MDI usually surpass.[17]

A variation of the supine load-and-shift test can be performed with the patient seated and the arm at the side. The humeral head is centrally compressed in the glenoid fossa with the translating hand at the proximal humerus. The scapula is stabilized at the anterior and posterior aspects of the acromion with the free hand to allow accurate grading of the translation. Additional tests that can demonstrate increased translation include the Fukuda test, the push-pull test, and the jerk test.[17]

Because the examination of strength can provoke pain and spasm, it should always follow the instability assessment. The examination concludes with an assessment of sensory function and the reflexes of the peripheral nerves of the brachial plexus.

Radiologic Evaluation

Plain radiographs should be obtained to identify uncommon bone lesions, such as Bankart and Hill-Sachs lesions, and glenoid dysplasia. Because MDI is a clinical diagnosis based on the findings from the history and physical examination, we have not found any reason to order more sophisticated imaging studies.

Nonoperative Management

Nonoperative management includes patient education and a specific program of physical therapy. Patients learn that their lax shoulder has become deconditioned from its usual state and that they need to regain both strength and neuromotor coordination of the stabilizing muscles of the rotator cuff, deltoid, and scapula. To support this explanation, the patient often can be shown that the contralateral shoulder is equally loose yet functions normally without pain. Burkhead and Rockwood[14] reported satisfactory results in 29 of 33 (88%) multidirectionally unstable shoulders treated with a specific program of physical therapy.

Before the patient starts an exercise program, pain can be managed with a combination of brief immobilization, nonsteroidal anti-inflammatory drugs, and occasionally a mild analgesic. The exercise program consists of two phases. Phase I concentrates on progressive resistance exercises utilizing elastic elements for strengthening the rotator cuff and deltoid musculature. As progress is made, strengthening exercises for the scapula-stabilizing muscles are added. Phase II begins at the 10- to 12-week mark, when additional exercises are added to retrain humeroscapular coordination and awareness. Exercises are continued for a minimum of 6 months. A program of maintenance exercises is then given, to be followed indefinitely.

Surgical Management

Surgery is an option for patients who were compliant with a specific exercise program but who remain symptomatic. Surgery is not offered to voluntary dislocators with emotional problems or to behaviorally immature teenagers.

While several surgical procedures have been described, an open inferior capsular shift, as originally described by Neer and Foster,[1] is the standard procedure and continues to be the most commonly used. Additional procedures include glenoid osteotomy[22] and arthroscopic inferior capsular shift.[23] Both procedures have yielded satisfactory results; however, the literature to date is sparse. Arthroscopic, laser-assisted capsular "shrinkage" procedures remain experimental at present.

Technique for Inferior Capsular Shift

Interscalene block anesthesia is recommended because it allows the patient to stand at the completion of surgery for application of a modified shoulder spica cast. First, an examination under anesthesia is performed, followed by skin preparation and draping. An anterior approach has been used exclusively by the senior author (J.J.B.) because it is the only single incision that allows for a complete shift of the capsule, closure of the rotator interval capsule, and repair of unexpected anterior Bankart lesions.

The incision is made from the tip of the coracoid process to the apex of the axilla in line with the natural skin creases, and the deltopectoral interval is developed. The clavipectoral fascia is incised lateral to the conjoined tendon-muscle unit up to the coracoacromial ligament. The subscapularis tendon is incised sharply 1 cm medial to the lesser

tuberosity, beginning superiorly at the rotator interval. After the scalpel has incised through two thirds of the anterior thickness of the length of the tendon, it is turned coronally, and dissection is carried medially at the same tendon depth (Fig. 2, A). When the subscapularis muscle fibers are encountered, dissection deepens to remove the entire subscapularis muscle belly from the underlying capsule. Once freed, the tendon is retracted medially with traction sutures (Fig. 2, B). The rotator interval capsule defect is then imbricated in 30 degrees of external rotation with the arm at the side.

A lateral capsular incision begins at the rotator interval and extends inferiorly 2 to 3 mm lateral to the articular cartilage. Access can be gained for posterior capsule release by externally rotating and slightly flexing the adducted arm. The axillary nerve, which is relatively protected by this positioning, is kept away from the incising blade by a blunt retractor. The amount of posterior release is adjusted just enough for the shift to eliminate the posterior pouch of redundant tissue. A secondary incision is made in the capsule, aimed at the center of the anterior aspect of the glenoid (Fig. 2, C). Traction sutures are placed at the corner of each leaflet. The humeral head is retracted posteriorly with a humeral-head retractor, and the intra-articular contents are inspected. Note is made of the condition of the articular surfaces and the labral complex attachment.

A dental burr is used to decorticate the bone adjacent to the articular surface on the surgical neck of the humerus. The shift is performed with the arm in 30 degrees of abduction, 40 degrees of external rotation, and 10 degrees of flexion. The inferior flap is shifted superiorly, eliminating excessive capsular volume posteriorly and inferiorly,

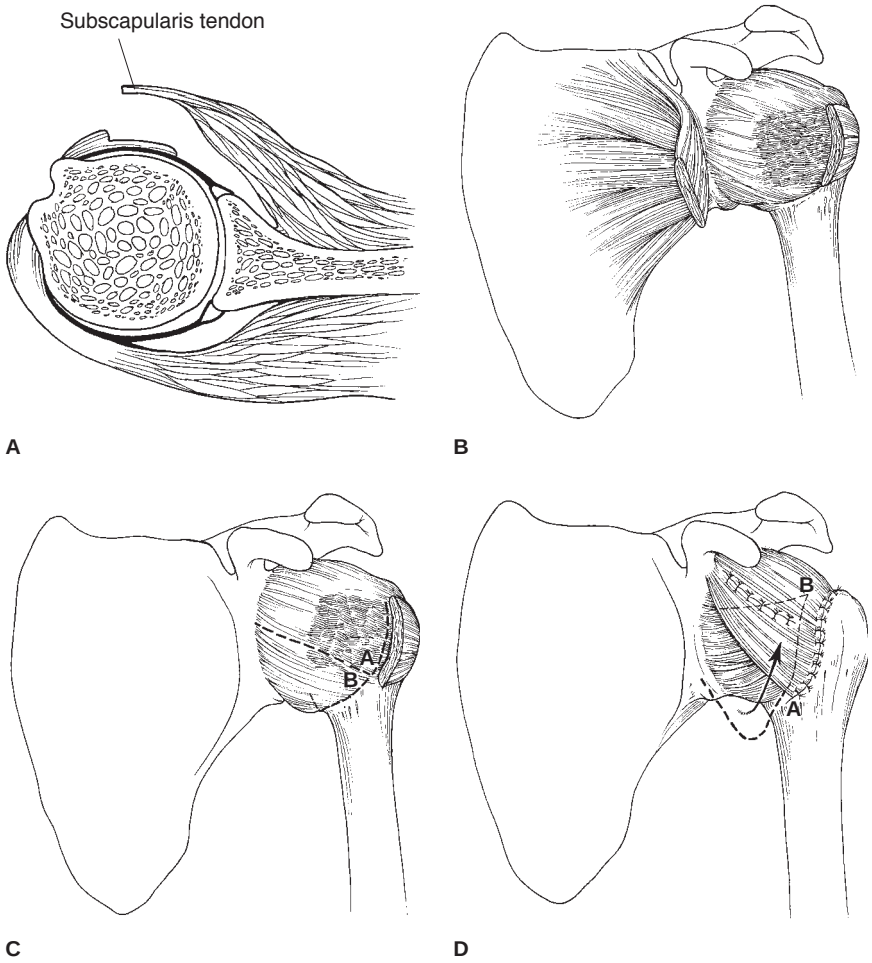

Fig. 2 **A,** The anterior two thirds of the subscapularis tendon is dissected medially, leaving the posterior portion of the tendon to reinforce the anterior capsule. **B,** The subscapularis muscle belly and the anterior portion of the tendon are retracted medially. **C,** The capsule is incised in a "T" fashion, creating superior and inferior leaflets. **D,** The capsule is advanced and shifted; the superior flap overlaps the inferior flap.

and is sutured to the cuff of preserved lateral capsular tissue. The superior leaflet is shifted inferiorly and is similarly repaired (Fig. 2, D). The subscapularis tendon is repaired at its anatomic length. Nonabsorbable suture material is used throughout these reconstructive steps.

The application of a modified shoulder spica cast is recommended because it is the most certain way to immobilize the reconstructed capsule during the acute healing phase, and it eliminates the worry of compliance with brace wear. The cast is applied with the arm in neutral rotation and in 10 to 15 degrees of abduction. To reduce potential strain on the rotator interval capsule repair, an assistant pushes cephalad on the olecranon until the cast is firm. When the cast is applied properly, the shoulder will be in a mildly shrugged position.

Aftercare

A standard protocol of postoperative exercises is used as a general outline. During the healing and

stretching phases of postoperative management, the standard protocol is adhered to rigidly for fear that rapid gains in motion will result in recurrent instability. When strengthening exercises are initiated, the program is individualized depending on the patient's progress.

The spica is removed at week 6, and a sling is provided to ease the transition from rigid immobilization. During weeks 6 to 10, activities of daily living are allowed below the level of the shoulder and within 45 degrees of external rotation. At week 10, a stretching program is begun for forward elevation (limit, 160 degrees) and external rotation (limit, 45 degrees), emphasizing gradual restoration of range of motion. At weeks 14 to 16, deltoid and rotator cuff strengthening begins. At weeks 18 to 20, exercises for the scapular stabilizers are added.

Contact sports are permitted once full strength and conditioning have been restored, usually at 10 months. Examples of activities discouraged indefinitely include wrestling, waterskiing, and certain lifting exercises, including bench presses and dips.

Outcomes

There have been only a few published reports of the results of surgical treatment of MDI. These demonstrate a high degree of patient satisfaction and subjective stability in patients treated with an open inferior capsular shift. In the original article by Neer and Foster,[1] 39 patients were reevaluated more than 1 year after surgery, of whom 17 (44%) were followed up for more than 2 years. One patient experienced recurrent anterior subluxations 7 months postoperatively. The remaining patients achieved satisfactory results, as defined by the absence of recurrent instability events or significant pain and by the return of normal strength and the ability to participate in full activities, as well as the capacity for elevation within 10 degrees of that possible in the contralateral shoulder and external rotation within 40 degrees. Three patients had neurapraxia of the axillary nerve.

Cooper and Brems[5] reported on 38 patients (43 shoulders) with a minimum follow-up of 2 years (average follow-up, 38 months). Symptomatic MDI recurred in 4 shoulders (9%) in 4 patients within 2 years of surgery; one instance of MDI was attributable to a defined event of significant trauma, and three instances presumably occurred because the repair became stretched. The remaining 34 patients were subjectively satisfied with the status of their shoulder, although 5 patients (15%) had persistent episodes of apprehension.

Bigliani et al[24] reported on surgical treatment of 49 patients with MDI. An anterior approach was used when largely anteroinferior instability was identified (34 patients) and a posterior approach was used when instability was greatest posteroinferiorly (15 patients). The results after an average follow-up interval of 5 years were satisfactory for 91% of the patients treated with an anterior approach and for 100% of the patients treated with a posterior approach.

Summary

A diagnosis of MDI is arrived at on the basis of a careful history and physical examination. Most patients can be successfully treated with a well-executed exercise program. For the minority of patients for whom nonoperative management is a failure, surgical reconstruction can be reasonably recommended. The most widely reported surgical procedure is an open inferior capsular shift. When combined with meticulous aftercare, this procedure has yielded favorable results in the relatively few series published to date.

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. Neer CS II: Dislocations, in Neer CS II (ed): *Shoulder Reconstruction*. Philadelphia: WB Saunders, 1990, pp 273-341.
3. Thomas SC, Matsen FA: An approach to the repair of glenohumeral ligament avulsion in the management of traumatic anterior glenohumeral instability. *J Bone Joint Surg Am* 1989;71:506-513.
4. Neer CS II: Involuntary inferior and multidirectional instability of the shoulder: Etiology, recognition, and treatment. *Instr Course Lect* 1985;34: 232-238.
5. Cooper RA, Brems JJ: The inferior capsular-shift procedure for multidirectional instability of the shoulder. *J Bone Joint Surg Am* 1992;74:1516-1521.
6. Hawkins RJ, Abrams JS, Schutte J: Multidirectional instability of the shoulder: An approach to diagnosis. *Orthop Trans* 1987;11:246.
7. Bigliani LU, Kurzweil PR, Schwartzbach CC, Wolfe IN, Flatow EL: Inferior capsular shift procedure for anterior-inferior shoulder instability in athletes. *Am J Sports Med* 1994;22:578-584.
8. Altchek DW, Warren RF, Skyhar JM, Ortiz G: T-plasty modification of the Bankart procedure for multidirectional instability of the anterior and inferior

types. *J Bone Joint Surg Am* 1991;73: 105-112.
9. Warner JJP, 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.
10. 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.
11. Lippitt SB, Vanderhooft JE, Harris SL, Sidles JA, Harryman DT II, Matsen FA III: Glenohumeral stability from concavity-compression: A quantitative analysis. *J Shoulder Elbow Surg* 1993;2:27-35.
12. Blasier RB, Guldberg RE, Rothman ED: Anterior shoulder stability: Contributions of rotator cuff forces and the capsular ligaments in a cadaver model. *J Shoulder Elbow Surg* 1992;1: 140-150.
13. Ozaki J: Glenohumeral movements of the involuntary inferior and multidirectional instability. *Clin Orthop* 1989; 238:107-111.
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. Blasier RB, Carpenter JE, Huston LJ: Shoulder proprioception: Effect of joint laxity, joint position, and direction of motion. *Orthop Rev* 1994;23: 45-50.
16. Lephart SM, Warner JJP, Borsa PA, Fu FH: Proprioception of the shoulder joint in healthy, unstable, and surgically repaired shoulders. *J Shoulder Elbow Surg* 1994;3:371-380.
17. Matsen FA III, Thomas SC, Rockwood CA Jr: Anterior glenohumeral instability, in Rockwood CA Jr, Matsen FA III (eds): *The Shoulder*. Philadelphia: WB Saunders, 1990,vol 1, pp 526-622.
18. 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.
19. Rowe CR, Pierce DS, Clark JG: Voluntary dislocation of the shoulder: A preliminary report on a clinical, electromyographic, and psychiatric study of twenty-six patients. *J Bone Joint Surg Am* 1973;55:445-460.
20. Jerosch J, Castro WHM: Shoulder instability in Ehlers-Danlos syndrome: An indication for surgical treatment? *Acta Orthop Belg* 1990;56:451-453.
21. Hawkins RJ, Bokor DJ: Clinical evaluation of shoulder problems, in Rockwood CA Jr, Matsen FA III (eds): *The Shoulder*. Philadelphia: WB Saunders, 1990, vol 1, pp 149-177.
22. Nobuhara K, Ikeda H: Glenoid osteotomy for loose shoulder, in Bateman JE, Welsh RP (eds): *Surgery of the Shoulder*. Philadelphia: BC Decker, 1984, pp 100-103.
23. Duncan R, Savoie FH III: Arthroscopic inferior capsular shift for multidirectional instability of the shoulder: A preliminary report. *Arthroscopy* 1993; 9:24-27.
24. Bigliani LU, Pollock RG, Owens JM, McIlveen SJ, Flatow EL: The inferior capsular shift procedure for multidirectional instability of the shoulder. *Orthop Trans* 1993;17:576.