Current Reviews in Musculoskeletal Medicine (2023) 16:123–144
https://doi.org/10.1007/s12178-023-09822-6

SHOULDER INSTABILITY (J DINES AND C CAMP, SECTION EDITORS)

Management of Shoulder Instability in Patients with Underlying Hyperlaxity

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Accepted: 27 January 2023 / Published online: 23 February 2023

Abstract

Purpose of Review Shoulder instability in patients with underlying joint hyperlaxity can be challenging to treat. Poorly defined terminology, heterogeneous treatments, and sparse reports on clinical outcomes impair the development of best practices in this patient population. This article provides a review of the current literature regarding optimal management of patients suffering from shoulder instability with concomitant hyperlaxity of the shoulder, from isolated shoulder joint hyperlaxity to congenital hypermobility spectrum disorders (HSD).

Recent Findings Current research shows specialized physiotherapy protocols focused on strengthening of periscapular muscles and improvement of sensorimotor control are a promising non-surgical therapeutic avenue in certain patients, which can be augmented by device-based intervention in select cases. If surgical treatment is warranted, arthroscopic techniques such as pancapsular shift or plication continue to demonstrate favorable outcomes and are currently considered the benchmark for success. The long-term success of more recent innovations such as coracoid process transfers, conjoint tendon transfers, subscapularis tendon augmentation, and capsular reconstruction remains unproven. For patients affected by connective tissue disorders, treatment success is generally less predictable, and the entire array of non-operative and operative interventions needs to be considered to achieve the best patient-specific treatment results.

Summary In the treatment of shoulder instability and concomitant hyperlaxity, specialized physiotherapy protocols augmented by device-based interventions have emerged as powerful, non-operative treatment options for select patients. Successful surgical approaches have been demonstrated to comprehensively address capsular redundancy, labral lesions, and incompetence of additional passive stabilizers in a patient-specific fashion, respective of the underlying connective tissue constitution.

Keywords Shoulder instability · Hyperlaxity · Hypermobility · Ehlers Danlos · Shoulder stabilization

Introduction

Shoulder instability is a frequent reason for shoulder-related disability in the adolescent and adult patient population [1, 2]. While there exists an extensive body of evidence on the surgical management of isolated unidirectional instability, optimal treatment in certain patient subgroups, such as those with underlying hyperlaxity, remains the subject of ongoing controversy. In particular, the optimal management of patients with concomitant shoulder joint hyperlaxity, an independent risk factor for the recurrence of shoulder instability [3, 4], remains controversial. Hyperlaxity is prevalent in approximately 5–15% of the shoulder instability cases [5] and has been shown to be predictive of failure for soft tissue repair both in primary [6–9] and revision situations [10, 11]. As such, it is an established component of risk scoring systems, such as the instability severity index score, that quantify the risk of recurrence of shoulder instability [7].

This article is part of the Topical Collection on *Shoulder Instability*

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Shoulder hyperlaxity exists in different grades of severity and manifestations. It can be found in isolation, localized to the shoulder joint, or in the context of generalized ligamentous hyperlaxity, as in patients with symptomatic hypermobility spectrum disorders (HSD) or related disorders such as hypermobile (Type III) Ehlers-Danlos-Syndrome (hEDS).

The primary objective of this paper is to review the current literature regarding optimal management of patients suffering from shoulder instability with concomitant hyperlaxity of the shoulder, presented along the continuum from isolated shoulder joint hyperlaxity to hypermobility disorders. As terminology, treatment algorithms, and clinical outcomes reported in the literature are relatively heterogenous, a concise understanding of best practices in the diagnosis and treatment of concomitant hyperlaxity in the setting of shoulder instability may assist clinicians in the management of this challenging combination of conditions.

Definition

Shoulder hyperlaxity is defined as an increase in joint translation due to greater-than-normal laxity of the capsule and/or increased capsular volume [12, 13, 14•]. This constitution typically allows for increased glenohumeral translation, distractability of the joint, and increased active range of motion (ROM)[15]. Laxity is a measure of joint translation and hyperlaxity is a supraphysiologic amount of joint translation that is typically measured somewhat subjectively. The most common manifestations are excessive joint mobility and hyperabduction. In hyperlaxity without instability, clinical stability in the glenoid is maintained [16••], and neither subjective instability (apprehension) nor objective instability (symptomatic subluxation or dislocation) is encountered. In its non-pathologic expression, shoulder hyperlaxity may be advantageous, as evident by the disproportionately high prevalence in overhead athletes, who may achieve superior athletic performance as a result of their increased ROM [12]. However, hyperlaxity may progress to pathological instability of the joint when the functional balance of active and passive stabilizers is disrupted. The resulting combination of hyperlaxity and instability has been shown to be particularly challenging to treat [12].

Etiology of Hyperlaxity

Approximately 10% of the population is affected by constitutional shoulder hyperlaxity, with females being affected more commonly [17]. This constitutional hyperlaxity can be classified as either localized hyperlaxity, which is limited to the shoulder joint, or generalized hyperlaxity, affecting multiple joints [18]. Shoulder hyperlaxity can be congenital or acquired [19, 20•] as the result of repetitive overload, stretching, or microtrauma of the passive stabilizers of the shoulder, as is typically found in overhead athletes [21].

Congenital hyperlaxity also encompasses a variety of connective tissue disorders, including HSDs, hEDS, Marfan's syndrome, and other disorders, that result in shoulder instability due to an incompetency of passive stabilizers. For the purpose of this paper, we will summarize HSDs and hEDS together, as commonly recommended, as these conditions are similar in terms clinical symptoms, etiology, and treatment strategies. Other etiologies such as Marfan's syndrome, Loeys-Dietz syndrome, and osteogenesis imperfecta will not be covered in this paper, as these have distinct clinical manifestations, and the overall incidence of shoulder instability related to these conditions is relatively scarce.

Symptomatic, disabling HSD/hEDS has a prevalence of approximately 1 in 500 individuals [22]. Joint dislocations or subluxations occur in > 95% of patients with hEDS [23••, 24], with the shoulder being among the most commonly affected [24]. The conditions are believed to have a genetic basis, as they are typically inherited in an autosomal dominant manner with variable expression and incomplete penetrance [25]. In contrast to other diseases of connective tissue that have a known abnormality in the genes encoding collagen or fibrillin proteins, a pathognomonic structural abnormality of connective tissue has not yet definitively been identified in HSD/hEDS. Further complicating the condition, the phenotype of HSD/hEDS includes symptoms unrelated to the musculoskeletal system, such as autonomic nervous system dysfunction, gastrointestinal dysmotility, and anxiety disorders [18]. Given the structural abnormality of the connective tissue, shoulder instability as a part of the symptom complex can be notoriously challenging.

Localization Along the Continuum of Shoulder Instability

Once shoulder instability that requires treatment occurs in the setting of underlying shoulder hyperlaxity, it is essential to localize the combination of instability and hyperlaxity along the continuum of shoulder instabilities to select the optimal treatment strategy. This may, however, be challenging in a complex system of overlapping, non-comprehensive classifications of shoulder instability [26–29], as most of these classification systems do not allow for unambiguous identification of the hyperlax subgroup of patients. Within the Stanmore classification, patients with shoulder instability and concomitant hypermobility tend to belong to Polar Group II, characterized by an atraumatic onset based on an underlying structural defect [28]. In the classification system proposed by Matsen et al., these patients most commonly can be classified as AMBRI-type (Atraumatic, Multidirectional, Bilateral, Rehabilitation, Inferior Capsular Shift) instability [27]. The classification system that best allows for identification of patients with concomitant laxity was first introduced by Gerber and Nyffeler, which explicitly includes

hyperlaxity as the decisive criterion for subsequent classification [26]. In this classification system, special attention is turned toward the adequacy of the trauma leading to the onset of instability.

Furthermore, while these two entities often overlap, a concise differentiation of unidirectional instability with concomitant shoulder hyperlaxity and the rarer entity of multidirectional instability (MDI) of the shoulder (2–10% of the cases) is required to guide decision making [30, 31]. MDI was defined by Neer as symptomatic instability in two or more directions, one of which is inferior [32], and requires a distinct therapeutical management [33, 34].

Lastly, it should be determined whether a certain component of instability in the hyperlax patient is "functional." "Functional instability" is a recently proposed distinct subtype of shoulder instability, characterized by a pathologic activation pattern of the periscapular muscles, and the percentage of patients with concomitant hyperlaxity is especially high in this patient population [29, 35]. Functional instability is most similar to Polar type III in the Stanmore classification and can be further stratified into (non-)positional and (un-)controllable subtypes [29]. As such, patients affected by functional instability closely resemble a subpopulation of patients previously termed as "voluntary dislocators" [29].

Diagnosis

In the patient's history—besides typical shoulder instability symptoms reported by a patient with shoulder instability—an anamnesis of prior subluxation and a family history of hyperlaxity may indicate concomitant hyperlaxity [12]. As hyperlaxity may lower the threshold for a joint dislocation, a special focus should be turned towards evaluating adequacy of the index-trauma leading to instability [36]. Also, pain or numbness and tingling while carrying heavy loads that exert inferior traction on the arm may be associated with inferior hyperlaxity in a more severe form [12]. Furthermore, symptoms indicative of the severity of instability, such as dislocation during sleep, or any voluntary component in controlling the symptoms of instability should be assessed.

Complementing the standard shoulder instability physical examination [37•], existing signs of hyperlaxity of the shoulder include a large Sulcus sign in neutral rotation that does not decrease with external rotation (indicative of a concomitant laxity of the rotator interval) [38]; a positive Gagey hyperabduction test expressed by a glenohumeral abduction > 105° [39]; increased translation in the anterior and posterior drawer test [39] and an external rotation of > 90° in a supine position [40]. Oftentimes, a Jerk test is also positive with posterior loading and the clunk of reduction with horizontal abduction. While the patient may not tolerate hyperlaxity assessment of the ipsilateral shoulder in the acute posttraumatic situation, an examination of the contralateral shoulder may reveal hyperlaxity. Especially in the anterior and posterior drawer test, special attention should be turned towards whether the test elicits apprehension, which is typical of instability, versus greater degrees of laxity without any sign of subjective instability.

Radiographic work-up includes standard x-rays and magnetic resonance images, which typically do not show evidence of major structural defects such as glenoid bone loss or pathologic version in the hyperlax patient population [41]. However, these structural defects require treatment if encountered [42–45]. Typical findings in the setting of shoulder joint hyperlaxity may include evidence of hypoplastic labrum configurations [46], a wide Hill Sachs defect [20•], and an enlarged capsular volume [12, 13]. In contradistinction to conventional teaching, a recent study by Raynor et al. showed that labral tears are encountered not infrequently in the setting of MDI [33]. Special tear configurations requiring distinct management such as a humeral avulsion of the glenohumeral ligament (HAGL), glenolabral articular disruption (GLAD) or anterior labroligamentous periosteal sleeve avulsion (ALPSA) lesions [9, 47–50]. In the HSD/hEDS population, inferior subluxation of the humeral head as well as glenoid dysplasia maybe be appreciated.

Clinical manifestations such as recurrent musculoskeletal injury, hyperlaxity symptoms in one or more joints, and poor proprioception may increase the suspicion for HSD/hEDS [51, 52]. Depending on patient age, the presenting symptoms can be classified into 3 distinct phases of hEDS: hypermobility without marked pain in adolescence, the onset of pain in the second to fourth decade, and stiffness occurring in late adulthood [23••]. Patients with suspected HSD/hEDS should be examined for additional systemic features including chronic pain, autonomic dysfunction, gastrointestinal dysmotility, fatigue, psychiatric symptoms such as anxiety or depression, skin fragility, and easy bruising [24]. The diagnostic criteria that were most recently updated in 2017 are listed in Table 1 [53]. Criterion 1 involves the use of the Beighton score [54] to detect generalized joint hypermobility. The Beighton scoring system consists of 5 physical exam tests of joint hypermobility, 4 of which are repeated bilaterally (Table 2). Beighton score values ≥ 5 [55] typically indicate generalized laxity, although patients with lower scores may meet the criteria if they are positive on a 5-part questionnaire (5-PQ) [56]. If all 3 criteria are fulfilled, the individual receives a diagnosis of hEDS (Table 3). Patients who do not meet all 3 criteria but still have symptoms of generalized joint hypermobility receive the diagnosis of HSD (Table 3). While typically a clinical diagnosis, genetic testing may be used to identify hEDS or other connective tissue diseases.

Table 1 Criteria for hypermobile Ehlers-Danlos Syndrome [53, 57]

Criteria	Characteristics
First—Generalized Joint Hypermobility	• Beighton score ≥ 6 for children and adolescents • Beighton score ≥ 5 for adults up to age 50 • Beighton score ≥ 4 for adults > 50 years old with hEDS • If Beighton score is ≤ 4 and person is positive for ≥ 2 of the 5-part questionnaire, they receive 1 Beighton score point
Second	**Feature A – Connective Tissue; must have ≥ 5 characteristics** **Fascia Attributes** • Soft or velvet-like skin • Hyperextensible skin • Irregular striae • Bilateral piezogenic heel papules • Multiple abdominal hernias • Atrophic scarring involving ≥ 2 sites and without formation of papyraceous or hemosiderotic scars • Pelvic floor or rectal prolapse in children, men, or nulliparous women without a historical medical condition **Marfanoid Attributes** • Dental crowding and high-arched palates • Mild or severe Mitral valve prolapse (MVP) based on echocardiographic criteria • Arm-to-height span ≥ 1.05 • Arachnodactyly, defined as: (1) positive wrist sign on both sides; (2) positive thumb sign on both sides • Aortic root dilatation with Z-score > + 2 **Feature B – Family History** • More than 1 first-degree relative who has hEDS **Feature C – Musculoskeletal; must have at least 1** • Musculoskeletal pain in ≥ 2 more limbs; must occur daily for at least 3 months • Widespread pain for ≥ 3 months • Recurring joint dislocations or instability, without trauma • ≥ 3 atraumatic dislocations in the same joint or ≥ 2 atraumatic dislocations in 2 different joints occurring at different times • Joint instability at ≥ 2 sites, without a history of trauma a the joints
Third	**All the following must occur** • Absence of pathological skin fragility • Exclusion of heritable and acquired connective tissue disorders, including autoimmune rheumatologic conditions • In patients with an acquired connective tissue disorder (Ex: systemic lupus erythematosus or rheumatoid arthritis) diagnosis of hEDS requires meeting features A and B of the second criteria; Feature C of criterion 2 cannot be counted towards an hEDS diagnosis • Exclusion of diagnoses that include joint hypermobility by way of hypotonia or connective tissue laxity. Alternative diagnoses include neuromuscular and hereditary disorders of connective tissue

**In order for a patient to be diagnosed with hEDS, they must meet classification of the first Criteria and possess at least 2 characteristics of any of the features in the second Criteria. Additionally, they must hold characteristics from the third Criteria that result from hEDS

Table 2 Beighton Score Criteria [57]

Movement	Scoring
Passively dorsiflex 5th metacarpophalangeal joint by 90 degrees or more	1 Point (left side) 1 Point (right side)
Oppose the thumb to the volar aspect of the ipsilateral forearm	1 Point (left side) 1 Point (right side)
Hyperextend the elbow by 10 degrees or more	1 Point (left side) 1 Point (right side)
Hyperextend the knee by 10 degrees or more	1 Point (left side) 1 Point (right side)
Place hands flat on the floor without bending knees	1 Point

*4 or more points is a sign of generalized joint hypermobility

Table 3 Comparison between characteristics of hypermobility spectrum disorder and hypermobile Ehlers-Danlos Syndrome [57]

HSD (Hypermobility Spectrum Disorder)	hEDS (Hypermobile Ehlers-Danlos Syndrome)
Generalized, peripheral, localized, or previous joint hypermobility	Generalized joint hypermobility
Musculoskeletal structural challenges	Musculoskeletal structural challenges
Joint instability	Joint instability
Soft-tissue pathology	Soft tissue pathology
Musculoskeletal pain	Musculoskeletal pain Skin pathology Tissue weakness Marfanoid body type Cardiac valve challenges
Family history of HSD is common	First degree relative with hEDS is common

Treatment Concepts

Whereas contemporary treatment concepts in non-hyperlax patients are proactive in surgically restoring shoulder instability, especially in patients younger than 25 years with a high functional demand [3, 58, 59], treatment regimens tend to be more conservative with increasing shoulder hyperlaxity, especially in the presence of HSD/hEDS.

Non-surgical Treatment in the Absence of HSD/hEDS

Conservative treatment options in the setting of shoulder instability with concomitant hyperlaxity are typically indicated after a first-time shoulder dislocation. Factors favoring the indication of a conservative regimen include the absence of major structural lesions such as labral tears or glenoid/humeral bone loss, patient age > 25 years, increased severity of hyperlaxity, and a functional component of instability [60, 61].

Contemporary non-operative treatment regimens commonly start with post-traumatic immobilization and emphasize an early rehabilitation protocol focusing on increasing ROM three weeks after the trauma [62, 63]. As repairing the passive shoulder stabilizers is not the primary aim, the focus is instead optimizing the toning and proprioceptive capacities of the active glenohumeral stabilizers centering the humeral head and controlling scapular movement [12, 64, 65]. Historically, successful physiotherapy protocols have been largely based on the concept of progressive resistance, focusing on strengthening the periscapular and scapulothoracic musculature using increasingly stiff elastic bands with increasing ROM [64, 65]. More recent rehabilitation protocols such as the "Derby Shoulder Instability Program" emphasize the role of the sensorimotor system and thus involve plyometric training, kinetic chain exercises, and closed chain weight-bearing exercises on uneven surfaces, with the goal of restoring shoulder proprioception and joint stability [66]. To sustain clinical success (Table 4), continued individual daily exercises are warranted following a phase of supervised physiotherapy.

More recently, innovative conservative treatment regimens can include the implementation of a "shoulder pacemaker" device. This may be a particularly suitable option for patients who have failed prior physiotherapy and for patients affected by a functional component of shoulder instability, in which hyperlaxity is highly prevalent [70•]. The treatment regimen consists of multiple hour-based treatment sessions of electrical muscle stimulation (EMS) therapy, utilizing EMS devices to transmit alternating current to a patient's scapula-retracting muscles and external rotators during active exercises [70•, 71]. The length of treatment depends on patient prognosis and the length of persisting symptoms after surgery. Given a correct indication, favorable clinical outcomes with significant improvement in clinical outcomes scores have been reported (Table 5).

Surgical Treatment in the Absence of HSD/hEDS

Surgical management is typically recommended in the presence of concomitant injuries or following failure of conservative treatment, characterized by recurrent instability, subluxations, or a subjective feeling of instability > 6 months. Also, a patient age < 25 years at first time dislocation may influence the decision to proceed with surgery, as high recurrence rates of instability after conservative treatment strategies have been reported in this population [58, 63].

Surgical principles in this patient collective include restoration of labral integrity, reduction of the increased capsular volume, and possibly restoration of additional incompetent passive stabilizers [13, 73, 90, 91••]. Regardless of the specific surgical technique, the capsular volumetric reduction

Table 4 Clinical outcomes of non-surgical treatment for hyperlaxity and shoulder instability patients in the absence of a connective tissue disorder ($N=5$)

Study	Hyperlax Population[1]	Definition of Hyperlaxity	Treatment	Clinical Outcomes
Tillander et al. (1998) [67]	Patients with instability and multidirectional hyperlaxity (n = 20/35)[2]	Positive sulcus sign, negative apprehension tests, & graded on a scale from 0 to 3, according to Marshall et al[6]	Rehabilitation program targeting rotator cuff muscle strength training and scapular stabilization	- Mean follow up: 28 months (9–47) - Subjective satisfaction: 9/20 (45%) satisfied - Median Constant score: 82 (range 68–98)
Bateman et al. (2015) [66]	Patients with recurrent atraumatic shoulder instability (n = 18/18)	Beighton score ≥ 5; Instability Polar Type 2 and 3 in the Stanmore Classification	The Derby Shoulder Instability Rehabilitation Programme	- Mean follow-up: 4.5 months (1.4–11.8) - Mean OISS: decrease from 37.06 to 20.39 ($p<0.001$) - Mean WOSI: increase from 47.47% to 84.23% ($p<0.001$) - Mean WOSI 'Physical' domain: increase from 52.27% to 85.61% ($p<0.001$) - Median WOSI 'Sport and Work' domain: increase from 35.13% to 88.75% ($p<0.001$) - Median WOSI 'Lifestyle' domain: increase from 50.50% to 87.63% ($p<0.001$) - Median WOSI 'Emotions' domain: increase from 30.83% to 91.00% ($p<0.001$)
Watson et al. (2018) [65]	Patients with multidirectional instability of the shoulder (n = 22/43)[3]	Positive sulcus sign; Beighton score > 4	Watson MDI 12-week exercise program with a focus on stability and muscle control	- Mean follow up: 4.6 months (3.6) - Mean WOSI: decrease from 1264.63 to 482.23 ($p<0.001$) - Mean MISS: increase from 46.95 to 76.32 ($p<0.001$) - Mean OSIS: decrease from 35.76 to 20.67 ($p<0.001$) - Mean NRS: decrease from 4 to 1.6 ($p<0.001$)
Bateman et al. (2019) [68]	Patients with Polar Type 2 or 3 instability (n = 51/66)[4]	Clinical signs of shoulder laxity in at least one direction	The Derby Shoulder Instability Rehabilitation Programme	- Mean follow up: 30 weeks (6–51) - Mean OISS: decrease from 38.00 to 21.96 ($p<0.001$) - Mean WOSI: increase from 45.10% to 85.81% ($p<0.001$) - Mean WOSI 'Physical' domain: increase from 47.98% to 81.19% ($p<0.001$) - Mean WOSI 'Sport & Work' domain: increase from 40.17% to 82.00% ($p<0.001$) - Mean WOSI 'Lifestyle' domain: increase from 50.73% to 83.45% ($p<0.001$) - Mean WOSI 'Emotions' domain: increase from 32.84% to 79.78% ($p<0.001$)

Table 4 (continued)

Study	Hyperlax Population[1]	Definition of Hyperlaxity	Treatment	Clinical Outcomes
Moroder et al. (2020)[69]	Patients with posterior positional shoulder instability who failed conventional therapy (n = 7/14)[5]	Beighton score ≥ 5, as well as a positive sulcus sign, Gagey test, and Walch test	Electric muscle stimulation by a shoulder-pacemaker	- Follow-up: 2 weeks, 4 weeks, 3 months, 6 months, 12 months, and 24 months - Median Subjective Shoulder Value: increase from 50 to 88% - Median Rowe score: increase from 40 to 94% - Median WOSI score: increase from 38 to 70% ($p < 0.001$) - Young age ($p = 0.005$), low weight ($p = 0.019$), shoulder activity level ($p = 0.003$), and baseline WOSI score ($p = 0.04$) were associated with higher outcomes

Abbreviations: *WOSI*, Western Ontario Shoulder Instability Index; *OSIS*, Oxford Shoulder Instability Score; *MISS*; Melbourne Instability Shoulder Score; NRS, numerical rating scale

[1]Number of hyperlax patients out of total patient population

[2]A total of 35 patients were present in this study; only 20 received non-operative management alone

[3]43 total patients were included in the study; 22 were classified with hyperlaxity

[4]66 patients were included in the study, but 15 were lost to follow-up

[5]7 patients were classified as hyperlax, with 14 total patients in the study

[6]Marshall JL, Johanson N, Wickiewicz TL, et al. Joint looseness: a function of the person and the joint. Med Sci Sports Exerc 1980: 12: 189–94

and thus the degree of stabilization needs to be tailored to the patient's individual demand, as the ROM demands of certain patient cohorts of (overhead-)athletes may deviate from the general population [12].

Inferior Capsular Shift

The open inferior capsular shift procedure was first introduced in 1980 by Neer et al. [92] to address the patulous inferior capsular pouch typical of hyperlaxity patients. This procedure relies on a modified T-shaped capsulotomy through an anterior approach, which is performed horizontally between the inferior (IGHL) and middle glenohumeral ligaments (MGHL) in anterior shoulder instability [93]. Since it was first described, this procedure has been modified through a posterior approach, and an arthroscopic variation of this technique has been described [12]. As the shoulder's external rotation ROM depends on the anteroinferior capsular volume, it is recommended to maintain the arm in lower degrees of abduction and external rotation during tensioning of the capsule to preserve adequate postoperative ROM [12, 93]. In the case of persistent intraoperative instability, this procedure can be combined with a rotator interval closure [12]. Given the correct indication and technique, the inferior capsular shift technique has historically reported favorable post-surgical outcomes with low revision rates (Table 5) [67, 72–75, 81, 82].

Capsular Plication/Capsulorraphy

Harnessing the advantages of arthroscopic visualization, targeted patient-specific capsular volume reduction, and decreased surgical morbidity, the capsular plication or capsulorraphy techniques has more recently been proposed for this patient collective. Biomechanical studies show that a unidirectional anteroinferior capsular shift may lead to increased posterior translation and subluxation [94, 95], which may ultimately lead to degenerative joint wear [95–97]. Even while the effect on translation is limited [98], biomechanical investigations suggest that a more anatomic, combined anterior and posterior capsular volume reduction by plication is required for optimal restoration of physiologic joint biomechanics, irrespective of the direction of instability [99, 100]. Upon completion of the capsulorraphy, a reduction of the capsular volume up to 57% [101, 102] and restoration of the physiologic tension of the posteroinferior glenohumeral ligament (PIGHL) can be achieved in this procedure [103•].

Different technical variations of the capsular plication or capsulorraphy procedures have been described, which

Table 5 Clinical outcomes of surgical treatment for hyperlaxity and shoulder instability patients in the absence of a connective tissue disorder ($N=5$)

Study	Hyperlax Population[1]	Definition of Hyperlaxity	Treatment	Clinical Outcomes
Lebar et al. (1992) [72]	Patients with multidirectional instability (n = 3/10)	"Demonstration of hypermobility in other joints such as wrists, elbows, and knees"	Open inferior capsular shift procedure	- Mean follow up: 28 months - Mean ASES pain score: 2.75 pre-surgery and 4 post-surgery - Mean internal rotation: 5° pre-surgery and 8° post-surgery - Mean AAS score: 2.7 pre-surgery and 3.3 post-surgery
Bigliani et al. (1994) [73]	Athletes with anterior inferior shoulder instability (n = 59/63)	"Demonstratable inferior sulcus sign"	Open inferior capsular shift procedure	- Mean follow up: 4 years (1–9) - Mean external rotation loss: 7° (0°-30°) - Mean forward elevation loss: 175° (165°-180°) - Subjective outcomes: 67% "excellent," 27% "good," 3% "fair," and 3% poor - 97% of patients experienced pain relief after surgery - 58 (92%) patients returned to sport - 47 (75%) patients competed at pre-surgery level - Redislocation rate: 2 (2.9%)
Tillander et al. (1998) [67]	Subgroup of patients with multidirectional hyperlaxity (n = 14/35)[1]	"Assessed according to Marshall et al."[4]; graded on a scale from 0 to 3	Initial non-operative physiotherapy and open inferior capsular shift surgery	- Mean follow up: 37 months (9–53) - Median Rowe score for hyperlax subgroup: 65 (20–95) - Median Constant score for hyperlax subgroup: 69 (50–79) - Patient satisfaction: 12 (85%) patients of MDH and instability subgroup satisfied - Subgroup with MDH and instability: 45% success rate
Bak et al. (2000) [74]	Patients with multidirectional glenohumeral instability (n = 25/25)	Positive drawer test and sulcus sign	Open inferior capsular shift procedure	- Mean follow-up: 54 months (25–113) - Median Rowe Score: 92.5 (20–100) - Median UCLA Score: 33 (23–35) - Subjective outcomes: 24 shoulders (92%) were "excellent" (9) or "good" (15) (UCLA) - 23 (88%) shoulders were "excellent" (n = 14) or "good" (n = 9) - 84% of patients returned to pre-surgery sport level 5 months post-surgery - 57% of patients continued to play their sport at follow-up time - Failure rate: 8% (defined as shoulder instability post-surgery)
Marquardt et al. (2005) [75]	Patients with atraumatic anterior-inferior shoulder instability who had failed non-operative management (n = 35/35)	"Anterior and posterior draw tests " as described by Altchek et al."[13]	Arthroscopic modified capsular shift procedure	- Mean follow up: 7.4 years (4.0–11.4) - Mean Rowe Score: preop: 36.2 (SD = 13.5); postop: 90.6 (SD = 19.7) - 81.6% of shoulders experienced no loss of range of motion - Subjective outcomes: 78.4% of patients were "excellent" post-surgery - 72% of patients returned to same level of sport - Failure rate (= redislocation): 10.5%
Boileau et al. (2007) [76]	Patients with anterior shoulder instability and associated capsular deficiency (n = 36/36)	External rotation (with arm at side) > 85 degrees	Arthroscopic Bristow procedure + Bankart repair	- Mean follow-up: 19 months (12–36) - Mean loss of external rotation: 9° (arm at side) post-surgery - Mean loss of external rotation (in abduction) post-surgery: 15° - Mean loss in internal rotation post-surgery: 0° - Mean Walch-Duplay score: 87 points - Subjective outcomes: 28 (78%) "very satisfied," 5 (14%) "satisfied," 3 (8%) "disappointed" - Return to sport: 30 (91.0%) - Failure rate: 3 (8%) (recurrent instability post-surgery)

Table 5 (continued)

Study	Hyperlax Population[1]	Definition of Hyperlaxity	Treatment	Clinical Outcomes
Baker et al. (2009) [77]	Patients with multidirectional shoulder instability (n = 40/40)	Presence of a grade 2 + sulcus sign, not changing with external rotation	Arthroscopic (pan)capsular shift with suture anchors, and rotator interval closure (n = 10)	- Mean follow-up: 33.5 months (24–65) - Mean ASES score: 91.4 (59.9–100) post-surgery - Mean WOSI score: 91.1 (72.9–100) - ROM: 91% of patients had "complete" or "good" ROM post-surgery - Strength: 63% had "normal", 35% had "slightly" decreased in strength post-surgery - Return to sport: 86% - Failure rate: n = 2 (4.7%)
Chiang et al. (2010) [78]	Patients with traumatic anterior–inferior shoulder instability (n = 45/45)	"Capsular laxity was evaluated with direct visualisation and palpation with a probe"	Arthroscopic posteroinferior capsular plication and rotator interval closure	- Mean follow-up: 77.1 months - Mean UCLA score pre-surgery: 19.35 ± 1.63; post-surgery: 33.35 ± 3.22 ($p < 0.0001$) - Mean Rowe score pre-surgery: 36.66 ± 7.07; post-surgery: 93.66 ± 16.83 ($p < 0.0001$) - Mean ASES score pre-surgery: 47.77 ± 2.72; post-surgery: 95.84 ± 12.54 ($p < 0.0001$) - Mean external rotation (arm at side) pre-surgery: 1.93° ± 2.8°; post-surgery: 1.97° ± 1.86° ($p = 0.57$) - Subjective outcomes: 42 shoulders had "excellent" post-surgical stability (93.3%) - Return to sport: 100% (42) of patients without reoccurrence of instability - Failure rate: 6.6%
Chechik et al. (2010) [79]	Subgroup of patients with recurrent anterior shoulder dislocations (n = 37/83)[9]	Positive inferior sulcus sign with arm in external rotation, anterior/posterior drawer tests, the ''drive through'' sign, an "RI height" > 15 mm on glenoid side, according to Wynne Davies et al[10]	Arthroscopic Bankart repair and rotator interval closure	- Mean follow-up: 24.1 months (16.8–94.2) - ABR + ARIC patients had less range of motion, but similar results (75%) to ABR group - Joint hyperlaxity (41% of ABR + ARIC patients) was associated with recurrent shoulder dislocations and poor post-surgical outcomes - Return to sport: 22 (59.4%) ABR + ARIC patients played at pre-surgery level; 30 (65%) ABR patients played at pre-surgery level - Failure rate: 3 (8.1%) ABR + ARIC patients experienced redislocations at 42 months post-surgery
Hovelius et al. (2012) [80]	Patients with anterior instability (n = 201/319)[12]	"In shoulders with the sulcus sign or when the joint capsule was considered too redundant"	Capsular shift and capsulopexy for hyperlax patients; Bristow-Latarjet procedure for all patients	Outcomes of the hyperlax patients - Mean follow up: 17 years (10–23); n = 167 - Mean follow up: 6 years (5–8.3); n = 34 - Capsular shift + capsulopexy subgroup: Mean WOSI f: 78.4;,ean SSV: 74.0; mean DASH: 5.8 - Capsular shift + capsulopexy + closure of capsule group: Mean WOSI: 85.3; mean SSV: 80.1; mean DASH: 5.0 - Recurrent instability: 25% (capsular shift + capsulopexy patients) - Recurrent instability: 18% (capsular shift + capsulopexy + closure of capsule patients) - Revision Rate: 13 (4%) patients

Table 5 (continued)

Study	Hyperlax Population[1]	Definition of Hyperlaxity	Treatment	Clinical Outcomes
Fleega et al. (2012) [81]	Patients with anteroinferior shoulder instability (23/75)[5]	Positive anterior apprehension tests, excessive anterior translation, and an elicited positive "drive through" sign in arthroscopy	Arthroscopic inferior capsular shift	- Minimum follow up: 7 years (7–10.5) - Forward elevation range of motion: decreased 0.9° ($p=0.64$) - Internal rotation range of motion: increased 2.1° ($p<0.001$) - ASES score: increased from 70.76 points to 97.53 ($p<0.001$) - UCLA score: increased from 21.97 to 33.84 points ($p<0.001$) - Shoulder redislocations: 4%
Heers et al. (2012) [82]	Patients with post-traumatic anterior instability (n = 37/60)[2]	Joint hyperlaxity "Grade II" or more	Open capsular shift and labral refixation	- Mean follow-up: 3.6 years (2–8) - Mean Rowe score: 88.7 points - Post-operative scores: no difference between hyperlax and non-hyperlax subgroups - Subjective outcomes: 55% of redislocation patients achieved a "very good," "good" (34%), and "satisfactory" (10%) result - Failure rate: 5% (shoulder dislocations post-surgery)
Jones et al. (2012) [83]	Patients with anterior instability (n = 20/20)	"Grade II finding or greater" and a positive drive-through sign during examination	Arthroscopic capsular plication	- Mean follow-up: 3.6 years (2.0–5.5) - Mean KJOC score: 82 (18.2, 28–100) - Mean SANE score: 86 (17.5%, 30%-100%) - Glenohumeral range of motion: no significant difference between operative and non- operative shoulder post-surgery - Return to sport: 18 (90%) patients - 17 (85%) patients returned to preinjury level of play - Failure rate: 10%
Abdelhady et al. (2015) [84]	Patients with recurrent anterior shoulder dislocations and generalized ligamentous laxity (n = 13/13)	Beighton Score > 6 points	Latarjet procedure	- Mean follow up: 33.6 months - Mean external rotation: 69.29° post-surgery - Subjective outcomes: "excellent" (85.7%), "fair" (7.1%), and "bad" (7.1%) - Failure rate: 1 (defined as a redislocation)
Vavken et al. (2016) [85]	Adolescents with generalized ligamentous hyperlaxity (n = 10/15)[14]	Beighton Score > 6 points	Open inferior capsular shift surgery	- Mean follow up: 2 years - Mean ASES score: 88 ± 10 (mean 7.5 years post-surgery) - Mean DASH score: 14 ± 14 (mean 7.5 years post-surgery) - Subjective outcomes: 9 (60%) were "very satisfied," 4 (27%) were "satisfied," and 1 (6%) was "dissatisfied" - Return to sport: 9 (64%) - Failure rate: 7% (defined as recurring instability and pain)
Raynor et al. (2016) [33]	Patients with multi-directional instability (n = 8/86)[8]	Positive sulcus sign with inferior translation > 1 cm and humeral head translation of grade 2 or more in anterior or posterior direction	Arthroscopic pancapsular capsulorrhaphy for all cases and rotator interval closure in select patients	- Mean follow-up: 3.3 years (2.0–6.6) - 7 patients (16.7%) experienced instability after initial surgery - Patient satisfaction: 9 atraumatic patients - Return to sport: 76.7% (23) at same or slightly lower level - Change of activity: 37.5% of atraumatic patients due to instability post-surgery - Failure rate: 13% (3)

Table 5 (continued)

Study	Hyperlax Population[1]	Definition of Hyperlaxity	Treatment	Clinical Outcomes
Ropars et al. (2016) [86]	Patients with anterior capsular redundancy and anterior shoulder instability (n = 39/77)[11]	External rotation > 85 degrees (arm at side), positive sulcus sign, and Beighton score ≥ 5	Latarjet procedure and capsulorrhaphy	- Mean follow-up: 55 months (24–90) - Mean Rowe score: 83.9 (35–100) in the anterior capsular redundancy (ACR +)subgroup - Mean Constant-Murley score: 91 (64–100) - Mean Walch-Duplay score: 79.8 (25–100) - Comparisons: Anterior capsular redundancy patients were associated with female gender (p = 0.048); n = 17 (ACR +) versus 6 (ACR-) - Positive sulcus sign: More ACR + patients (23) had positive sulcus signs (p = 0.001) compared to ACR- patients (38)
Maiotti et al. (2021) [16••]	Athletes with reoccurring shoulder anterior instability, hyperlaxity, and glenoid bone loss < 15% (n = 397/397)	According to the Neer and Coudane-Walch tests	Arthroscopic Bankart repair and arthroscopic subscapularis augmentation	- Mean follow-up: 28.7 (24–109) - External rotation: decrease in 15° (adduction), 10° (abduction) - Mean Rowe score: increase from 68.5 to 92.5 (p = 0.037) - Mean WOSI score: 321 - Mean ASES score: increase from 71.5 pre-surgery to 97.4 post-surgery (p = 0.041)
Mitchell et al. (2021) [87]	Patients with multidirectional shoulder instability (n = 42/42)	Positive sulcus sign, capacious capsule, positive drive-through sign on arthroscopy	Arthroscopic capsular repair	- Mean follow up: 6.3 years (2.8–10.2) - Mean SANE score: 83.3 - Mean PASS score: 85.0 - Return to sport: 56% - Failure rate: 13 (26.0%)
Kazum et al. (2022) [88••]	Patients with symptomatic shoulder instability and hyperlaxity (n = 19/19)	Negative apprehension tests	Arthroscopic Trillat surgery	- Mean follow-up: 24.8 months (12–51) - Range of motion: decrease of 22.1° ± 15.8 and 12.4° ± 10.1 post-surgery - Anterior apprehension test: negative in all patients post-surgery - Mean Constant score: increase from 67.1 ± 10.6 (35–78) pre-surgery to 81.2 ± 3 (75–87) post-surgery (p < 0.05) - Mean Walch-Duplay score: increase from 30.8 ± 19.9 (− 25–65) pre-surgery to 91.8 ± 11.7 (60–100) post-surgery (p < 0.05) - Mean Rowe score: increase from 32.1 ± 12.9 (0–55) pre-surgery to 95.8 ± 9.3 (60–100) post-surgery (p < 0.05) - Mean VAS score: decrease from 2.2 ± 1.7 (0–5) pre-surgery to 0.2 ± 0.5 (0–2) post-surgery (p < 0.05) - Mean SSV score: increase from 58.4 ± 11.7 (30–70) pre-surgery to 88.4 ± 6.9 (70–100) post-surgery (p < 0.05) - Failure rate: 0% (defined as a recurrent dislocation or subluxation)

Table 5 (continued)

Study	Hyperlax Population[1]	Definition of Hyperlaxity	Treatment	Clinical Outcomes
Gruskay et al. (2022) [89••]	Patients with multidirectional instability (n = 44/44)	Degree of anterior shift, posterior shift, and anterior translation	Arthroscopic pancapsulorraphy and rotator interval closure in select patients	- Mean follow-up: 9.0 years (5.1–14.6) - Mean SF-12 PCS score: increase from 42.5 (28.7–57.8) pre-surgery to 55.7 (45.5–63.1) post-surgery ($p < 0.001$) - Mean ASES score: increase from 61.6 (21.6–100) pre-surgery to 89.7 (48.3–100) post-surgery ($p < 0.001$) - Mean SANE score: increase from 59.2 (5–98) pre-surgery to 89.7 (59–99) post-surgery ($p < 0.001$) - Mean QuickDASH score: decrease from 40.8 (2.2–79.5) pre-surgery to 10.5 (0–50) post-surgery ($p < 0.001$) - Instability etiology: no difference between traumatic versus non-traumatic subgroups - Post-surgical instability: 29% (traumatic and atraumatic subgroups) - Failure rate: 5 (10.2%) (defined as having revision surgery or ASES post-surgery score < 65) - Survivorship rate: 88% at 5 years and 82% at 8 years

For clinical studies reporting on patient populations in which only a subgroup of patients were affected by concomitant hyperlaxity, only the outcome of this subgroup is reported, if possible. Abbreviations: *WOSI*, Western Ontario Shoulder Instability Index; *OSIS*, Oxford Shoulder Instability Score; *MISS*; Melbourne Instability Shoulder Score; *NRS*, numerical rating scale; *ASES*, American Shoulder and Elbow Society; *SF-12* PCS, short form physical component; *SANE*, Single Assessment Numeric Evaluation; *Dash*, Disabilities of the Arm, Shoulder, and Hand; *ABR*, Arthroscopic Bankart repair; *ARIC*, arthroscopic rotator interval closure; *UCLA*, University of California at Los Angeles Shoulder Score; *ROM*, range of motion

[1]Total number of hyperlax patients out of entire patient cohort

[2]37 patients were diagnosed with hyperlaxity out of a total of 60 patients

[3]5 patients out of the 25 patient cohort were classified as having hyperlaxity

[4]Marshall JL, Johanson N, Wickiewicz TL, et al. Joint looseness: a function of the person and the joint. Med Sci Sports Exerc 1980: 12: 189–94

[5]23 patients were diagnosed with hyperlaxity out of the 75 total patients in the study

[6]59 patients were diagnosed with hyperlaxity out of the 63 total patients of the study

[7]3 patients were diagnosed with hyperlaxity out of the 10 total patients available for follow-up

[8]8 patients were classified with hyperlaxity out of the 22 patients classified with an atraumatic onset of MDI, out of the total 86 patients in the study

[9]37 patients, who showed a much higher rate of hyperlaxity, underwent arthroscopic Bankart repair with arthroscopic rotator interval closure out of the 83 patients in the study

[10]Wynne-Davies R, Gormley J. Clinical and genetic patterns in osteogenesis imperfecta. Clin Orthop 1981;159:26–35

[11]39 patients presented with anterior capsular redundancy and a high prevalence of joint hyperlaxity, out of a total of 78 patients

[12]201 patients underwent Bristow-Latarjet repair from 1986 to 2004, with 319 total patients in the study

[13]Altchek DW, Warren RF, Skyhar MJ, 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

[14]10 patients were classified with hyperlaxity with 5 patients being excluded as a result of Ehlers-Danlos Syndrome

include plication without glenoid fixation [46, 91••] as well as a suture anchor-based techniques [104, 105]. Advantages of suture anchor based approaches include a lower risk for labral displacement compared to a suture-only fixation to an intact labrum [100] and superior strength especially in the posteroinferior quadrant, where the capsule is known to be least robust [106]. A direction of the plication stitch from inferior-to-superior has been demonstrated to result in comparable reduction of humeral translation but superior external rotation compared to a medial-to-lateral direction [95]. Especially in case of a dysplastic labrum, which is frequently seen in the hyperlax patient subgroup, the post-operative "bumper" height created by capsular plication has been associated with superior postoperative success [107•]. Consistently favorable results have been reported following arthroscopic pancapsular plication in the setting of multidirectional hyperlaxity [33, 77, 83, 87, 89••] (Table 5), which is thus considered the current arthroscopic gold standard. More recently, authors have proposed that posteriorly, a posteroinferior plication using a 1–2 anchor construct, shifting the capsular tissue superomedially to reduce the postero-inferior axillary pouch may be sufficient to facilitate a symmetric reduction of capsular volume [78, 108•, 109]. With small case series reporting promising outcomes, more comprehensive evidence is necessary to validate this technique[78] (Table 5).

Rotator Interval Closure

The technique of rotator interval closure (RIC) relies on a shift of the rotator interval, either in the medio-lateral or infero-superior direction, and can be performed open or arthroscopically [110]. The aim is to shorten and tension the coracohumeral ligament to adress inferior and posterior instability [110]. However, given the heterogenous terminology and technique descriptions, there exists no consensus for the optimal indication [110, 111]. While the procedure reduces the overall glenohumeral translation most prominently in the inferior direction in biomechanical models [19, 112], data on a clinically relevant effect on stability remain heterogenous [38, 113, 114]. Furthermore, studies associated the procedure with a loss of ROM [111, 113]. While limited evidence supports RIC as a standard procedure in shoulder stabilization, contemporary concepts suggest employing RIC in the setting of unilateral or multidirectional instability with a significant (postero-)inferior component or inferior hyperlaxity, as well as patients with a positive sulcus sign in 30 degrees of external rotation [110]. Performing an additional RIC tailored to the patient-specific conditions as a part of a more comprehensive stabilization surgery has demonstrated convincing results in specific indications (Table 5) [33, 77–79, 89••].

Arthroscopic Subscapularis Augmentation

Arthroscopic subscapularis augmentation (ASA) alongside the classic Bankart repair, has been shown to be a promising option to manage a combination of shoulder hyperlaxity, anterior shoulder instability, and glenoid bone loss (GBL) [16••]. During the ASA procedure, a partial subscapularis tenodesis at 2 o'clock (right shoulder) is performed to (re-) tension the subscapularis tendon. Though a relatively new technical innovation, studies have shown that the ASA procedure has led to favorable post-operative outcomes, sustaining a functional range of motion (Table 5) [16••, 115]. It has also been shown that for patients with hyperlaxity and shoulder instability, ASA combined with a Bankart repair yields superior outcomes to isolated Bankart repair [16••].

Bone Block Transfer

There is limited evidence on the utility of isolated bone-block transfers, such as the Latarjet procedure, in the setting of instability and hyperlaxity, as patients with concomitant hyperlaxity do not typically develop massive glenoid or humeral bone loss. Furthermore, there is concern that the increased translation in the setting of hyperlaxity cannot be corrected by the addition of a bone block [116]. However, the combination of a bone block transfer with a combined soft tissue repair has been identified as a promising avenue of treatment. Studies reporting on the outcomes of surgical techniques adding a suture-anchor based Bankart repair [76], a concomitant capsular shift [80], or an inferior capsular shift [86] to a bone block transfer procedure reported favorable (Table 5).

Acknowledging the potential drawbacks of a bone-block transfer, like the Latarjet procedure, such as the necessity to detach the labrum, abrade the glenoid, and pass the coracoid block through the subscapularis tendon, modern techniques such as the arthroscopic Trillat procedure seek to eliminate these drawbacks by harnessing the beneficial sling-effect of a coracoid transfer in the setting of hyperlaxity. This procedure consists of a closing-wedge osteotomy of the coracoid and subsequent screw fixation and has demonstrated promising clinical results in initial case series (Table 5) [117••].

Treatment in the Setting of Shoulder Instability and HSD/hEDS

Non-surgical Treatment Considerations for Patients with Shoulder Instability and HSD/hEDS

Patients who are diagnosed with HSD/hEDS should receive education on the nature of the condition and should be provided resources for further support. Given the abnormal soft tissue constitution, first-line treatment for patients with shoulder

instability in the setting of a HSD/hEDS diagnosis is typically conservative. Non-surgical management includes physical and occupational therapy for the management of shoulder instability and other musculoskeletal issues related to the diagnosis. Treatment regimens are similar to patients diagnosed with hyperlaxity in the absence of HSD/hEDS, with a focus on improving joint proprioception and stability in addition to strengthening the periscapular muscular stabilizers [12]. Exercises should be tailored to the primary direction of instability, and weights > 2 kg should be discouraged in the early phases of rehabilitation due to risk of inferior subluxation [118]. Patients may be referred to a physical therapist specializing in the management of HSD/hEDS and related conditions. For patients with an uncertain diagnosis, referral to a rheumatologist or clinical geneticist can be beneficial. Additional specialist referrals may be necessary depending on the systemic clinical manifestations. As chronic pain in EDS is common and can be severe, a multidisciplinary approach to pain is typically an important component of treatment [119]. Outcomes of non-surgical treatments for HSD/hEDS shoulder instability patients can be seen in Table 6.

Surgical Treatment in the Presence of HSD/hEDS

Surgical management of patients with HSD/hEDS is particularly challenging due to inherent collagen defects that contribute to ligamentous laxity and impaired healing [24, 122]. Furthermore, there may be an increased risk of bleeding in patients with hEDS due to blood vessel friability [123••, 124, 125]. Outcomes for surgical treatments of HSD/hEDS shoulder instability patients can be seen in Table 7.

Arthroscopic Pancapsulorraphy/Pancapsular Shift

Patients with HSD/hEDS typically have massively increased capsular redundancy [46, 132, 133•], often resulting in a multidirectional type of instability. As a result, a combination of different technical approaches may be warranted in cases of severe shoulder instability. As such, patients may benefit from an arthroscopic pancapsular shift that addresses both the anterior and posterior capsule [105]. Although the procedure has reported favorable outcomes in patients without connective tissue diseases [33, 73, 89••, 133•], the procedure may be less durable in patients with HSD/hEDS due to recurrent stretching of the capsular tissue over time [134]. This becomes apparent in a case series by Vavken et al. reporting on 15 patients (18 shoulders) with shoulder hypermobility and a diagnosis of connective tissue disease who underwent open inferior capsular shift and capsular plication for multidirectional shoulder instability. In this cohort, 8 (53%) patients reported continuing instability events after the surgery, with the majority of these being subluxations [85].

Graft-Based Capsular Reconstruction

For hEDS patients with particularly poor-quality tissue or prior failed shoulder stabilization surgery, a capsular reconstruction may be used in conjunction with a capsular shift procedure to reinforce the repair. Technical notes describe a combination of a standard inferior capsular shift procedure with an anterior and/or posterior capsular augmentation with Achilles [128, 135], gracilis [136] or tibialis anterior [137] tendon allograft, such as the technique described by Braun, Millett et al. Given the challenging underlying condition, case series have demonstrated favorable outcomes with acceptable failure rates (Table 5) [129•, 135]. Graft reconstruction of the coracohumeral ligament for patients with EDS has also been described in the literature, although data on outcomes are not yet available [138].

Bone Block Transfer

Given the unpredictability of soft tissue-based techniques in these patients, additional bony reconstructive procedures may be warranted, particularly in cases of bone loss or failed prior stabilization surgery. Armstrong et al. [134] described an all-arthroscopic approach to treating patients with HSD/hEDS with recurrent MDI and bone loss. The technique involves anterior and posterior glenoid bone reconstruction using tricortical iliac crest autograft followed by posterior capsular repair and anterior capsular repair and plication. Peebles, Provencher et al. [132] described a technique relying on distal tibial allograft (DTA) for glenoid bone reconstruction, an open capsular shift using a modified T-plasty to reduce capsular volume and a RIC for additional soft-tissue restraint. Presently, outcomes for bony reconstruction procedures in patients with HSD/hEDS are sparse in the literature (Table 5).

Reverse Total Shoulder Arthroplasty (RTSA) and Arthrodesis

If joint preserving stabilization procedures are unsuccessful, a conversion to a RTSA may be considered. Especially in the setting of long-standing instability, instability arthropathy may ensue, requiring a joint replacing treatment option. A study by Rogers et al. [130••] demonstrated that RTSA and TSA procedures can be used successfully in patients with hEDS, with improvements in pain and no reoperations in a cohort of 10 patients. However, the cohort demonstrated a relatively high complication rate of 3/10, with 2 patients demonstrating postoperative instability treated conservatively. As a last resort, arthrodesis may be used to achieve stabilization of the glenohumeral joint, with adequate postoperative ROM permitted by scapulothoracic motion [131].

Table 6 Clinical studies assessing non-surgical treatment and clinical outcomes for HSD/hEDS shoulder instability patients ($N=2$)

Study	Population	Definition of Hyperlaxity	Treatment	Clinical Outcomes
Spanhove et al. (2022) [120••]	hEDS/HSD patients with MDI ($n=21$)	hEDS/HSD patients with MDI	Randomized controlled trial of 2 home-based exercise programs	- Minimum 24 week follow-up: - PROs - Mean WOSI improved by 325 points ($p=0.001$) - Mean DASH improved by 8.6 points ($p=002$) - Mean PSFS improved by 4.3 points ($p=0.01$) -.Mean GROC improved by 1.02 points ($p=0.001$) - No significant improvement in Tampa Scale for Kinesiophobia (TSK) (p = 0.12)
Liaghat et al. (2020) [121]	Patients with HSD and shoulder instability and/or pain for > 3 months ($n=12$)	Shoulder symptoms AND Generalised HSD: Beighton score ≥ 5/9 for females < 50 years and ≥ 4/9 for females > 50 years and males, or historical HSD: Beighton score 1 point below age and sex-specific cutoffs and positive 5-part questionnaire	16-week heavy shoulder strengthening program targeting scapular and rotator cuff muscles	- Follow-up: following 16 week therapy - Mean WOSI improved 528 points (N.S., p > 0.05) - Shoulder strength measurements improved by 28–31% (N.S. $p > 0.05$) - At follow-up, more participants had negative instability tests except for anterior load and shift compared to preoperatively

Abbreviations: *WOSI*, Western Ontario Shoulder Instability Index; *DASH*, Disabilities of the Arm Shoulder, and Hand; *PSFS*, Patient-Specific Functional Scale; *GROC*, Gloal Rating of Change; *HSD*, Hypermobility Specturm Disorder; *EDS*, Ehlers-Danlos Syndrome (hEDS, hypermobility type); *PROs*, patient reported outcomes; *N.S.*, not significant

Table 7 Clinical studies assessing surgical treatment and clinical outcomes for HSD/HEDS shoulder instability patients ($N=5$)

Study	Population	Definition of Hyperlaxity	Treatment	Clinical Outcomes
Vavken et al. (2015) [85]	15 adolescents (18 shoulders) with generalized ligamentous hyperlaxity or Ehlers-Danlos syndrome	Clinical or genetic diagnosis of a connective tissue disease (ex. EDS or Marfan Syndrome), or a Beighton Score > 6 points	Open inferior capsular shift surgery	- Mean follow-up:7.5 years - Patient satisfaction: 13/15(87%) satisfied - Mean postoperative ASES: 87.7 ± 9.4 - Mean postoperative QuickDASH: 13.9 ± 13.7 - Failure (low patient satisfaction and recurrent dislocations): 1/14 (7%) Survivorship with no recurrent instability events: 7/15 47%)
Aldridge et al. (2003) [126]	Female adolescent patient with EDS and bilateral MDI (n = 1 patient, 2 shoulders)	EDS and MDI	Thermal capsulorraphy	- Mean 2 year follow-up: - 1 shoulder with no subsequent instability 1 shoulder with occasional subluxations
Galano et al. (2008) [127]	16-year old female patient with hEDS with MDI (n = 1)	hEDS and MDI	Arthroscopic capsular plicatiion	- Mean follow-up: 21 months - Subjective outcomes: pain free, successful return to activities of daily living - Physical exam: ROM symmetric bilaterally, negative apprehension test, grade 2 anterior and posterior translation - Failure rate: 0%
Macdonald et al. (2008) [126]	Patients with MDI and prior failed shoulder stabilization surgery (n = 4/8 (10 shoulders))[1]	EDS confirmed via genetic testing and MDI	Achilles allograft anterior capsular reconstruction	- Mean follow-up: 27 months (3–70) - Mean WOSI improved 14.2 to 42.0 - 6/8 patients would have procedure again for short-term relief - Failure rate (revisions or conversions): 11/16 procedures (69%)
Chaudhury et al. (2012) [128]	Female patient with hEDS and bilateral MDI with multiple prior failed shoulder stabilization surgeries (n = 1 patient, 2 shoulders)	hEDS and MDI	Open inferior capsular shift and anterior capsular reconstruction with Achilles allograft	- Follow-up: 4 months (L shoulder) and 3 years (R shoulder) - Outcome: no residual instability, patient satisfied with both shoulders
Schoorl et al. (2021) [129•]	Patients with hEDS and severe anteroinferior instability or MDI (n = 4 patients, 5 shoulders)	hEDS confirmed via genetic testing and severe anteroinferior instability or MDI	Open capsular shift and anterior capsular augmentation with Achilles allograft	- Mean follow-up: 3.6 years (2–5) - Mean VAS pain improved from 7 to 2 - Mean postoperative SSV: 84 - Mean postoperative ASES score: 77.3 - 1 shoulder underwent revision surgery for recurrent instability

Table 7 (continued)

Study	Population	Definition of Hyperlaxity	Treatment	Clinical Outcomes
Rogers et al. (2021) [130••]	Patients with hEDS and primary osteoarthritis or rotator cuff arthropathy (n = 10/30, mean age 55 years, all female)[2]	Clinical diagnosis of hEDS	RTSA (for rotator cuff arthropathy) or TSA (for primary osteoarthritis)	- Mean follow-up: 60.7 ± 21 months - PROs: VAS Pain improved from 6.5 to 1.7, ($p < 0.001$), - No difference between hEDS and non-EDS patients for postoperative VAS Pain ($p = 0.78$) - ROM: Improved forward elevation from (96° to 138° ($p = 0.04$); improved external rotation from (36° to 57° ($p = 0.16$), improved internal rotation from (3.5° to 4.7° ($p = 0.10$, N.S.) - No difference between hEDS and non-EDS patients for postoperative ROM ($p = 0.75$, 0.77, 0.84) - Complications: 3/10 hEDS patients (2 recurrent instability, 1 acromial stress fracture) - Reoperations: 0/10 hEDS patients
Legato et al. (2018) [131]	25-year old female patient with EDS and severe MDI with 3 prior failed stabilization surgeries (n = 1)	EDS and MDI	Shoulder arthrodesis	- Adequate fixation achieved - Postoperative ROM: able to reach top of head and lumbar spine through scapulothoracic motion

Clinical studies assessing surgical treatment and clinical outcomes for shoulder instability patients diagnosed with a connective tissue disorder. For clinical studies reporting on patient populations in which only a subgroup of patients were diagnosed with a connective tissue disorder, only the outcome of this subgroup, if possible, is reported

Abbreviations: *WOSI*, Western Ontario Shoulder Instability Index; *ASES*, American Shoulder and Elbow Society; *quickDASH*, Disabilities of the Arm Shoulder, and Hand shortened form; *SSV*, Subjective Shoulder Score (SSV); *VAS*, Visual Analogue Score; *EDS*, Ehlers-Danlos Syndrome (hEDS, hypermobility type); *PROs*, patient-reported outcomes; *ROM*, range of motion; *N.S.*, not significant

[1] 4 out of 8 patients had a diagnosis of EDS

[2] 10 hEDS patients compared to matched cohort of 20 patients without hEDS

Conclusion

In an effort to advance best practices in the patient-specific treatment of shoulder instability, the management of concomitant hyperlaxity has recently received increased attention. Current research shows specialized physiotherapy protocols focused on strengthening of periscapular muscles and improvement of sensimotor control are a promising conservative therapeutical avenue, which can be augmented by device-based intervention in select cases. If surgical treatment is warranted, traditional arthroscopic techniques such as pancapsular shift or plication continue to demonstrate favorable outcomes and are currently considered the benchmark for success. The long-term success of more recent innovations such as coracoid process osteotomies, subscapularis tendon augmentation and capsular reconstruction yet remains to be confirmed. For patients affected by connective tissue disorders, treatment success is less predictable, and the entire array of conservative and surgical options needs to be considered in achieving a patient-specific satisfactory treatment result.

Declarations

Conflict of Interest The authors report the following conflicts of interest not relevant to the work: Peter J. Millett, MD, MSc receives royalties, is a paid consultant, and receives research support from Arthrex, Inc, and owns stock in VuMedi. Patrick Quinn, BA, Marco C. Rupp, MD, and Joan C. Rutledge are employed through the Steadman Philippon Research Institute (SPRI): During the past calendar year, SPRI has received grant funding or in-kind donations from Arthrex, Canon, DJO, Icarus Medical, Medtronic, Ossur, Smith + Nephew, SubioMed, Stryker and Wright Medical. Marco C. Rupp, MD additionally reports grants from an AGA Fellowship, supported by Arthrex, Inc.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

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