Abstract: The wide spectrum of shoulder instability is difficult to include in 1 classification. The distinction between traumatic, unidirectional, and atraumatic multidirectional instability is still widely used, even though this classification is not sufficiently precise to include all the different pathological findings of shoulder instability. We present “minor instability,” which is a pathological condition causing a dysfunction of the glenohumeral articulation, especially in combination with microtrauma, repetitive or not, or after a period of immobilization or inactivity. When “minor shoulder instability" is suspected, the patient’s history and detailed clinical examination represent the most important factors when establishing the diagnosis. In particular, the apprehension test stressing the middle glenohumeral ligament (MGHL)/labral complex in the position of midabduction and external rotation may be painful and may even reveal anterior instability or subluxation. Conventional radiographs are negative in most cases, as is magnetic resonance imaging arthrography. It is only after an accurate arthroscopic assessment that the pathological lesion can be found. The major pathological process can be identified at the level of the anterior superior labrum, in particular the MGHL complex, and appears as hyperemia, fraying, stretching, loosening, thinning, hypoplasia, or even absence. It may, however, be difficult to distinguish between a normal variant and a pathological lesion. Clinical symptoms and examination should always be correlated with arthroscopic findings. Recommended treatment is to restore shoulder stability and thereby prevent shoulder pain secondary to the increase in laxity. A reduction in range of motion should be expected during the postoperative phase, at least up to six to nine months. External rotation is usually permanently reduced by a few degrees. Key Words: Shoulder—Instability—Labrum—Subluxation—Middle glenohumeral ligament.

R ecent advances in arthroscopic shoulder surgery have added enormously to the information relating to the pathological anatomy of shoulder instability. Traditionally, glenohumeral joint instability has been regarded as either TUBS (traumatic unidirectional Bankart lesion, responds to surgery)\(^1\) or AMBRII (atraumatic, multidirectional, bilateral, responds to rehabilitation, inferior capsular shift, and interval closure).\(^1,2\) This classification is still meaningful, but it is not comprehensive enough to include all the different kinds of shoulder instability. It has become evident that there is a large spectrum of instability patterns between these 2 conditions, which cannot be classified strictly as either TUBS or AMBRII. In particular, there is a group of “subtle” conditions, which may be identified as “minor shoulder instability,” that are responsible for pain and dysfunction in the shoulder.\(^3\)

Minor shoulder instability is defined as shoulder pain secondary to shoulder laxity, which cannot be defined as TUBS or AMBRII. Minor shoulder insta-
cludes an acquired instability in overstressed shoulder (AIOS)\textsuperscript{4-7} and an atraumatic minor shoulder instability (AMSI).\textsuperscript{7} AIOS may represent shoulder subluxation, anteroinferior subluxation, dead arm syndrome (in throwing athletes), or chronic microtrauma associated with capsular laxity.

In patients with TUBS, the common finding is the typical Bankart lesion, as well as the Hill-Sachs lesion, whereas in AMBRI no obvious structural lesions are found in a joint with thin, weak structures and large capsular volume. AIOS describes a pathological process, related to overstretch (chronic microtrauma), involving the superior half of the capsular-ligamentous complex. In AMSI, anatomic variants of the middle glenohumeral ligament (MGHL) are expected.

AIOS occurs most frequently in overhead athletes (ie, pitchers, volleyball players, and tennis players) or in heavy “overhead young workers,” such as builders, painters, and forklift drivers. Both static and dynamic shoulder stabilizers are of major importance because dysfunction in either of these will lead to the overload and failure of its counterpart, with subsequent minor glenohumeral instability. Different theories exist in the literature as to why these overhead activities can lead to AIOS. Townley\textsuperscript{8} noted that the instability was related to the dysfunction of the MGHL. Andrews et al.\textsuperscript{9} showed that overhead athletes who have excessive external rotation plus tightness in internal rotation develop injuries to the superior labrum and anteroinferior glenoid rim in the absence of a complete anterior capsular-labral detachment. Harryman et al.\textsuperscript{10} noted that a posterior capsular retraction results in superior translation of the humeral head. Jobe\textsuperscript{11} and Walch et al.\textsuperscript{12} believe that a recurrent abduction/external rotation movement leads to the progressive weakening of the anterior-inferior translation of the humeral head. Savoie et al.\textsuperscript{13} have shown that microtrauma in midabduction/external rotation can lead to the detachment of the MGHL. Burkhart and Morgan\textsuperscript{14} proposed that abduction/external rotation might stress the bicipital anchor to the posterior glenoid labrum (peel-back mechanism). The SLAP lesion is thus responsible for subtle posterior-inferior instability, which can mimic anterior-inferior pseudolaxity. Moreover, the presence of contracted posterior inferior capsule results in inappropriate translation of the humeral head and injury to the biceps anchor. We believe that traction applied to the insertion slowly enlarges the sublabral foramen and changes the tension of the MGHL, which will contribute to the development of minor instability. In the presence of a sublabral foramen and if fraying and/or the detachment of the anterior biceps anchor is noted, the traction of the cordlike MGHL will stretch the attachment and damage the anchor. This lesion may in fact evolve to become a SLAP lesion in overhead athletes. In addition, given enough time, repetitive overhead microtrauma may stretch the MGHL causing subtle anterior instability and posterior-superior impingement.

The MGHL is the primary anterior stabilizer at 45° of abduction and limits external rotation. In subjects who perform overhead activities, this ligament can fail, leading to abnormal anterior translation and AIOS.

AMSI is a rare condition and very seldom discussed in the literature. Patients complain about shoulder pain after a period of inactivity such as pregnancy or immobilization. This group of patients does not generally display generalized joint laxity. These patients may have static anatomic variants of MGHL (absence, hypoplasia, or a large sublabral hole or Buford complex),\textsuperscript{15-18} Equilibrium is disturbed, congenital “insufficient” static stabilizers are overloaded, and symptoms develop.

Clinical Findings

Patients with minor shoulder instability complain of pain in the posterior-superior aspect of the affected, generally dominant, shoulder. Sometimes, the pain radiates toward the arm. The pain is often diffuse and difficult to pinpoint. Patients describe snapping and popping, “dead arm,” painful subluxation, or transient locking.

On examination, range of motion testing in patients with AIOS reveals increased external rotation in abduction combined with reduced internal rotation. Stressing the MGHL-labral complex in midabduction (between 45° and 80°) and external rotation causes pain or apprehension. The load and shift or fulcrum test can reveal increased translation and crepitus. Strength testing typically reveals no deficits. However, the Jobe test,\textsuperscript{19} Whipple test,\textsuperscript{20} and Yocum test\textsuperscript{21} may sometimes be positive. Positivity of these tests is caused by a painful reaction, but it may also be caused by irritation of the rotator cuff related to an internal impingement that can be associated with an anterior microinstability. In some patients, the Neer’s impingement test\textsuperscript{22} can also be positive, mimicking subacromial impingement. The actual cause is superior and posterior translation of the humeral head, causing subacromial bursitis and injury to the bursal side of the rotator cuff (internal impingement). Tests stressing the bicipital anchor, such as the O’Brien active compression test (which is best for evaluating an anterior type II SLAP),\textsuperscript{23} crank test,\textsuperscript{24} anterior slide test,\textsuperscript{25} biceps
load test, or forced shoulder abduction and elbow flexion test, can be positive.

Imaging studies are of little value; conventional radiographs are negative in most cases, as is magnetic resonance imaging and magnetic resonance imaging with arthrography. We propose the Castagna test for minor instability. The patient is positioned with 45° of glenohumeral abduction. The arm is maximally externally rotated. Posterior/superior pain is associated with a loose anterior joint capsule and MGHL. If pain is relieved with relocation, this represents a positive Castagna test (Fig 1A and B). The Castagna test is similar to the Jobe relocation test except that the Jobe test is performed with the arm at 90° abduction.

**TREATMENT**

Variants of the MGHL should be evaluated carefully in presence of minor shoulder instability, in particular when another associated lesion is found. An indirect sign of minor instability is the distance between the long head of the biceps and the rotator cuff. This distance increases in patients with minor shoulder instability because of the relative superior displacement of the humeral head related to insufficiency in the MGHL complex. Capsular volume can appear to be increased in these shoulders, and the drive-through sign may be positive. In the presence of a large sublabral hole, the superior labrum should be examined carefully. If fraying and looseness are found, it is likely that traction is slowly damaging the labral insertion, thus enlarging the sublabral foramen, affecting the tension on the MGHL, and contributing to the development of minor instability. In the presence of a Buford complex and if fraying and/or detachment of the anterior biceps anchor is noted, it is likely that traction of the cordlike MGHL will stretch the biceps attachment and damage the biceps anchor. This lesion may in fact evolve to a SLAP lesion in overhead athletes. In patients with AMSI, an insufficient MGHL may be the only pathology. A stretched or loose ligament can be assessed by using the arthroscopic probe or with dynamic testing (humeral head translation and rotation).

Once diagnosis of pathological condition is done, surgical treatment is recommended. Surgeons treating these lesions should have a good knowledge of capsulolabral shoulder anatomy. The type of abnormality should be well recognized and surgical treatment must be tailored to the specific injury to avoid causing a stiff shoulder. The anatomy and pathology of the superior glenoid rim have become better defined with the development of arthroscopy. Normally, in 70% of cases, the MGHL appears as a folded thickening in the anterior capsule that crosses the subscapularis tendon at the 45° angle to insert on the anterior-superior neck of the glenoid. Normal variants of MGHL are recognized and well described in the literature. The MGHL ligament can appear as a cordlike ligament (smooth ropelike structure) that can attach as normally at the neck of the glenoid superiorly to the anterior-superior rim or associated with a sublabral...
Minor shoulder instability may be caused by microtrauma (AIOS) or anatomic variants combined with muscle atrophy as a result of immobilization (AMSI). The lesion involves the MGHL. An arthroscopist must distinguish between normal variants and pathological findings. When nonsurgical treatment fails, arthroscopic plication and a shift of the anterior capsule represents a MGHL reconstruction. Surgical stabilization results in resolution of primary minor shoulder instability and secondary impingement.

CONCLUSION

Minor shoulder instability may be caused by microtrauma (AIOS) or anatomic variants combined with muscle atrophy as a result of immobilization (AMSI). The lesion involves the MGHL. An arthroscopist must distinguish between normal variants and pathological findings. When nonsurgical treatment fails, arthroscopic plication and a shift of the anterior capsule represents a MGHL reconstruction. Surgical stabilization results in resolution of primary minor shoulder instability and secondary impingement.

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