Generalized joint laxity and multidirectional instability of the shoulder

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Abstract

Generalized joint laxity and shoulder instability are common conditions that exhibit a wide spectrum of different clinical forms and may coexist in the same patient. Generalized joint laxity can be congenital or acquired. It is fundamental to distinguish laxity from instability. Laxity is a physiological condition that may predispose to the development of shoulder instability. A high prevalence of generalized joint laxity has been identified in patients with multidirectional instability of the shoulder. Multidirectional instability is defined as symptomatic instability in two or more directions. The diagnosis and treatment of this condition are still challenging because of complexities in its classification and etiology. These complexities are compounded when multidirectional instability and laxity exist in the same patient. With an improved understanding of the clinical symptoms and physical examination findings, a successful strategy for conservative and/or surgical treatments can be developed. Conservative treatment is the first-line option. If it fails, different surgical options are available. Historically, open capsular shift has been considered the gold standard in the surgical management of these patients. Nowadays, advanced arthroscopic techniques offer several advantages over traditional open approaches and have shown similar outcomes. The correct approach to the management of failed stabilization procedures has not been yet defined.

Key Words: arthroscopy, generalized joint laxity, multidirectional instability, open capsular shift, shoulder.

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Generalized joint laxity

Generalized joint laxity is characterized by increased length and elasticity of normal joint restraints, resulting in a greater degree of translation of the articular surfaces. This is detectable as an increased range of motion and increased distractibility (1).

This hyperlaxity can be congenital and acquired. Congenital hyperlaxity is usually caused by connective tissue disorders, such as Ehlers-Danlos syndrome, Marphan syndrome, osteogenesis imperfecta, and benign hypermobility syndrome (2, 3). However, it is not necessarily related to a pathological condition. The prevalence of non-pathological hyperlaxity in the general population is between 5 and 15% (4, 5). It becomes less common as individuals age and is slightly more prevalent in females than in males (6, 7). Acquired joint hyperlaxity is commonly observed in athletes (swimmers, gymnasts, pitchers, etc.) (8-10). In this specific population, repetitive microtrauma or repetitive use during training and competitions result in stretching of the normal capsuloligamentous restraints. Male and female athletes are equally affected (1).

Generalized joint laxity does not require any treatment.

Generalized joint laxity and unidirectional instability of the shoulder

Hyperlaxity must be distinguished from instability. Shoulder laxity is the physiological presence of asymptomatic translation of the shoulder joint. There is a wide spectrum of asymptomatic laxity in the anterior, posterior and inferior planes (11). Shoulder instability, on the other hand, is a pathological condition, characterized by the presence of symptoms in conjunction with abnormal laxity, which is indicative of deficient static and dynamic glenohumeral stabilizers (12, 13). Several studies suggest that individuals with generalized joint laxity are at risk of musculoskeletal injuries (14-17). A clear relationship has been demonstrated between generalized joint laxity and both knee (16, 17) and ankle (14, 15) injuries.

The association between generalized joint laxity and anterior shoulder instability continues to be debated. Although there is no gold standard to define hyperlaxity, numerous clinical scoring systems have been proposed. Chahal et al. (18) examined hyperlaxity and shoulder external rotation greater than 85° in 57 patients who sustained a primary traumatic anterior shoulder dislocation and 92 age-matched students without a history of shoulder or knee injuries. The Hospital Del Mar score (19) was used to assess hyperlaxity. It ranges from 0 to 10 and is derived by assigning one point for each of the following criteria:

1. passive hyperextension of the metacarpophalangeal joint of the little finger of 90° or more;
2. passive apposition of the thumb to the flexor aspect of the forearm at less than 21 mm;
3. passive elbow hyperextension of 10° or more;
4. passive shoulder external rotation of 85°or more;
5. passive hip abduction of 85°or more;
6. hyperextension of the first metatarsophalangeal joint beyond 90°;
7. patellar hypermobility, defined as excessive passive displacement medially and laterally as assessed by three or more quadrants of displacement;
8. excessive range of passive ankle dorsiflexion and eversion of the foot with the knee flexed to 90°;
9. passive knee hyperflexion, defined as 'knee makes contact with the buttock';
10. appearance of ecchymoses after hardly noticed, minimal trauma (historical datum).

A score of 4/10 or higher for men and 5/10 or higher for women suggests the presence of generalized joint laxity. The authors (18) showed that generalized joint laxity and increased external rotation are predisposing factors for anterior shoulder dislocation.

Cameron et al. (20), applying the Beighton criteria (21), confirmed the association between hyperlaxity and shoulder instability. The Beighton scale comprises five items:

1. passive dorsiflexion of the fifth finger beyond 90°;
2. passive thumb opposition to the forearm;
3. active elbow hyperextension beyond 10°;
4. active knee hyperextension beyond 10°;
5. forward flexion of the trunk with knees fully extended so that the palms of the hands rest flat on the floor.

Each item except for trunk flexion is scored bilaterally and the total score ranges from 0 to 9. The authors showed that patients with a Beighton score of 2 or greater were nearly 2.5 times more likely to have experienced an episode of shoulder instability than patients with lower scores.

The Beighton scale is the most popular scoring system for the clinical assessment of hyperlaxity, but unfortunately its cutoff point remains arbitrary.

A recent study by Ranalletta et al. (22) assessed hyperlaxity in 100 male patients affected by recurrent anterior dislocation and 100 age-matched males without a history of shoulder instability. A Beighton score greater than six was considered indicative of hyperlaxity. No differences were identified in the rate of generalized joint laxity between the two groups.

Shoulder instability occurs when the normal stabilizing mechanisms are disrupted. Patients with anterior traumatic shoulder instability without hyperlaxity can experience recurrent dislocations or subluxations due to a structural injury of the capsulolabral complex or secondary glenoid or humeral head bone loss. By contrast, patients affected by shoulder instability with hyperlaxity are more likely to experience recurrent subluxations than frank dislocations (9, 10, 23). They can develop structural lesions of the capsulolabral complex, but do not usually show any secondary osseous lesions.

Joints

A careful physical examination must be performed in order to ascertain the direction of the instability and the possible presence of shoulder hyperlaxity and generalized joint laxity.

The direction of instability can be determined by identifying the provocative position that reproduces instability symptoms. Several tests are commonly used: apprehension test, relocation test, anterior and posterior drawer, anterior and posterior load and shift (24). Inferior shoulder laxity is best evaluated using the sulcus sign test (25) and Gagey test (26). The sulcus sign test is performed with the arm in 0 to 20° of abduction, and both neutral and 30° of external rotation. Humeral head displacement greater than 1 cm from the acromion is considered indicative of a high degree of inferior laxity, but is not considered abnormal unless it reproduces the patient's symptoms (pain and symptoms of instability). The same test with the arm in 30° of external rotation is specific for deficiency of the rotator interval.

The Gagey test is indicative of inferior laxity if the passive abduction is greater than 105° or if there is marked asymmetrical hyperabduction, i.e. a greater than 20° difference, when compared with the abduction on the contralateral side. Anterior laxity is defined by an excessive external rotation, greater than 85°, with the arm at the subject's side (27, 28).

As mentioned above, there exist numerous clinical scoring systems for assessing generalized joint laxity. The most common is the Beighton scale. Although there is no formal consensus, a Beighton score of four or more is usually considered indicative of hyperlaxity (1).

Radiographs and computed tomography (CT) are commonly used to evaluate the presence of osseous defects or bony abnormalities; magnetic resonance imaging (MRI) and magnetic resonance arthrography (MRA) offer detailed information about capsulolabral structures, the long head of the biceps, and the rotator cuff. MRA is the most sensitive and specific imaging modality for assessing labral pathologies, capsular tears and volume.

Unidirectional shoulder instability with hyperlaxity always requires surgical treatment. Open and arthroscopic techniques showed comparable results in patients without osseous defects (29). The advantages of arthroscopic treatment include less surgical dissection, easier treatment of concomitant pathologies, and easier access to the posterior capsulolabral complex, if necessary. The aim is to create a stable joint by fixing labral lesions and reducing laxity and excessive volume through retensioning of the static capsulolabral stabilizers. A capsulolabral repair with anchors will be performed on the side of the instability and additional capsular plications on the opposite side may be also performed, depending on the degree of capsular laxity.

A high prevalence of hyperlaxity has been reported in recurrent instability after both open and arthroscopic stabilization procedures. The prevalence of hyperlaxity in failed open procedures, including Bankart repair, capsular shift, Latarjet procedures and bone block augmentation, ranges from 61 to 100% (28, 30-32).

Recent studies also showed a significant association between hyperlaxity and recurrence of instability after arthroscopic procedures (27, 33). Voos et al. (33) evaluated 83 patients who underwent arthroscopic Bankart repair with suture anchors at an average follow-up of 33 months (range, 24-49 months). They showed that patients with hyperlaxity have a 3.3-fold higher risk of recurrent instability than patients without hyperlaxity.

Multidirectional instability

In the 1980s, Neer and Foster coined the term "multidirectional instability" (25), defined as symptomatic involuntary instability of the glenohumeral joint in more than one direction (anterior and/or posterior, and inferior).

The first challenge is to identify and classify patients with multidirectional instability (MDI). Key points to consider in this regard are: the etiology (traumatic, atraumatic, microtraumatic) and the amount of symptomatic translation and volitional control (voluntary and involuntary instability). We classify MDI, as follows:

• MDI without hyperlaxity;
• MDI with hyperlaxity;
• Voluntary instability with hyperlaxity.

Multidirectional instability without hyperlaxity

MDI without hyperlaxity is a very rare condition, usually associated with a traumatic onset. Physical examination will show shoulder instability in anterior and posterior directions. Tests for inferior laxity in the affected shoulder or anterior and inferior laxity in the contralateral shoulder will be negative. CT and MRI will be helpful in the detection of osseous and capsulolabral lesions, respectively. Patients without significant bony abnormalities will be eligible for an arthroscopic treatment. An anterior and posterior capsulolabral repair with anchors will be performed according to the extent of the capsulolabral lesions.

Multidirectional instability with hyperlaxity

The etiology of MDI in patients with hyperlaxity can be atraumatic or due to repetitive microtrauma.

Atraumatic MDI is usually characterized by pain and a sensation of instability. Symptoms develop gradually in these patients: at the beginning, pain during high-demand activities or provocative positions can be the only complaint; subsequently, instability symptoms may progress with subluxations and/or dislocations occurring during activities of daily living.

Repetitive microtrauma is the most common etiology in patients involved in repetitive overhead activities, particularly in sports such as volleyball, swimming and gymnastics. An accumulation of shear forces caused by persistent shoulder subluxation or microtrauma leads to a loss of chondrolabral containment with subsequent development of labral injuries (12, 13).

The prevalence of generalized joint laxity in patients with MDI and shoulder laxity ranges between 40 and 70% (12, 25, 34, 35).

Shoulder stability is guaranteed by the interaction of static and dynamic stabilizers. Bony and capsulolabral ligamentous structures are the primary static restraints of the shoulder. Insufficiency of these structures places higher demands on other shoulder stabilizers.

Dynamic stabilization is provided by the rotator cuff, long head of the biceps and scapula stabilizers.

MDI in patients with hyperlaxity is usually characterized by a patulous and redundant capsule, increased glenohumeral volume and labral injuries. Recent studies also reported abnormal scapular kinematics and an atypical pattern of muscle activity, suggesting that neuromuscular control is a contributing factor in the etiology of multidirectional instability (36-38).

The diagnosis of MDI is primarily clinical. Patients are usually young, between the second and the third decade of life. They may present with a frank history of instability or, more often, with an insidious onset and aspecific activity-related pain exacerbated by positions that can provoke instability such as throwing (anterior instability), carrying heavy loads (inferior instability) or pushing (posterior instability).

Decreased strength and declining athletic performance may also be reported. Inferior instability can be also be associated with numbness and tingling, secondary to traction on the brachial plexus, elicited when carrying heavy objects (39). Correlation of symptoms with the arm position is important to ensure the correct diagnosis. A comprehensive physical examination is one of the most important aspects. Both shoulders should be evaluated, observing any asymmetry, abnormal motion, muscle atrophy and scapular winging. Specific tests assessing the direction of instability and the degree of shoulder laxity must be performed. Moreover, generalized joint laxity must be also evaluated. Failure to address all the components of instability may result in treatment failure. Patients affected by MDI usually show positive instability tests in more than one direction, a positive sulcus sign and a positive Gagey sign. CT and MRI or MRA are helpful to further delineate structural injuries.

In patients without significant bony abnormalities, non-operative treatment is the mainstay of the therapeutic approach. The goal is to rehabilitate deficient structures to restore stability and alleviate symptoms. This form of treatment does not affect the static joint restraints, but it improves tone and proprioceptive control of the dynamic stabilizers. The focus is on strengthening of the rotator cuff and periscapular muscles in order to maximize the concavity-compression mechanism and stabilize the glenoid platform (40). The result is improved humeral head centering and more robust opposition to shear forces. A minimum of six months is a reasonable duration for a first-

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line treatment. Several studies showed variable results with success rates between 30 and 80% (41-43). Surgical treatment should be considered after failure of conservative treatment or in the presence of bony abnormalities. It should be individualized in order to address the specific anatomical cause of the instability and reduce the shoulder hyperlaxity to obtain a more normal functional range of motion.

A recent study (44) compared the kinematic parameters and muscle activity of patients with MDI treated by physiotherapy or by surgical treatment and postoperative physiotherapy, before and after treatment. The authors showed that the significant alteration in shoulder kinematics observed in these patients cannot be restored by physiotherapy alone. Surgical treatment and physiotherapy allow bilinear scapulothoracic and glenohumeral rhythms and restoration of the normal relative displacement between the rotation centers of the scapula and humerus. Moreover, the duration of muscular activity was found to be almost normal, after surgery and postoperative rehabilitation.

Historically, open capsular shift has been the standard in the operative treatment of patients with multidirectional instability. A humeral-based (or lateral-based) capsular shift was first described by Neer and Foster in the 1980s (25). Subsequent cadaveric studies showed a significant capsular volume reduction, between 50 and 60% (45-47). Miller et al. (48) compared volume reduction of the glenohumeral joint using three different open capsular shift techniques: the humeral-based T-shaped capsular shift as described by Neer and Foster (25), the glenoid-based (or medial-based) T-shaped capsular shift as described by Altcheck et al. (34), and a vertical capsular imbrication as described by Wirth et al. (49). The humeral-based T-shaped capsular shift resulted in a greater significant reduction than the glenoid-based shift (48.9% *vs* 36.8%).

In recent years, attention has been focused on arthroscopic techniques. An arthroscopic inferior capsular shift is essentially an arthroscopic modification of the glenoid-based open inferior capsular shift described by Altcheck et al. (34). Cadaveric studies comparing open and arthroscopic capsular shift reported conflicting results. Cohen et al. (50) found that arthroscopic capsular plication using three 1-cm capsulolabral plication sutures resulted in a volume reduction of 22%, whereas humeral-based capsular shift reduced joint volume by nearly 50%. By contrast, Sekiya et al. (51) showed that arthroscopic plication resulted in a mean decrease of 58 ± 12%, whereas open inferior capsular shift resulted in a mean difference of 45 ± 11% (p=0.006). Subsequent studies showed that capsular volume reduction is related to the amount of capsular plication; therefore, the volume reduction increases when the arthroscopic capsulolabral plication is increased from 5 to 10 mm (52, 53). Yamamoto et al. (54) confirmed that the glenoid-based capsular shift decreases joint volume and increases responsiveness of intra-articular pressure to downward loading. Recently, Ponce et al. (55) noted that a 1-cm capsular plication stitch results in a roughly 10% volume reduction of the glenohumeral joint; therefore, five simple capsular plication stitches result in a volume reduction equivalent to an open humeral-based capsular shift.

Only one clinical study (56) reported joint volume reduction following arthroscopic capsular shift. The authors applied the same technique both in cadaveric specimens and in a clinical setting. They reported 37.9% of volume reduction in the cadaveric model and 58.8% of volume reduction in the clinical setting. However, clinical studies reported success rates of 80 to 97% after open capsular shift (25, 34, 35, 57). High success rates, ranging between 68 and 100%, have also been reported after arthroscopic capsular shift (58-60). A recent systematic review (61) comparing open capsular shift *versus* arthroscopic capsular plications showed that arthroscopic capsular plications yield comparable results to open capsular shift with regard to recurrent instability, return to sport, loss of external rotation, and overall complications.

Arthroscopic treatment is essentially based on the intraoperative findings and includes different techniques: pancapsular plications, capsulolabral repair with suture anchors, and rotator interval closure. Before the procedure is begun, the direction and magnitude of the glenohumeral translation are confirmed by examining the patient under anesthesia. A diagnostic arthroscopy is performed to identify the patulous capsule and possible labral injuries (**Fig. 1**). MDI may exist with or without labral tears (12), but large circumferential labral tears, termed the "triple labral lesion", have been described (62). Therefore, a capsulolabral
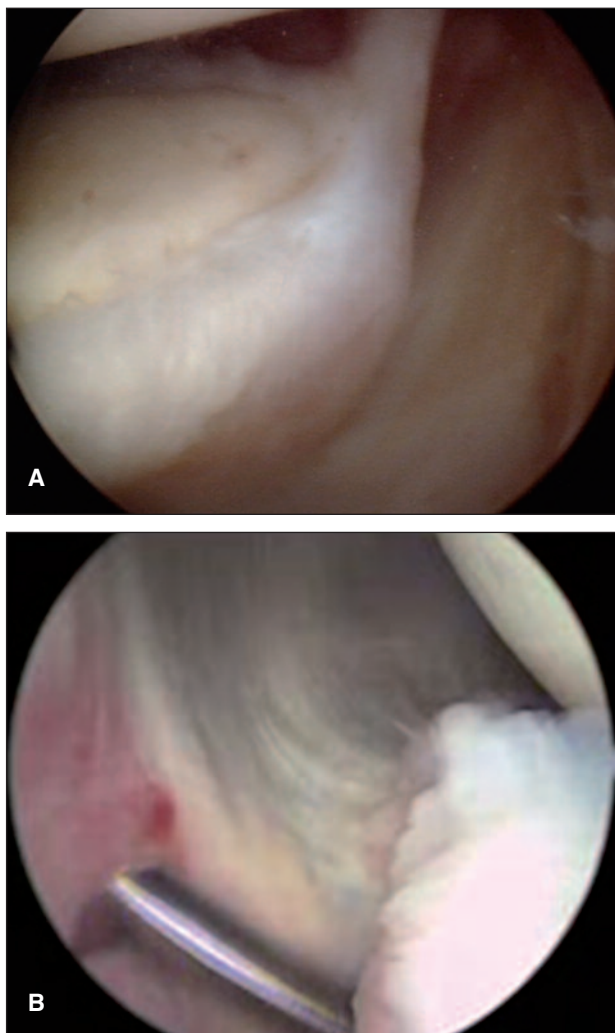
Fig. 1. MDI of a left shoulder. Diagnostic arthroscopy identifies the patulous capsule at the superior (A) and inferior (B) aspect of the joint.

Fig. 2. Capsular plications are performed to decreases the volume of the shoulder capsule using the labrum as the fixation point of the sutures.

repair with suture anchors can sometimes be needed. Suture anchor repairs use the glenoid as the fixation point. By contrast, capsular plication, a surgical technique that imbricates and decreases the volume of the shoulder capsule with a suture, uses the labrum as the fixation point (63) (**Fig. 2**). It is generally believed that capsular plications are sufficient if the labrum is completely intact. However, although it has been shown that the labrum is a solid fixation point (64), concerns about shear stress, an unrecognized Kim lesion (**Fig. 3**) and suture breakage make anchor fixation preferable because it remains the strongest and most predictable repair construct. The sequence of repair is dictated by
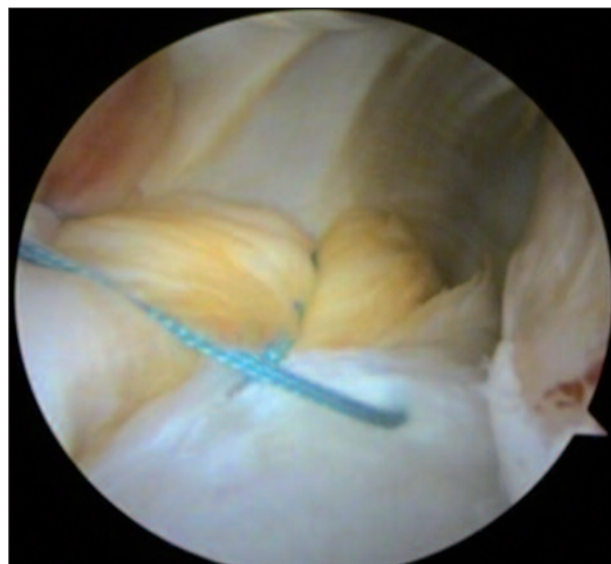
the primary direction of instability and the extent of the capsulolabral pathology. Considering that each plication decreases capsular volume and the size of the working area, inferior pathology is usually treated first. The final repair construct should produce a glenoid bumper providing sufficient tension along the tissue without capsular redundancy.

The role of the rotator interval in the pathoanatomy of MDI has long been debated. It has been suggested that the capsule and the structures of the rotator interval can limit the postero-inferior translation (65), therefore a rotator interval closure can be an additional procedure performed in the treatment of MDI (**Fig. 4**). Biomechanical studies reported a reduction of anterior translation and external rotation (66, 67). Only one recent study (68), which compared a superior-inferior closure with an arthroscopic medial-lateral closure, reported a reduction of posterior translation after medial-lateral closure. Clinical studies showed high success rates when rotator interval closure was associated with capsular plications or Bankart repair (69, 70). Although the results of operative treatment show high success rates, treatment of a failed stabilization procedure is still challenging, as there exists no clear algorithm. Two recent reviews suggest simply performing a new comprehensive review of the patient's history, physical examination and diagnostic imaging (71, 72);
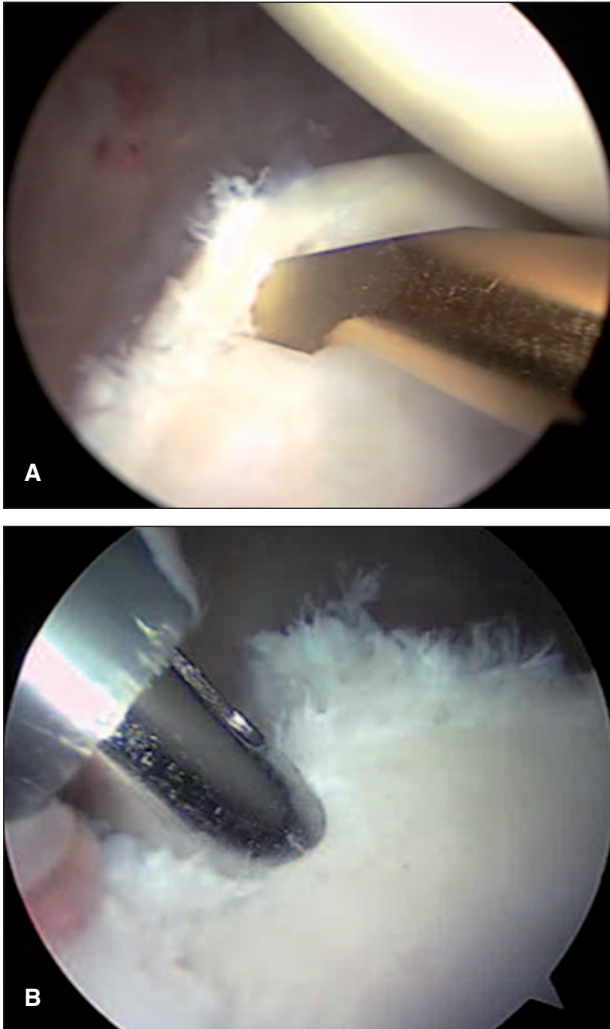
Fig. 3. Kim lesion (A) is contraindicated for capsular plications. Anchor fixation (B) is preferable because the repair construct is stronger and more predictable.

Fig. 4. Rotator interval closure can be an additional procedure performed in the treatment of MDI.

therefore, reassessment of the direction of instability, shoulder laxity, generalized joint laxity, sports activities and presence of glenoid and humeral head osseous defects. Arthroscopic techniques could still be appropriate, in patients without osseous defects, to treat residual capsular laxity or labral injuries. However, there is also a role for the open capsular shift. It is usually indicated for patients with specific soft-tissue pathology, including humeral avulsion of the glenohumeral ligaments, midsubstance capsular rupture and capsular deficiency. Moreover, although uncommon in traumatic instability or multidirectional instability, in the presence of significant osseous defects, open reconstructi-
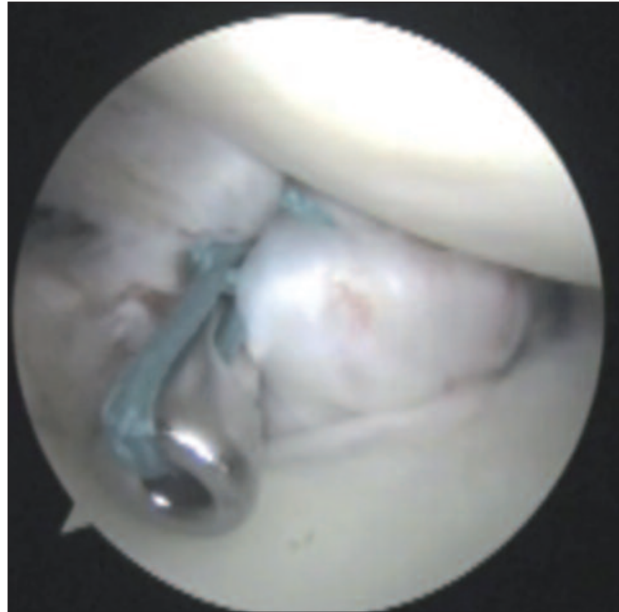
ve techniques, such as Latarjet or bone block procedures, must be performed in addition to an open capsular shift.

Voluntary instability with hyperlaxity

Voluntary shoulder instability is characterized by a patient's ability to subluxate or dislocate her/his shoulder using selective muscle contraction and relaxation. This pathological entity was first described by Rowe in 1973 (73). Most of the patients in that report had ligamentous laxity.

The clinical signs and symptoms of voluntary instability and the treatment algorithm for affected patients have not yet been clearly defined. Conservative treatment, based on strengthening of the rotator cuff and periscapular muscles, is usually the first-line treatment. Surgical treatment is recommended after failure of non-operative treatment. Only two studies reported the outcome of surgical treatment (74, 75). Fuchs et al. (74) reported the results of a posterior capsular shift procedure in a consecutive series of twenty-six shoulders affected by involuntary as well as voluntary posterior instability. Seven shoulders had previously been

operated on. At an average follow up of 7.6 years (range, 1.8 to 14.6 years) subjective results were excellent for sixteen shoulders, good for eight, and fair for two. Recently, Greiwe et al. (75) reported good and excellent results after arthroscopic capsular shift in a group of ten patients with voluntary instability.

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