Introduction

Glenohumeral dislocations and subsequent shoulder instability represent a frequent and well-studied pathology [35, 86]. While there is a broad consensus throughout the literature concerning the definition of this pathology, therapeutic strategies in certain patient subgroups remain subject to ongoing controversy. Within the dominating entity of anteroinferior shoulder instabilities, which account for approximately 74–97% of cases [34, 35, 55], the treatment of patients presenting with concomitant supraphysiological joint laxity (defined as hyperlaxity) remains challenging and controversially discussed. In general, hyperlaxity has been identified as an independent risk factor for the incidence of recurrent shoulder instability with an odds ratio of 2.68 [53, 54] and can be found in approximately 13% of patients suffering from first-time shoulder dislocations [34]. Furthermore, hyperlaxity has been shown to be predictive for failure of arthroscopic Bankart repair in both primary [5, 10, 62, 77] and revision cases [72, 75]. Consequently, hyperlaxity is included in risk scoring systems as a major factor predictive of failure of arthroscopic Bankart repair [5]. To mitigate this risk, a reliable diagnosis and appropriate treatment are crucial for the successful management of this high-risk patient population. The present article aims to elucidate the distinct clinical patterns of patients suffering from anteroinferior shoulder instability with concomitant multidirectional hyperlaxity and provides a current concept of an arthroscopic technique to address this complex pathology.

Classification of shoulder instability

To adequately identify the subgroup at risk mentioned above, correct classification along the continuum of shoulder instabilities is essential. Multiple factors such as direction of dislocation, joint hyperlaxity, muscular dysbalances, bony defects, type and mechanism of event at onset of instability, number of dislocations as well as surgical history need to be considered in the classification of shoulder instability [46]. Consequently, there are various attempts in the literature to classify combinations of these features into the different types of instability, resulting in a complex system of overlapping, non-comprehensive classifications [23, 39, 49, 76].

Most of these classification systems do not allow for concise identification of the anteriorly unstable, "hyper lax subgroup" at risk, reflecting the clinical problem of correct identification and subsequent choice of therapeutic algorithm. For example, in the classification proposed by Matsen et al., the patients affected by anteroinferior instability and concomitant multidirectional hyperlaxity tend towards AMBRI-type (Atraumatic, Multidirectional, Bilateral, Rehabilitation, Inferior Capsular Shift) instability while also incorporating certain characteristics of the TUBS-type (Traumatic, Unilateral, Bankart defect, Surgery) instability [76]. In the Stanmore classification, the subgroup is located on the structurally defective axis tending towards Polar Group II [39]. Only Gerber and Nyffeler proposed a classification explicitly including hyperlaxity and direction of the instability, thereby allowing for the identification of the anteriorly unstable, multidirectionally hyperlax subgroup in their classification system [23].

Clinically, a strict separation of patients with anteroinferior instability and general hyperlaxity from traumatic anteroinferior instability without hyperlaxity is crucial in the selection of a subsequent treatment algorithm, but prone to errors. A seemingly adequate traumatic event at the onset of symptoms may lead to misclassification as a traumatic Polar Group I, TUBS type of instability and concomitantly to suboptimal treatment. Inversely, a clear differentiation from multidirectional instability (MDI) is also necessary. MDI is rare—accounting for only 2–10% of all cases of shoulder instability [46, 55]—and definitions vary significantly [31, 41, 79]. However, unidirectional instability with multidirectional hyperlaxity, while infrequently termed and classified as multidirectional instability in the literature [32], should be understood as a separate entity and distinguished from the term MDI, which should be used exclusively for patients with symptomatic instability in the an-
Comparison of typical findings in (arthro-)magnetic resonance imaging of an anteroinferiorly unstable shoulder with concomitant multidirectional hyperlaxity (a) and without hyperlaxity (b). Typical findings in patients with anteroinferior instability and concomitant multidirectional hyperlaxity include an anteroinferior Bankart lesion (white arrow, aI) and capsular redundancy with a wide inferior recess (white arrow, aII), compared to patients without hyperlaxity where bony defects, such as a prominent Hill–Sachs lesion (white arrow, bI & II), are typically found.

Diagnosis

To avoid misclassification, clinical assessment should focus on an evaluation of the appropriateness of the trauma, the existence of signs of hyperlaxity of the (contralateral) shoulder and should include a thorough radiographic assessment. More precisely, the patient should be questioned regarding the exact mechanism of dislocation, previous episodes of shoulder instability as well as potential recurrent shoulder dislocations. In the clinical assessment, besides positive “classical” anterior instability tests such as the anterior apprehension test, relocation release test and anterior load and shift tests, clinical hyperlaxity is indicated by supraphysiological translation in the anterior/posterior drawer test, positive Sulcus sign and Gagey test and possibly a Beighton score > 3 [20]. Posterior instability tests such as the posterior load and shift test or Jerk test are typically negative and should also be routinely performed [20]. In the presence of an appropriate traumatic mechanism at primary dislocation, which is usually associated with reduced tolerance to the clinical assessment of the ipsilateral side, specific attention needs to be paid to the clinical assessment of the contralateral side to detect clinical hyperlaxity.

The radiographic assessment consisting of standard radiographs in antero-posterior direction, axial view as well as y-view is commonly not associated with pathological findings in “hyperlax” patients. In contrast, magnetic resonance imaging (MRI) clearly identifies labral lesions such as an anterior Bankart lesion, often associated with a hypoplastic posterior labrum configuration [41] and, occasionally, concomitant asymptomatic posterior labral defects [30, 41]. The injection of contrast medium into the joint (arthro-MRI) further enhances soft tissue definition and facilitates superior visualization of the generally enlarged capsular volume indicative of shoulder joint hyperlaxity ([19, 32]; Fig. 1). While there is a low prevalence of concomitant bony defects in this collective, three-dimensional (3D) computed tomography can be employed in exceptional cases for quantification of posterior bone defects. As a result of the progressive increase in the prevalence of critical bone defects, radiological assessment of the shoulder joint is recommended in all cases of suspected critical bone defects.

Treatment

Patient-specific criteria warranting a non-operative strategy after primary shoulder dislocation include the absence of concomitant injuries, a patient age over 25 years and low-risk functional requirements [27, 54, 73]. In these cases, the established regimes include posttraumatic immobilization and early rehabilitation with increasingly permitted range of motion and actively assisted physiotherapy after 6 weeks [40, 69]. While an improvement of the insufficiency of passive shoulder stabilizers is not the primary aim, the focus of subsequent non-operative rehabilitation is the optimization of toning and proprioceptive capacities of the active glenohumeral stabilizers in (predominantly anteroinferior) humeral head centering and scapular control [11, 32, 80]. The physiotherapy regime is based on the principle of progressive resistance and relies on exercises targeting the deltoid muscles, rotator cuff muscles and scapulothoracic stabilizers employing elastic bands of progressively increasing stiffness within increasing degrees of elevation [11, 80]. After an initial phase of supervised therapy, lifelong self-guided maintenance exercises are recommended.

Operative treatment of anteroinferior instability with multidirectional hyperlaxity is generally warranted in patients
younger than 25 years [27, 40], in the presence of concomitant injuries and after failure of non-operative treatment indicated by recurrent symptoms of anteroinferior instability such as (sub-)luxations or subjective signs of persistent anteroinferior instability after more than 6 months. Typical patterns of a patient indicated for surgical treatment are illustrated in Fig. 2.

Arthroscopic treatment

Arthroscopic labral repair and capsular plication are the gold standard when glenoid bone loss is below a critical threshold of approximately 15%, with decision-making depending on the presence of an on-track Hill–Sachs lesion (HSL) and the functional requirements of the patient [1, 60, 71].

In general, arthroscopic shoulder stabilization can be performed in either the lateral decubitus position with the advantages of better visualization and instrument access, or the beach chair position with a quick, more anatomical setup and improved possibility for open conversion [16]. While results across multiple studies show comparable results, systematic reviews and a meta-regression analysis identify slightly lower recurrence rates (14.65% ± 8.4% vs. 8.5% ± 7.1%) and marginally higher patient satisfaction in patients treated in the lateral decubitus position compared to the beach chair position (93–100% vs. 85–87.5%) [16, 22].

To address the anteroinferior instability component of the pathology, a capsulolabral repair with the arthroscopic placement of a minimum of three suture anchors is necessary for sufficient biomechanical stability [45], anatomic restoration of anteroinferior stabilizers [17] and favourable clinical results [1, 10]. Conventional biocompatible anchors, as well as the recently introduced all-suture anchors in a knotted and knotless configuration can be employed for the capsulolabral repair, since no differences in biomechanical strength [36, 38] or clinical results are reported anywhere in the literature [6, 51, 84].

Since MR-arthrographic investigations revealed that possibly increased capsular volume rather than ligamentous laxity itself is the critical morphological feature of shoulder hyperlaxity, multiple studies advocate the role of capsular volume reduction as a key component in the treatment of shoulder instability [2, 7, 19, 19, 21]. Additionally, biomechanical models show efficient reduction of glenohumeral translation by performing a capsular shift and capsulolabroplasty [3, 7, 63, 70, 78], thereby restoring physiological capsular volume [21].

In the anteroinferior quadrant, capsular volume reduction is achieved by a capsulolabroplasty, which has proved biomechanical effectiveness [3, 70, 78] and produces favourable clinical results [7, 21]. Typically, a minimum of three anchors are used at the 5:30, 4:00 and 3:00 o'clock position (right shoulder) to achieve maximum stability. Postopera-
In recent decades, numerous different techniques for posterosuperior capsular volume reduction have been proposed [21, 52, 64]. Of these, capsular plication using simple sutures without any form of glenoid fixation has traditionally achieved favourable clinical results [21, 41]. However, while capsular plication to an intact labrum via sutures delivers similar strength of the construct compared to suture anchors, a suture-only technique is shown to cause a higher degree of labral displacement [67]. More specifically, dissociation of up to 1.5 mm has to be expected when not using glenoid anchors [67]. As the posterior capsule is known to be of a thinner, biomechanically less robust configuration [52], the literature advocates a low threshold in the decision for the use of suture anchors in posterosuperior capsulolabral surgery [52, 64]. For fixation, a simple stitch configuration is shown to be biomechanically sufficient [52]. Upon completion, a reduction of the capsular volume by up to 57% can be expected after combined posterosuperior and anteroinferior capsular plication [42, 82]. Furthermore, restoration of the physiologic tension of the posterior band of the inferior gleno-humeral ligament (PIGHL), inserting at the 7:30–8:50 position (right shoulder), can be achieved in this procedure [17]. Directing the plication stitch from inferior to superior has proved to be biomechanically superior to a medial-to-lateral stitch direction, preserving the range of rotation while achieving equal reduction of glenohumeral translation [3]. Finally, in the rare presence of a critical off-track HSL, posterosuperior plication as an adjunct to a Bankart repair delivers
similar resistance to anterior translation and Hill–Sachs engagement compared to a remplissage procedure while preserving the range of external rotation [81].

The management of concomitant injuries to the long head of the biceps tendon (LHBT) in the context of anteroinferior instability in the multidirectionally hyperlax shoulder is challenging. Biomechanical models have shown the LHBT to be a passive stabilizer of the glenohumeral joint by restricting omnidirectional glenohumeral translation by concavity compression [4, 29, 56], resisting torsional forces in the vulnerable ABER (abduction, external rotation) position [24, 68] and diminishing stress on the IGHL [68]. Furthermore, injuries to the superior labral anterior to posterior (SLAP) complex—especially the posterior part including the biceps anchor—have been demonstrated to biomechanically result in increased glenohumeral anterior and posterior translation [57, 58], which cannot be fully restored by biceps tenodesis [74]. While studies presenting data from biplane fluoroscopy investigations, which do not fully confirm these findings in vivo [24], do exist, the role for the LHBT as a passive glenohumeral stabilizer is still firmly postulated in the current literature. Thus, to preserve the LHBT as an additional stabilizer in the clinically hyperlax, anteroinferiorly unstable patient, a reconstructive procedure is recommended for concomitant injuries to the SLAP complex or LHBT when feasible. SLAP repair was demonstrated to provide convincing postoperative improvement of clinical scoring [65] and a return to sports [33] with an acceptable failure rate [65] in patients <35 years, the typical age of the patients presenting with anteroinferior instability with multidirectional hyperlaxity. In selected cases, where surgery is indicated in patients with anteroinferior instability and hyperlaxity >35 years with preexisting degenerative lesions of the biceps anchor, a biceps tenodesis may be considered due to the superior clinical results, a return to activity [18] and lower revision rates [15] in this age group. A combined approach addressing injury to the LHB with biceps tenodesis while attempting to preserve the biomechanical advantages
of an intact SLAP complex with a SLAP repair is not advised, as this results in significantly worse clinical outcome compared to isolated procedures [12].

While certain studies recommend rotator interval closure as an adjunct procedure when addressing clinical instability with arthroscopic Bankart repair and posteroinferior plication [13], there is no consensus on the indication of this procedure [14, 66]. Due to lack of consensus on the definition of the procedure, reports in the literature remain inhomogenous [14]. While biomechanically the closure of the rotator interval reduces overall glenohumeral translation—most markedly in the inferior direction [61, 83]—reports on the subsequent effect on glenohumeral stability remain ambiguous [26, 43, 48]. Moreover, rotator interval closure results in a significant loss of range of motion [14, 48]. While the literature suggests critically evaluating rotator interval closure in cases of multidirectional instability or shoulder instability with extensive inferior laxity [14, 66], there is insufficient evidence to recommend this procedure as a standard treatment for anteroinferior instability with multidirectional hyperlaxity.

In daily practice, the authors perform arthroscopic anteroinferior capsulolabroplasty and posteroinferior plication (Fig. 3) for anterior shoulder instability with multidirectional laxity. Preoperatively, anteroinferior instability and multidirectional hyperlaxity are confirmed by clinical examination under general anesthesia and the patient is placed in lateral decubitus position. After establishing a standard posterior viewing portal and the subsequent diagnostic visualization of the joint (Fig. 4a), an anterosuperior viewing and an anteroinferior working portal is created. Subsequently, the capsulolabral complex is carefully mobilized (Fig. 4b–d) and a bleeding bed is induced along the glenoid rim to enhance healing capacity (Fig. 4e). For posteroinferior capsular plication, a deep posterolateral portal

Fig. 4a Intraoperative arthroscopic images of a right shoulder in the lateral decubitus position. a Hypoplastic posterior labrum; b mobilisation of the anterior labrum; c creation of a bleeding bed; d placement of a posteroinferior anchor (7:00); e–f posteroinferior plication; g placement of an anteroinferior anchor (5:30); h placement of an anterior anchor (4:00); i completed anteroinferior stabilization
Intraoperative image of cannula setting with a standard posterior viewing portal, an antero-superior viewing and an antero-inferior working portal equipped with Twist-In™ cannulas (Arthex, Naples, FL, USA) and a deep posterolateral portal equipped with a Gemini cannula (Arthex, Naples, FL, USA), before the establishment of the 5:30 portal (Fig. 5).

The first suture anchor (FiberTak®-Soft 1.6mm all-suture anchor; Arthex, Naples, FL, USA) is introduced posteroinferiorly at the 7 o’clock position (right shoulder) at the chrondrolabral junction, and the anchor’s suture is passed through the capsular tissue 10–15 mm posteroinferiorly of the labrum to shift the PIGHL complex superomedially and reduce the capsular volume of the posteroinferior axillary pouch and posterior joint space. The knot is tightened while correct position and tension of the capsulolabral complex are ensured with a grasper superiorly of the knot.

Using the anteroinferior working portal at the 5:30 position [28], the first anteroinferior suture anchor is placed in the 5:30 position (right shoulder) at the chondrolabral junction (Fig. 4j). The AIGHL complex as well as the anteroinferior axillary pouch are purchased equivalently to the posteroinferior capsulolabraloplasty. In the same way, two further anchors are introduced at the 4:30 and 3:00 o’clock positions and a capsulolabral “bumper” is created at the anteroinferior circumference of the glenoid (Fig. 4j–I).

After tying the knots, anterior, posterior and inferior translation are carefully tested. If reduction of posterior translation is found to still be insufficient, a second posteroinferior anchor can be inserted at the 8 o’clock position in the same manner as the 7 o’clock anchor.

### Postoperative management

The postoperative management regime is equivalent to an anteroinferior Bankart repair. A sling orthosis is required to maintain immobilization and preservation of the repair and should be worn during the night and walking for 6 weeks postoperatively. For the first 3 weeks following surgery, range of motion in abduction is gradually increased from 45° to 90°, while external rotation is limited to 0°. After 6 weeks, a regime with actively assisted range of motion exercises is initiated. Strengthening exercises are initiated 3 months after surgery and no contact sports should be started before 6 months postoperatively.

### Pearls and pitfalls

- **For the posteroinferior portal, a cannula with a deployable wing feature should be employed to prevent dislocation of the cannula and enable enlargement of the posteroinferior working space by applying traction to the cannula.**
- **When establishing the 5:30 portal, a blunt preparation technique should be employed to spare neurovascular structures. A switching stick can be advanced to the humeral shaft and directed cranially to penetrate the capsule directly superior to the AIGHL complex and subsequently used for the placement of a cannula.**
- **When performing anteroinferior capsulolabroplasty via an anteroinferior portal only (not employing a deep anteroinferior [5:30] portal), the use of all-suture anchors provides the benefit of using curved guides to achieve optimal positioning of the anteroinferior (5:30) anchor.**
- **Knots should be tied in the capsular tissue away from the chondrolabral junction to avoid chondral damage to the glenoid or humeral head during glenohumeral motion.**
- **Anchors should be inserted at an angle of 135° to the glenoid surface to avoid chondral damage or anchor dislocation.**
- **During capsular plication, an inferior-to-superior stitching direction should be selected to sufficiently reduce inferior capsular redundancy.**

### Clinical take home message

- **Patients with multidirectional hyperlaxity represent a subgroup at risk of failure in arthroscopic Bankart repair for anteroinferior shoulder instability.**
- **With regard to classification and terminology, a clear differentiation to multidirectional instability and isolated anteroinferior instability must be ensured.**
The key factor for diagnosis is the physical examination of the affected shoulder with positive tests for unidirectional anteroinferior instability and concomitant signs of shoulder hyperlaxity.

Anteroinferior capsulolabralplasty and posteroinferior plication to reduce capsular redundancy restore stability while preserving physiological glenohumeral biomechanics.

A minimum of four suture anchors in a simple stitch configuration should be used at the 3–7 o’clock position (right shoulder) to generate a sufficient bumper and to reduce capsular volume effectively.

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