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Review article

Posterior shoulder instability

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ABSTRACT

Posterior shoulder instability (PSI) is defined by dynamic, recurrent and symptomatic partial or total loss of posterior joint contact. Anatomic risk factors comprise ligament hyperlaxity, glenoid retroversion or dysplasia, and high horizontal acromial morphology. Associated anatomic lesions comprise labrum lesions, posterior glenoid erosion and/or fracture, and anterior humeral head notching.

We distinguish two subcategories of PSI: functional and structural, respectively without and with anatomic lesions. In both categories, there may be anatomic risk factors. Clinically, functional PSI involves reproducible asymptomatic voluntary subluxation or sometimes reproducible involuntary subluxation. Functional PSI implicates impaired external-rotation rotator cuff and scapular stabilizer muscle activity. Treatment is non-operative, by rehabilitation and shoulder pace maker.

Structural shoulder instability involves anatomic lesions, often due to iterative microtrauma; pain is the most frequent symptom. It usually concerns young athletic subjects, but the clinical forms of structural and of anterior shoulder instability are not superimposable. Treatment may be surgical; arthroscopic labrum repair is effective in the absence of significant bone lesions, whereas otherwise posterior bone block is the treatment of choice. Surgical treatment of scapular features underlying structural PSI is improved by 3D preoperative planning, cutting guides and dedicated internal fixation systems.

Level of evidence: expert opinion.

1. Introduction

The shoulder joint is the least congruent in the human body [1]. Incidence of posterior instability is 4.64 per 100,000 per year, and significantly higher in 14–19 year-old males (31.80 per 100,000 per year in men, versus 5.23 in women) [2]. Posterior instability is much less frequent than anterior instability, ranging between 5% and 24% of cases of shoulder instability, depending on the report [3].

The definition and classification of posterior instability are currently controversial. In 2002, Gerber and Nyffeler [2] described a 3-part classification: group A, static instability; group B, dynamic instability; and group C, voluntary instability. In 2017, Moroder [1] described an "ABC" classification; group A, first episode pf instability; group B, dynamic instability; and group C, static instability.

The present study does not adhere to these classifications, but defines posterior shoulder instability as dynamic, recurrent and symptomatic partial or total loss of posterior joint contact.

This definition is more restrictive, but, as criteria, "dynamic", "recurrent" and "symptomatic" are closer to the definition of joint instability. Thus, the present article excludes:

1 the first episode of subluxation or dislocation, which does not meet the "recurrent" criterion;
2 locked posterior dislocation, which is a permanent loss of contact;
3 Walch B0 pre-arthritic [4] and B1, B2 and B3 arthritic static posterior subluxation [5], which do not meet the "dynamic" criterion;
4 and constitutional hyperlaxity, which is a risk factor that we shall see below and does not meet the "symptomatic" criterion. On the other hand, we consider associated unidirectional instabilities as a multidirectional instability.

Four questions are addressed here:

1 What are the anatomic and lesion-related factors for posterior shoulder instability?

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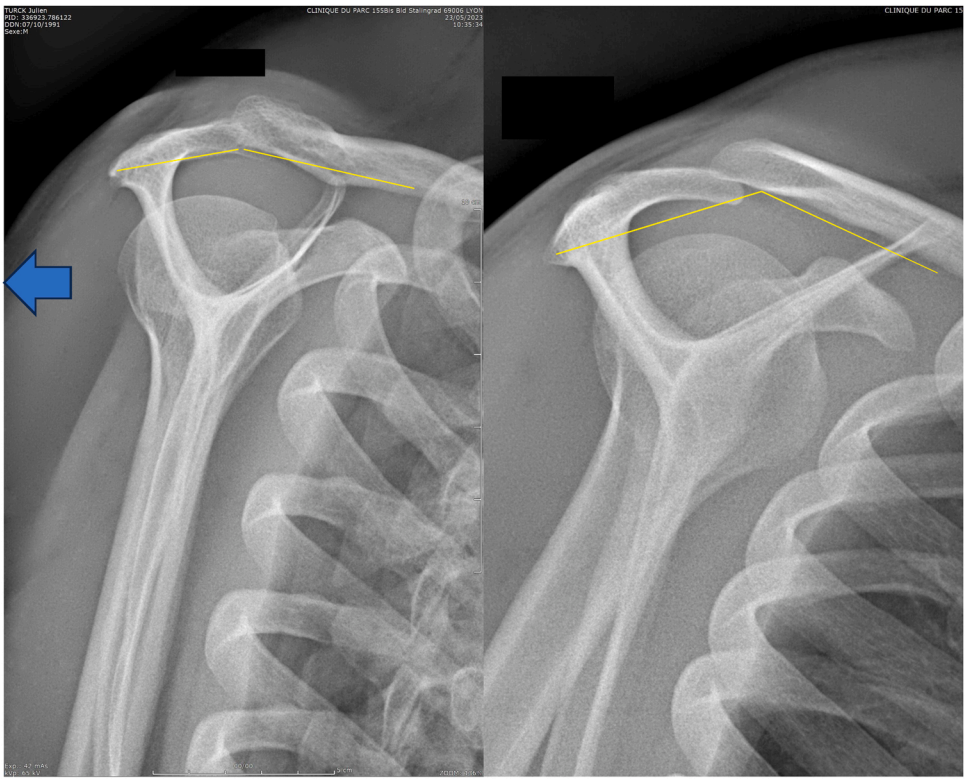
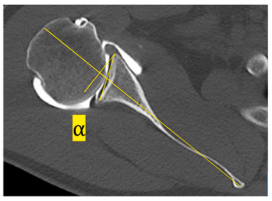


High flat acromion (“flat roof”) promoting posterior translation of the humeral head and posterior instability

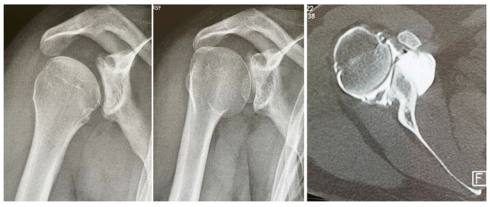
“Swiss chalet roof” acromion, reducing risk of posterior translation of the humeral head

Fig. 1. Acromial morphology.

- Glenoid retroversion measured using Friedman’s line. Normal retroversion is ≤10°



- Example of glenoid dysplasia due to insufficient development of tyhe posterior and inferior glenoid



- Glenoid dysplasia morphologies

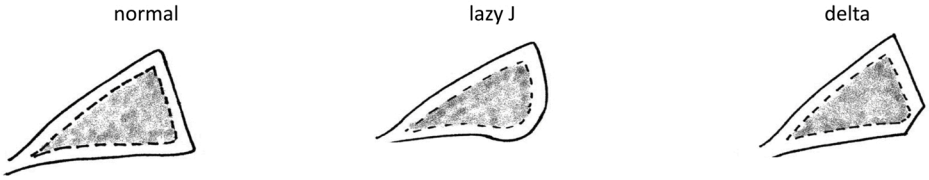
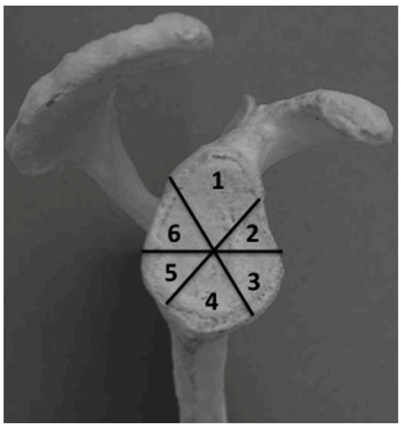


Fig. 2. Glenoid morphology.

2 What are the spectrum of clinical forms and definitions of posterior instability?
3 How to diagnose posterior instability?
4 What are the treatments for posterior instability?



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Clavert P. Glenoid labrum pathology. Orthop Traumatol Surg Res. 2015;101(1):S19-24.

Fig. 3. Six glenoid sectors according to Snyder.

2. What are the anatomic and lesion-related factors for posterior shoulder instability?

2.1. Anatomic factors

2.1.1. Acromion morphology (Fig. 1)

Meyer [6] identified a specific acromion morphology associated with posterior shoulder instability: higher location and more horizontal sagittal orientation, impairing bone resistance to posterior translation of the humeral head.

Conversely, a lower and more vertical acromion, with a "Swiss chalet roof" aspect, is at lower risk of posterior instability.

2.1.2. Glenoid morphology (Fig. 2)

Glenoid retroversion is a risk factor. The method using Friedman's line is the most widely employed, measuring the "alpha" angle [7] between the perpendicular to Friedman's line (neutral version line) and the line between the anterior and posterior edges of the glenoid. On this method, retroversion >10° is a risk factor for posterior instability [8]. Galvin [9] reported 8.2° mean retroversion in shoulders with posterior instability and 2.9° in those without.

According to Weishaupt [10], glenoid dysplasia is a risk factor for posterior instability. It is defined as insufficient development of the posterior and/or inferior part of the glenoid, and is characterized by scapular neck hypoplasia, posteroinferior glenoid bone deficit, jagged aspect, sometimes with a notch in the joint surface, and enlarged inferior glenohumeral joint line. It is usually primary, or in some cases secondary to obstetric brachial plexus palsy or neonatal joint infection. Weishaupt [10] identified 3 forms: normal morphology, rounded "lazy J" shape, and triangular "delta" shape.

According to Paul [11], the incidence of primary Walch C glenoid dysplasia [3] is 1.8%. Walch's original definition of type C was primary dysplasia with >25° retroversion. Paul [11] reported a series of 29 shoulders with 3D type C dysplasia and found a mean 37° retroversion, 23° inclination, decreased joint depth and 90% posterior subluxation of the humeral head; 62% of cases showed scapular neck hypoplasia, and the most frequent glenoid morphology was "lazy J".

2.1.3. Constitutional hyperlaxity

Walch [12] defined constitutional shoulder hyperlaxity on clinical examination by ≥85° elbow-to-body external rotation (ER1). It is generally bilateral and symmetrical. Other non-quantitative clinical signs comprise anterior and posterior joint drawer and Neer's sulcus sign; the only quantitative sign is >85° passive external rotation.

In constitutional shoulder hyperlaxity, translation and joint surface contact loss are physiological.

Constitutional shoulder ligament hyperlaxity is a risk factor for posterior instability and for failure of surgical management.

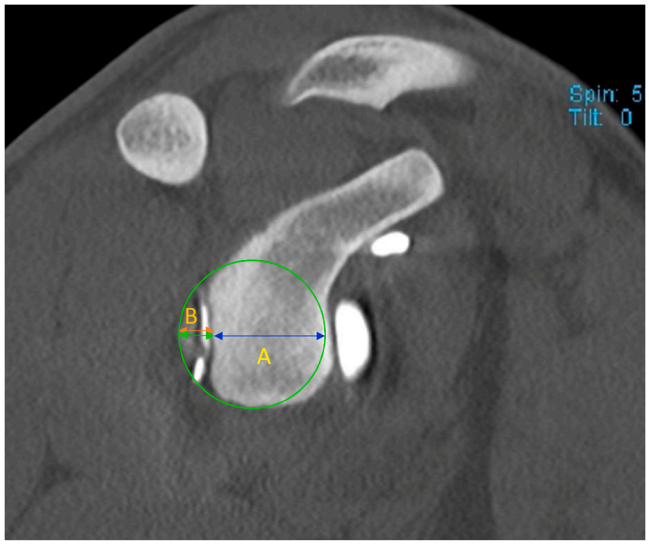


Fig. 4. Posterior glenoid bone defect (fracture or erosion) measured on 2D CT sagittal slice as A/A + B.

In case of constitutional shoulder hyperlaxity, generalized hyperlaxity should be screened for on Beighton score [13]. Suspicion of Ehlers-Danlos syndrome requires multidisciplinary and genetic management.

2.1.4. Humeral head morphology

Humeral head retrotorsion generally varies between 20° and 35° with respect to the elbow condyles. When it exceeds 35°, there is probably greater risk of posterior instability, although there are no dedicated studies.

2.2. Structural lesions in posterior shoulder instability

2.2.1. Glenoid side

The labrum is a major contributor to shoulder stability, with 3 functions: increasing the contact area between humeral head and scapula (2 mm anteroposteriorly and 4.5 mm superoinferiorly); contributing to the viscoelastic piston effect by maintaining −32 mm Hg intra-articular negative pressure; and allowing insertion of ligament and capsule stabilization structures [14].

Snyder [15] divided the glenoid into 6 sectors (Fig. 3). In case of posterior instability, labrum lesions are classically in sectors 5 and 6, and are known as "reversed Bankart lesions", by analogy to the

anteroinferior labrum lesions seen in case of anterior instability.

Posteroinferior labrum lesions may be associated with glenoid bone lesions, creating a glenoid defect, reverse bony Bankart lesion, or erosion. Glenoid defect can be measured on Pico's method [16] on 2D CT sagittal slices, using a circle adjusted on the anteroinferior edge of the inferior glenoid so as to approximate native glenoid morphology. The A-B line is the diameter of the best-fit circle and the A line is the diameter of the posteroinferior defect. Percentage posteroinferior defect is calculated as A/(A + B) (Fig. 4).

Posteroinferior lesions may be associated with chondral lesions, which are of poor prognosis for clinical and surgical results [17].

2.2.2. Humeral side

Posterior dislocation can lead to anterior humeral notching, known as "reverse Hill Sachs lesion" by analogy to anterior instability. These notches are pathological anatomic factors for recurrence of instability, especially as they induce engagement.

Moroder [18] defined a "gamma angle" to measure the notch on an axial slice. It is subtended by a line through the bicipital groove and a line through the medial part of the notch. Gamma angle exceeding 90° is considered as inducing engagement.

2.2.3. Ligaments

Two ligaments are largely involved in the posterior stability of the humeral head: the coracohumeral ligament, and the posterior band of the inferior glenohumeral ligament [19].

Lesions in the posterior band of the inferior glenohumeral ligament induce pathological posterior shoulder laxity, causing instability. Posterior shoulder laxity is hard to diagnose: clinically, as there is no posterior equivalent of the Gagey test [20] for anterior instability, and paraclinically, as intrinsic ligament lesions are hard to identify on imaging.

2.2.4. Muscles

The rotator cuff and periscapular muscles play a predominant role in posterior functional shoulder instability. Scapulothoracic dyskinesia and functional shoulder instability are often associated. These abnormal movements in the scapulothoracic joint result from defective muscle contraction.

3. What are the spectrum of clinical forms and definitions of posterior instability?

The clinical forms of posterior instability differ from those of true anterior instability. Patients are often young and athletic, but the sports differ, with contact and collision sports such as downhill cycling, rugby, wrestling and martial arts classically incriminated in posterior instability [21,22]. Trauma may be acute in adduction, internal rotation and flexion or with continuous backward axial loading, although repeated microtrauma is most often implicated.

Presenting symptoms comprise isolated pain, or recurrent episodes of subluxation or dislocation or both. Etiology is traumatic or microtraumatic. Epilepsy and electrocution induce posterior instability, classically bilaterally. Like in anterior instability, diagnostic, prognostic and therapeutic work-up includes screening for joint hyperlaxity.

Moroder's ABC classification [1] is a pathophysiological approach to posterior instability:

- Type A, first acute episode of posterior instability: A1 = subluxation, A2 = true dislocation;
- Type B, dynamic posterior instability: B1 = functional dynamic posterior instability, with abnormal muscle activation pattern, often associated with hyperlaxity; B2 = structural dynamic posterior instability, with labral or glenohumeral bone lesion secondary to inaugural trauma or iterative microtrauma;
- Type C, acquired or constitutional static posterior instability.

Dynamic posterior instability comprises the following clinical spectrum.

3.1. Recurrent true posterior dislocation

The main symptom is dislocation, defined by total loss of contact requiring reduction by external maneuver. This clinical form is superimposable on post-traumatic recurrent anterior dislocation.

3.2. Dynamic posterior subluxation

The main symptom is recurrent subluxation, defined by transient partial or total loss of contact with the humeral head. Subluxation may be reproducible or non-reproducible.

3.2.1. Non-reproducible subluxation

The patient cannot reproduce the subluxation. Clinical presentation and treatment are similar to recurrent true posterior dislocation. This clinical form is superimposable on recurrent anterior subluxation.

3.2.2. Reproducible subluxation

The patient can reproduce the subluxation.

- ***Voluntary reproducible subluxation***: controlled, painless, recurrent subluxation without apprehension, induced by muscular co-contraction. It may be associated with a particular psychological profile, with a recreational aspect or "party-trick shoulder".
- ***Involuntary reproducible subluxation***: uncontrolled recurrent subluxation, sometimes troublesome, painful and unwanted. It may occur at rest (non-positional) or in movement (positional).
- Voluntary and involuntary reproducible subluxation are classified as functional posterior instability, and involve no anatomic lesions; imaging is normal, and treatment is non-operative, as surgical results are unpredictable and often poor [23].
- ***Voluntary subluxation becoming involuntary***: The patient has lost control of subluxation, which is now occurring involuntarily. There is often a trauma leading from voluntary to involuntary subluxation. Imaging often shows lesions causing structural dynamic posterior instability. In this clinical form, anatomic lesions may require surgery.

3.3. Posterior unstable painful shoulder

Pain is the leading symptom [17,24].

Posterior unstable painful shoulder (P-UPS) is hard to diagnose, and involves a combination of clinical and imaging features: 1) isolated pain; 2) unnoticed posterior instability accident or iterative microtrauma revealed on exhaustive interview, this strong prevalence of microtrauma distinguishing posterior from anterior UPS; 3) no clinical history of shoulder dislocation or subluxation; 4) pain on posterior instability testing; 5) posterior and/or inferior labrum lesions on CT arthrography; and 6) no chondral lesions or osteoarthritis on CT arthrography. Patients presenting objective dislocation or subluxation are excluded from this category.

There may be a continuum between dynamic posterior instability and static posterior subluxation of the humeral head, but this remains to be demonstrated, although the hypothesis could account for the pathophysiology of static posterior subluxation.

The particular case of multidirectional instability associates several unidirectional instabilities: for example, anterior instability with traumatic dislocation associated with posterior UPS and circumferential labrum lesion. Usually, multidirectional instability is bidirectional. Some studies reported rates of up to 40% for posterior instability associated with instability in another direction [25].

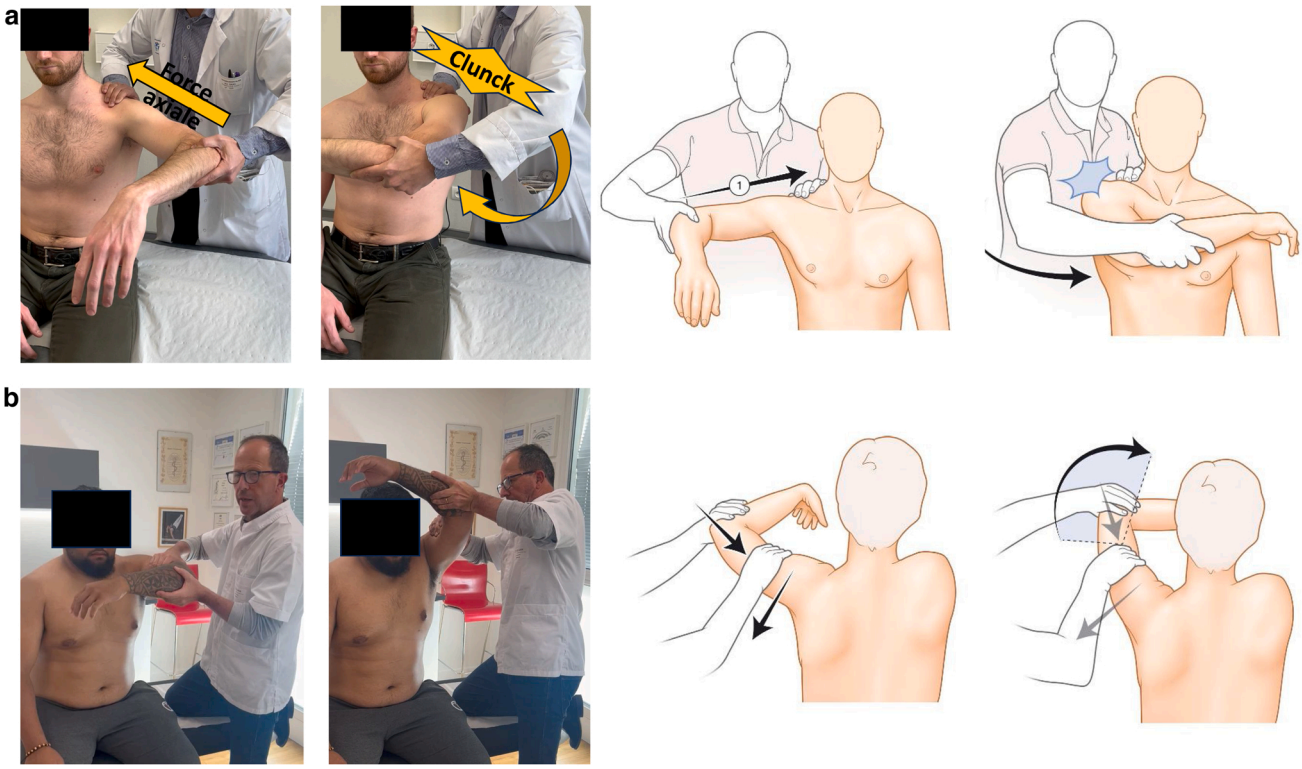


Fig. 5. a: Jerk test. b: Kim test.

4. How to diagnose posterior instability?

4.1. Clinical examination

4.1.1. Interview screens for factors for posterior instability

Inaugural trauma or first episode of instability (which can only rarely be identified), and repeated microtrauma in sport or at work.

The most frequently reported symptom is pain: posterior, deep and often associated with shoulder discomfort, jerk or cracking. Pain often impairs sports activities and strength [26,27]. The presentation of posterior instability, superimposable on anterior instability, mainly comprises apprehension and episodic dislocation or subluxation.

Interview should screen for voluntariness, painlessness and absence of serous discomfort, onset often in childhood, and often at a first consultation with normal paraclinical parameters.

Voluntariness and reproducibility are screened for. Reproduction of symptoms by the patient at rest or in flexion/adduction/internal rotation is experienced as unwelcome, and often uncomfortable or painful [28].

Patients present instability phenomena that can be reproduced on request, at rest (non-positional) or in movement (positional). These phenomena are experienced as unwelcome.

4.1.2. Physical examination is bilateral-comparative

Joint range of motion is assessed, and signs of hyperlaxity are screened for: sulcus test, multidirectional drawer, and >85° elbow-to-body external rotation (ER1).

Painful posterior drawer test is screened for, reproducing reported symptoms, often revealing a posterior labral lesion or apprehension or pain in flexion/adduction/internal rotation.

The jerk test and Kim test are classically performed with the patient seated, arm in 90° abduction and internal rotation (Fig. 5).

- Jerk test [21]: the examiner stands behind the patient and blocks the scapula with one hand and holds the elbow with the other. Via the elbow, a backward axial force is exerted on the arm and adduction then abduction are imposed with the forearm held horizontally. The test is positive if it induces a jerk, with or without pain, and reveals posterior labrum lesions.
- Kim test [29]: the examiner stands behind the patient and blocks the upper arm with one hand and holds the elbow with the other. Via the elbow, a backward axial force is exerted, and 45° anterior elevation and adduction are imposed. The test is positive if it is painful, and analyzes posteroinferior labral lesions.

The jerk test has 73% sensitivity and 98% specificity. The Kim test has 80% sensitivity and 94% specificity. Associating the two provides 97% sensitivity for posterior instability [27].

The "thumb test" was recently described to enhance clinical examination [30]. The examiner screens for pain or apprehension in anterior elevation and neutral adduction. The examiner's thumb is positioned behind the shoulder joint line, acting as a posterior block that alleviates the pain or apprehension. Sensitivity and specificity remain to be assessed.

Scapulothoracic dyskinesia should also be screened for. It is classically associated with the functional unstable shoulder described by Moroder [28], but may also be seen in structural instability.

4.2. Imaging

4.2.1. An anterior humeral notch and injury to the posterior edge of the glenoid, ranging from blunting related to dysplasia to actual fracture, are screened for on standard X-ray. Garth and Bernageau views are useful for exploring the posterior edge of the glenoid

The Lamy lateral view analyzes acromial morphology [6], and can reveal posterior cover defect in the humeral head (Fig. 6). The parameters comprise posterior acromial height (PAH) and posterior acromial tilt (PAT). In case of posterior instability, acromial slope is diminished, and the acromion appears flat and in a high position; PAH > 23 mm is associated.

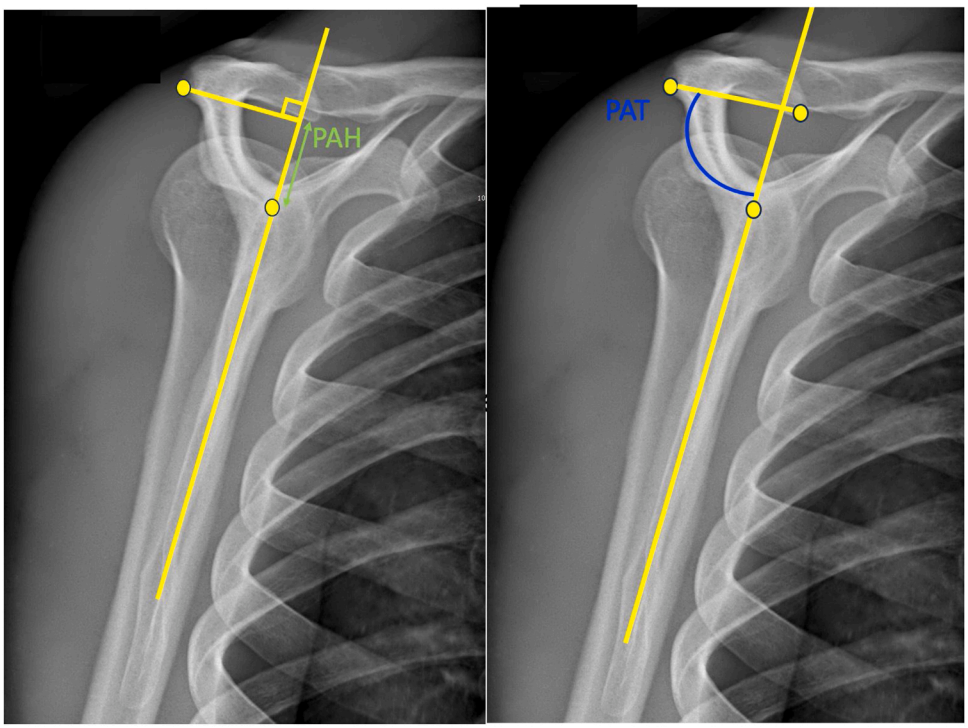


Fig. 6. Posterior acromial height (PAH) and posterior acromial tilt (PAT) measured on lateral X-ray in a patient with posterior instability.

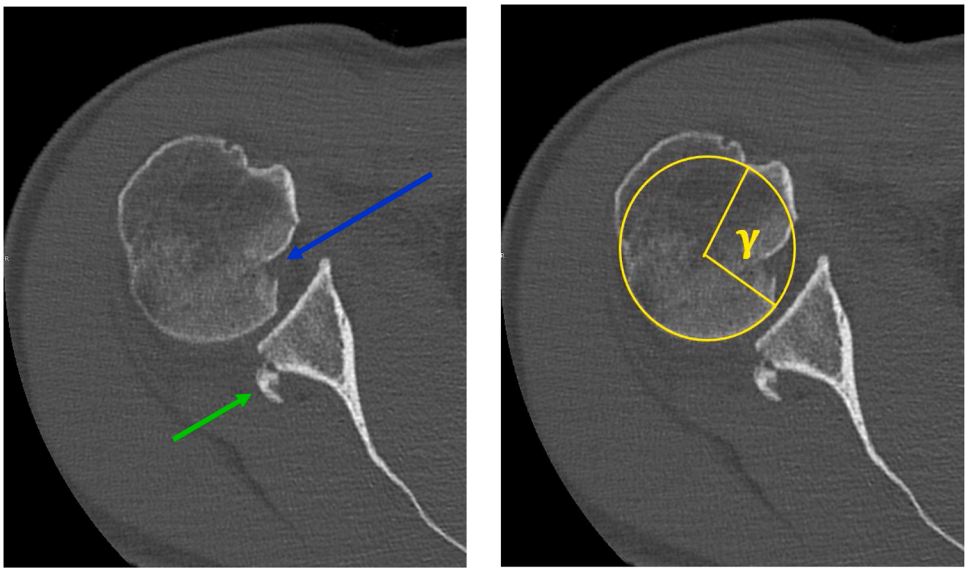


Fig. 7. CT transverse slice in a patient with anterior humeral head notch (blue arrow) and fracture of the posterior glenoid edge (green arrow). Best-fit circle (yellow) and γ angle (yellow). γ angle measured between the middle of the bicipital groove and the medial edge of the notch (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

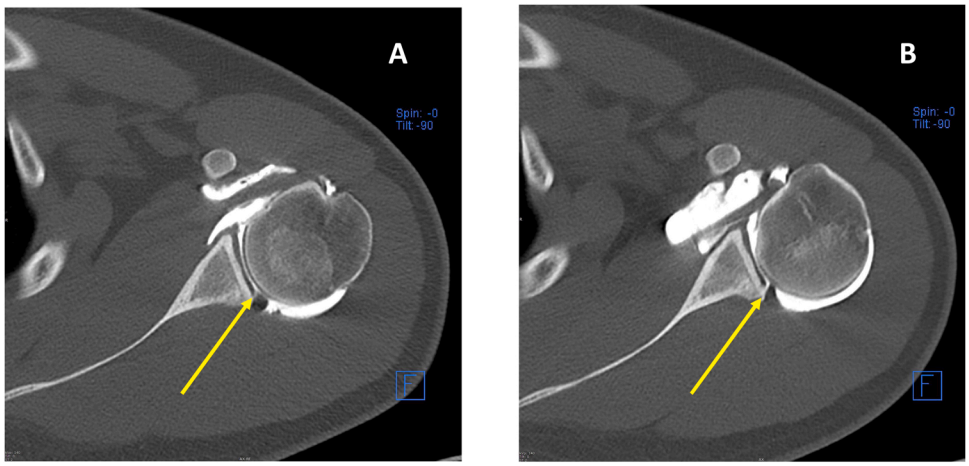


Fig. 8. Posterior Bankart lesion (yellow arrow) on CT arthrography transverse slice, in neutral (A) and internal rotation (B). Internal rotation of the humerus increases contrast medium passage to bone (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

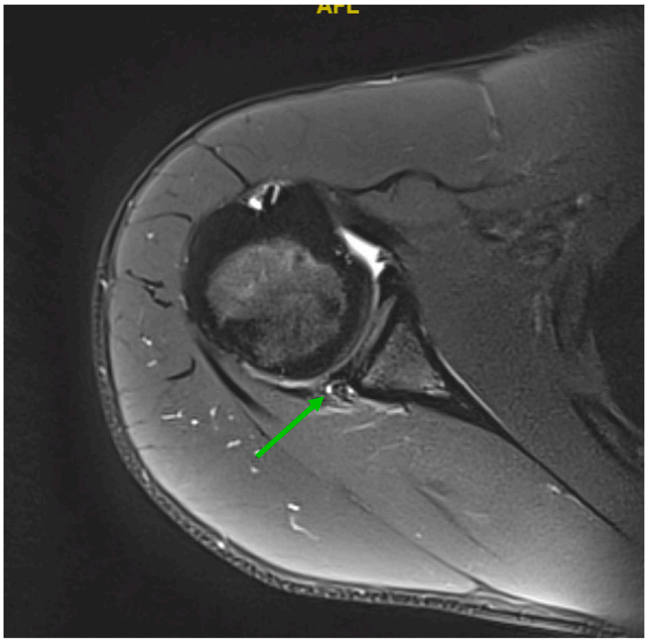


Fig. 9. Kim lesion (green arrow) on transverse MRI slice. Glenoid cartilage in continuity with the superficial facet of the labrum, but with detachment of deep facet in contact with the subchondral bone (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

4.2.2. CT analyzes glenoid and humeral bone parameters and screens for passage lesions (Fig. 7)

Glenoid dysplasia is reported in 49%–93% of studies [9,10,31]. Glenoid retroversion is associated with posterior instability, especially when it exceeds 10° [9,26,32,33].

Humeral notch depth is measured following Moroder [34], using the gamma angle. Gamma angle >90° indicates engagement. Parasagittal slices also assess glenoid defect in case of fracture or erosion. Passage lesions are reported in 25% of cases [24], in the form of fracture of the posterior edge of the glenoid or anterior head notch (reverse Hill Sachs lesion).

4.2.3. Contrast-enhanced MRI and CT arthrography analyze soft tissues, screening for labral lesions, often with associated paralabral cyst [35]

Posterior Bankart lesion is very common, defined as passage of liquid through a labral slit, associated with posterior capsule disruption (Fig. 8). Internal rotation of the humerus increases the passage of contrast medium in contact with the bone. Kim's lesion (Fig. 9), consisting in partial avulsion of the posterior labrum, should also be screened for. It is an incomplete split between the posteroinferior labrum and the joint cartilage, without labral hypermobility [36]; MRI finds incomplete avulsion or loss of height of the posterior labrum, without loss of continuity between the glenoid cartilage and the labral joint surface.

In posterior labrocapsular periosteal sleeve avulsion (POLPSA) [37], there are no associated labral lesions.

These posterior labral lesions may be under-diagnosed, in up to 50% of cases [38]. Unipolar cartilage lesions on the glenoid side (posterior glenolabral articular disruption: GLAD) are of poor prognosis, and screening should be systematic.

Lastly, posterior glenohumeral avulsion of the glenohumeral ligament (PHAGL) was reported in 1% of cases of posterior instability [39].

5. What are the treatments for posterior instability?

5.1. Non-operative treatment

Non-operative treatment is based on rehabilitation of proprioception, reinforcement of the external rotator and periscapular muscles, and avoidance of dislocating or subluxating movements [40,41], and is indicated in first line. Functional unstable shoulder without structural lesions should be treated non-operatively, and surgery is contraindicated. Psychological therapy may be required.

Moroder [42] advocated electrical neuromuscular stimulation by a "shoulder pacemaker" to manage positional functional unstable shoulder, which implicates impaired activity in the external rotation rotator cuff muscles and scapular stabilizers, which the "pacemaker" stimulates. He reported a series of 24 patients [43], for 16 of whom conventional rehabilitation had failed. Patients underwent between 9 and 18 hourly sessions at 1 session per week. Follow-up was 2 years. There were no complications, and all patients achieved shoulder stability without posterior subluxation. Treatment was particularly effective in young, low-weight patients with high activity and unilateral involvement. There was 1 failure, in a patient with glenoid dysplasia and strong retroversion and posterior malalignment of the humeral head. Moroder concluded that shoulder pacemaker treatment showed rapid and lasting efficacy in positional functional unstable shoulder. A multicenter randomized comparative study [44] reported that physiotherapy enhanced by electrical neuromuscular stimulation provided significant and clinically important improvement in positional functional unstable shoulder

Patient in lateral decubitus; Posterior incision 2 cm from posterolateral edge of acromion. Iliac graft harvesting (a: 2-3 cm; b: 3-4 cm; c: 1-1.5 cm).

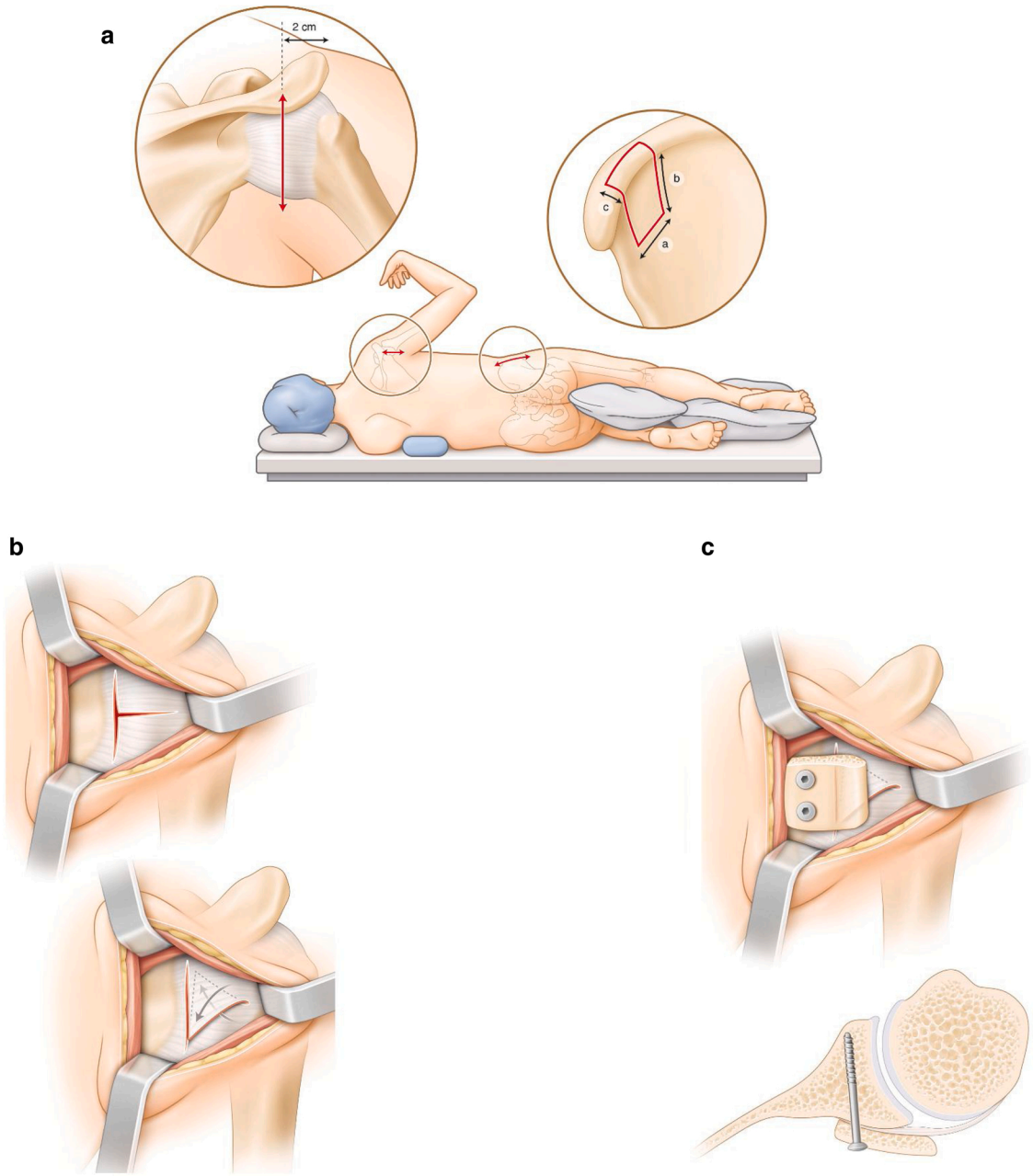


Fig. 10. a: Posterior iliac autograft bone block via dorsal approach. b: T capsuloplasty. c: Protruding extracapsular bone block fixed with 2 screws.

compared to classical physiotherapy alone.

5.2. Arthroscopic posterior labrum repair: reverse Bankart repair

If glenoid bone defect is less than 10% [45] and humeral notch gamma angle is less than 90° [18], isolated arthroscopic posterior labrum repair is the attitude of choice.

Bradley [46] reported 6.4% surgical revision for recurrence of instability after posterior labrum repair. Risk factors comprised female gender, dominant side involvement, concomitant rotator cuff lesions, narrow glenoid (26.4 mm vs 29.1 mm) and use of fewer than 3 anchors for repair.

The meta-analysis by DeLong [47] confirmed the superiority of arthroscopic compared to open surgery, with recurrence rates of respectively 8% and 19%. The arthroscopic technique was simple and reproducible. Six weeks' postoperative immobilization was in neutral rotation, and early rehabilitation avoided internal rotation, to protect the repair. The recurrence rate was 8.1%, surgical revision rate 7.6%, and residual pain rate 12.3%. The main risk factor for persistent pain was posterior glenoid cartilage involvement [16].

5.3. Filling the anterior humeral notch: reverse filling

Risk of humeral head engagement correlates with notch and

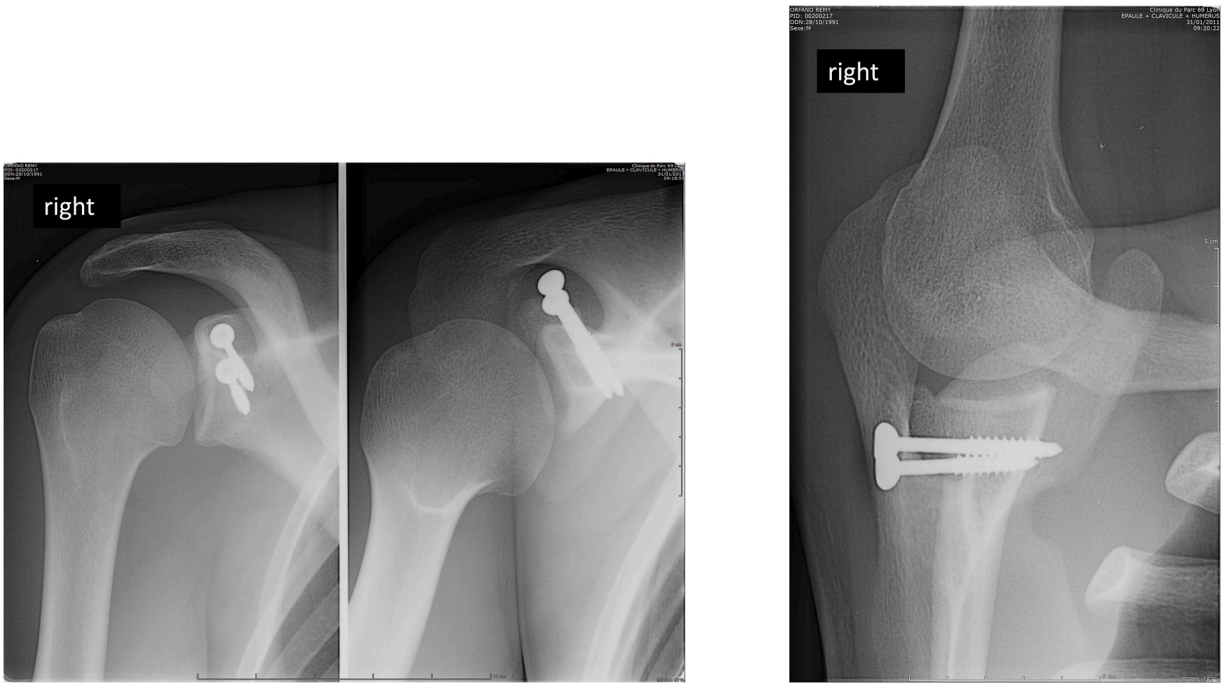
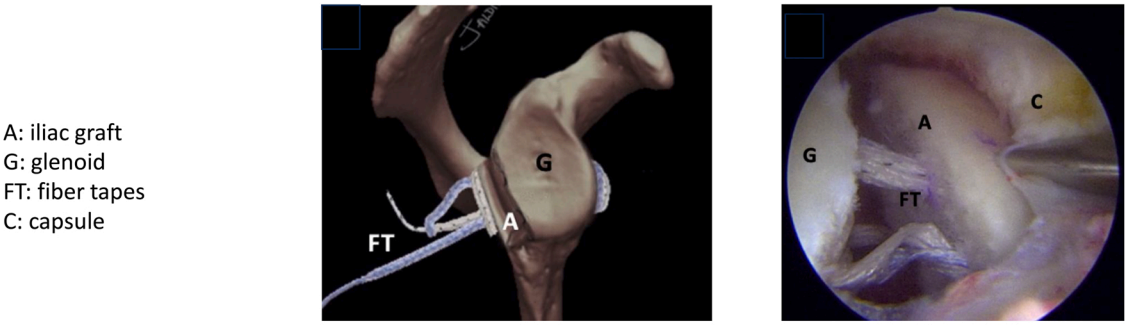


Fig. 11. Posterior iliac autograft bone block, Lévigne approach.



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Hachem A ilah, Rondanelli S R, Costa D'O G, Verdalet I, Rius X. Arthroscopic "Bone Block Cerclage" Technique for Posterior Shoulder Instability. Arthrosc Tech. 2020;9(8):e1171-80.

Fig. 12. Arthroscopic posterior iliac autograft bone block following Hachem.

posterior glenoid defect size. According to Moroder [18], engagement correlates with gamma angle >90°, decreasing by 2.3° per millimeter of glenoid defect.

Filling the anterior humeral notch with subscapularis tendon is an option to prevent posterior engagement of the humeral head. It is performed under arthroscopy, and consists either in folding the tendon into the notch or in releasing it then transposing and fixing it in the notch with 2 anchors with bands crossing the tendon and sutured as bridges [48].

Notch filling associated to posterior labrum repair is indicated in case of engagement (gamma angle >90°) with glenoid defect <10%.

Transposition of the lesser tuberosity of the humerus, following MacLaughlin, is performed in case of locked posterior dislocation, but less often in dynamic posterior instability.

5.4. Posterior glenoid bone-block

Lévigne [49] reported an iliac bone-block technique via a posterior approach. The bicortical autograft measured 3 × 3 × 1 cm, with the cancellous side on the posterior edge of the glenoid (Fig. 10). It was extracapsular, protruding by about 5 mm (Fig. 11). Posterior irritation by screw-heads is a frequent complication, often requiring secondary ablation.

Hachem [50] reported an arthroscopic posterior iliac allograft or autograft bone-block technique fixed by Endobutton (Fig. 12). It is tricortical, measuring 30 × 10 × 10 mm, extracapsular with posterior capsulolabral reinsertion on anchors at end of procedure.

Métais [51] reported an arthroscopic technique with acromial bone-block pediculated on a Kouvalchouk deltoid flap, as an alternative to iliac bone-blocks. It provides triple locking of the shoulder by the blocking effect, retentive hammock effect of the flap, and repair of the posterior capsule (Fig. 13).

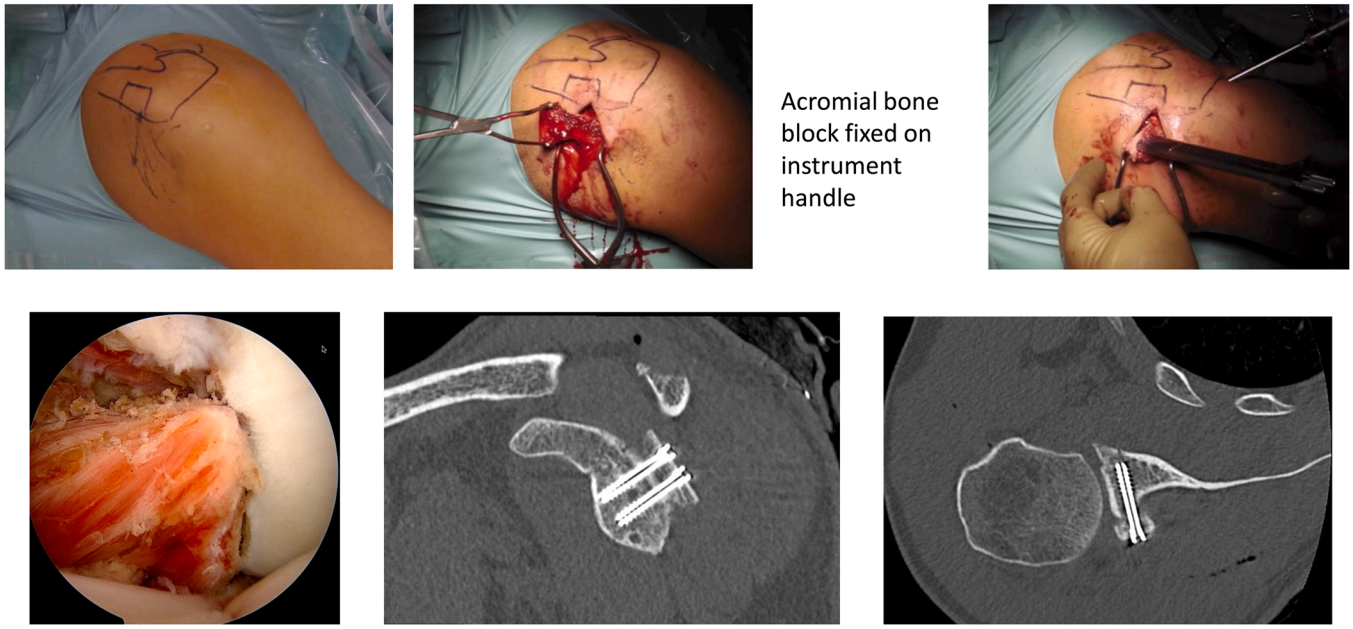

Fig. 13. Acromial bone block pediculated on Kouvalchouk deltoid flap under arthroscopy, following Métais.

Posterior glenoid bone-block associated to capsuloplasty is indicated for glenoid effects exceeding 10%, glenoid retroversion exceeding 10° and recurrence of instability after posterior labrum repair. Posterior bone-block is contraindicated in case of advanced primary posterior subluxating osteoarthritis.

There are few reports of large series with long-term follow-up of posterior glenoid bone-block. Servien and Walch [49] reported 21 cases of open posterior bone-block procedures with iliac autograft; mean age was 24.8 years (range, 17–40 years), and mean follow-up was 6 years. Twenty patients were satisfied or very satisfied. There were 3 recurrences of instability (14%), including 1 of recurrent dislocation and 2 of persistent apprehension, and 4 cases of glenohumeral osteoarthritis (19%). Shoulder pain persisted for 45% of patients. 65% returned to sport at their previous level, 20% at a lower level, and 15% changed or stopped their sport activity. A systematic review of the literature on arthroscopic or open posterior bone-block by iliac autograft [53] confirmed efficacy, but with high rates of revision for fixation screw removal, and with progression toward osteoarthritis.

5.5. Glenoid osteotomy and acromial spine osteotomy

Shoulder instability was defined above as *dynamic* loss of joint-surface contact, thus excluding *static* posterior subluxation. However, differential diagnosis between posterior instability and primary incipient osteoarthritis can be difficult, with a continuum between the two.

Glenoid dysplasia and retroversion exceeding 10° constitute a predisposition toward posterior instability and are risk factors for recurrence after posterior capsule-labrum repair [54]. In a systematic review of the literature, Malik [55], found that osteotomy to correct glenoid version was associated with a 22% rate of revision and 18.3% rate of complications, including 7.3% progression toward osteoarthritis and 5.5% iatrogenic fracture.

Correction of "high and horizontal" acromial morphology, reported by Meyer [6] in patients with posterior instability, could be a treatment option. Gerber [56] reported a case of correction of static posterior subluxation by combined osteotomy of the glenoid and acromial spine (scapular corrective osteotomy for posterior escape: SCOPE). This option, with preoperative planning and patient-specific cutting guides, is a promising option in posterior instability.

Table 1
Differences between posterior and anterior instability.

	Posterior instability	Anterior instability
Epidemiology	<10%	90%
Sport	Contact	Throwing/contact
Mechanism	Adduction/internal rotation	Abduction/external rotation
Etiology	Microtrauma++ Acute trauma	Acute trauma
Bidirectional instability	Systematic screening	Rare
Main symptom	Pain	Instability
Voluntary nature	Classical	Exceptional
Glenoid morphology	Retroversion >10° Dysplasia	Normal
Acromial morphology	High and flat	Low and vertical (Swiss chalet)
Isolated labral repair	Frequent indications	Few indications
Bone-block position	Protruding	Flush

6. Conclusion

Posterior shoulder instability is not superimposable on anterior instability; Table 1 presents the differences.

There are 2 main forms of posterior shoulder instability: functional and structural.

The functional unstable shoulders analyzed by Moroder [28] feature absence of anatomic lesions and do not require surgery; rehabilitation is the treatment of choice. Associating rehabilitation to shoulder pacemaker treatment for hypoactive external rotator cuff muscles and periscapular muscles gives excellent results [43,44].

Posterior structural unstable shoulder involves anatomic lesions, and treatment is usually surgical. If there are no severe bone lesions, Bankart posterior labrum repair is indicated, while glenoid and/or humeral bone lesions are treated by posterior bone-block.

Glenoid correction osteotomy can be useful, but with a high rate of complications [52].

Acromial correction osteotomy is another option [53].

There are correlations between posterior shoulder instability and static posterior subluxation leading to subluxating posterior osteoarthritis.

CRediT authorship contribution statement

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