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INSTRUCTIONAL LECTURE: SHOULDER & ELBOW

Diagnosis and treatment of posterior shoulder instability based on the ABC classification

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- Posterior shoulder instability (PSI) is less common than anterior shoulder instability, accounting for 2–12% of total shoulder instability cases.
- However, a much higher frequency of PSI has been recently indicated, suggesting that PSI accounts for up to 24% of all young and active patients who are surgically treated for shoulder instability.
- This differentiation might be explained due to the frequent misinterpretation of vague symptoms, as PSI does not necessarily present as a recurrent posterior instability event, but often also as mere shoulder pain during exertion, limited range of motion, or even as yet asymptomatic concomitant finding.
- In order to optimize current treatment, it is crucial to identify the various clinical presentations and often unspecific symptoms of PSI, ascertain the causal instability mechanism, and accurately diagnose the subgroup of PSI.
- This review should guide the reader to correctly identify PSI, providing diagnostic criteria and treatment strategies.

Keywords: posterior shoulder instability; classification; therapy; pathomechanisms

ABC classification

Due to the multifactorial etiology and pathomechanism, diagnosis and treatment decisions for posterior shoulder instability (PSI) remain a challenge in clinical practice (1, 2). This is why we introduced the ABC classification, which distinguishes three groups of PSI based on the nature of pathology (first-time, dynamic, or static) and two different subtypes, respectively, based on the pathomechanical causes. This classification aims to facilitate diagnosis and assist the treatment decision-making process (Fig. 1) (3).

First-time acute PSI (Group A)

Group A classifies patients with a first-time singular PSI event less than three months in the past, regardless of etiology. Type A1 stands for patients who reported a posterior subluxation with immediate spontaneous reduction and therefore mostly present with less severe bony or soft tissue defects, while type A2 includes patients with a dislocation requiring a self- or aided reduction maneuver because of temporary or persisting engagement to the posterior glenoid rim. As a result, severe structural defects, including impression fractures of the anteromedial humeral head in terms of a reverse Hill–Sachs lesion (RHSL), fracture-dislocations, posterior glenoid rim fracture as well as soft tissue lesions might occur among A2 patients (4, 5, 6, 7, 8).

The incidence rate of acute first-time traumatic PSI is almost 20 times lower compared to more common anterior shoulder instability, with an incidence rate of

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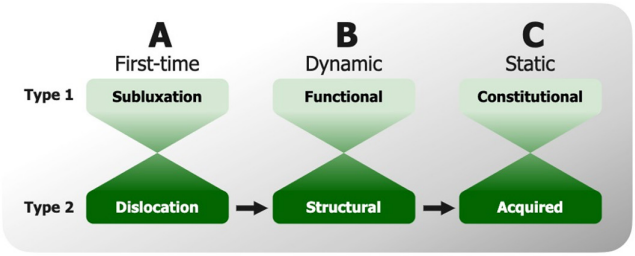

Figure 1

The ABC classification of PSI. Copyright © 2024 Elsevier Inc. Figure reproduced with permission from Moroder *et al.* 2024 (7), 'SECEC Didier Patte Prize 2023: The ABC of Posterior Shoulder Instability.' *Journal of Shoulder and Elbow Surgery* (https://doi.org/10.1016/j.jse.2023.11.019).

1.1 per 100,000 per year, mainly affecting male patients with a peak incidence in the young and active (20–49 years) and in the elderly (>70 years) population (4, 9). As a very rare condition, acute posterior dislocation with fracture-dislocation has been identified to affect only 2–4% of all shoulder dislocations, with an annual incidence of 0.6 in 100,000 (10, 11).

Type A1 is typically caused by a traumatic posterior-directed force in forward flexion, internal rotation, and adduction of the arm, resulting in mild to moderate shoulder pain and a restriction in range of movement. On the other hand, type A2 is typically produced by high-energy traumatic accidents leading to a posterior-directed force, seizures, or electric shocks resulting in aberrant muscle contractions and dysbalance of the internal and external rotators (10).

Dynamic PSI (Group B)

Group B includes patients with recurrent dynamic PSI, regardless of the time since onset. Within this group, patients' history, clinical examination, and imaging are crucial to differentiate both subtypes. Type B1 has been described as functional shoulder instability with either voluntarily (controllable) or involuntary (non-controllable) instability episodes during a specific phase of motion (positional) or even in neutral position (non-positional) due to pathological activation pattern of the rotator cuff as well as periscapular muscles (12). Possible causes include hypoactivity of the external rotators and posterior deltoid with concurrent hyperactivity of internal rotators (e.g. latissimus dorsi) as well as periscapular muscle imbalance with concomitant scapular dyskinesis (12, 13, 14, 15). On the other hand, type B2 describes instability caused by structural damages such as posterior Bankart lesion, posterior glenoid bone loss, RHSL, or a combination hereof due to a single trauma or recurrent microtrauma. These structural defects of the humeral head and the glenoid rim may easily progress to recurrent posterior subluxation or dislocation during motion, especially if patients have coexisting structural and functional deficiencies (hyperlaxity, capsular redundancy, glenoid flattening, or increased retroversion) after suffering from an initial traumatic event (3). Physically demanding shoulder activities such as volleyball, handball, weightlifting, or in the military population can cause repetitive microtraumatic posterior shoulder strain inducing dynamic PSI (12, 16).

Type B1 is the most encountered subtype of functional shoulder instability in general (12). Recently, type B1 has been identified to affect up to 3% of a young and active population with mostly atraumatic onset of first symptoms under the age of 16 years (17). Dynamic PSI can develop over an extensive period of time with the typical active adolescent patient not clearly attributing an initial traumatic event (17). In a young and active military population, PSI has been identified in 10% of all shoulder instability cases, and more than half were affected with recurrent PSI presumably not due to a single, acute traumatic injury but as an accumulative result of repetitive microtraumatic load during military activities such as push-ups, combatives, and weightlifting, which might indicate a higher

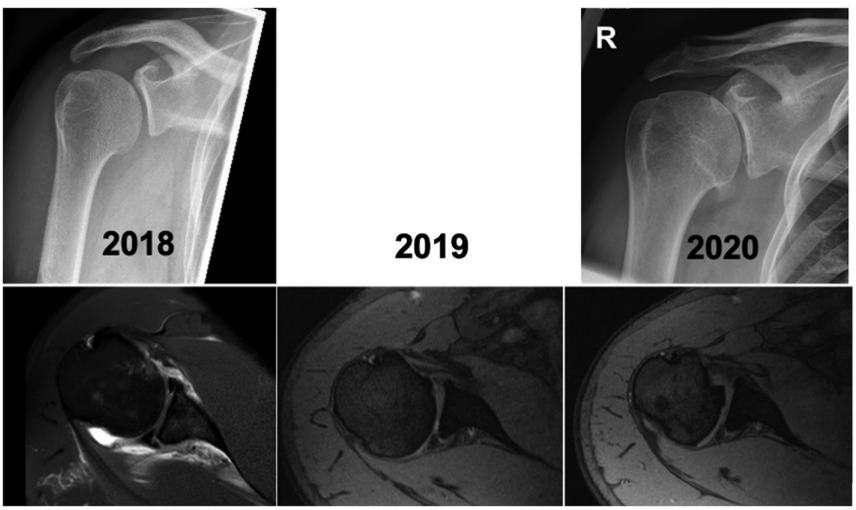

Figure 2

Progression from an acute posterior shoulder subluxation (type A1) to an acquired static PSI (type C2) in a middle-aged male over the course of three years. Copyright © 2024 Elsevier Inc. Figure reproduced with permission from Moroder *et al.* 2024 (7), 'SECEC Didier Patte Prize 2023: The ABC of Posterior Shoulder Instability.' *Journal of Shoulder and Elbow Surgery* (https://doi.org/10.1016/j.jse.2023.11.019).

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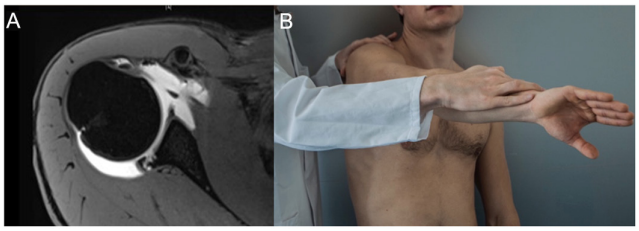

Figure 3

(A) Arthro MRI of a patient with structural PSI (type B2). A typical posterior labral tear without medialization but with a small cartilage defect is illustrated. (B) In order to detect a PSI type B2, the O'Brien test can be performed with the arm in 90° forward flexion and elbow fully extended. The arm is then horizontally adducted and internally rotated, so the thumb faces downward. The examiner applies a downward force to the arm against resistance by the patient (68). Copyright © 2024 Elsevier Inc. Figure reproduced with permission from Moroder *et al.* 2024 (7), 'SECEC Didier Patte Prize 2023: The ABC of Posterior Shoulder Instability.' *Journal of Shoulder and Elbow Surgery* (https://doi.org/10.1016/j.jse.2023.11.019).

occurrence of PSI than previously described in the literature (16).

Patients with type B1 suffer from aberrant muscle activation leading to visible recurrent posterior subluxations or dislocations during a specific phase or range of motion (12, 14). Group B2 patients may complain about nonspecific instability symptoms such as weakness, blockage, clicking noise, or shoulder pain, typically with the arm in flexion and internal rotation.

Static PSI (Group C)

Group C describes patients with PSI and static posterior glenohumeral decentering more than three months in the past regardless of etiology (7), which might be either of constitutional (type C1) or acquired (type C2) origin. Constitutional static PSI is inherent and thus atraumatic in its origin. It is hypothesized that type C1 might be constitutional due to several factors like less humeral retrotorsion, excessive glenoid retroversion, and anterior glenoid offset, as well as differences in acromion morphology (7, 18, 19, 20, 21, 22, 23) since bilateral cases have also been described in the literature (24). According to MRI measurements of 17 type C1 shoulders in 10 patients, significantly less humeral retrotorsion and higher muscle volumes of trapezius and deltoideus were demonstrated in type C1 patients compared to the healthy population (25). Pathologic muscle imbalances and scapular malposition might result in a disproportional articulation of the glenoid and the humeral head, and over time, this condition might induce secondary osseous malformation and lead to a permanently subluxated shoulder in the posterior direction. On the other hand, acquired static PSI is caused by trauma and potentially as a progression of an acute posterior shoulder subluxation or dislocation (A1 or A2) and structural PSI (B2) (Fig. 2) (26).

Type C1 has been observed in a case series of young patients who did not experience any history of trauma, inflammatory disease, or posterior glenoid erosion preceding the static PSI at presentation. Its epidemiological occurrence and risk factors are still poorly understood and need further investigation (24). Type C2 can be observed among young children (<1 years), affecting approximately 7% of all brachial plexus birth lesions (27). In these cases, progressive glenohumeral deformation and posterior glenoid dysplasia develop secondary to an underlying muscle force imbalance. Consequently, the humeral head is positioned excessively in the posterior direction, remaining in a static dislocated position, which may further develop early degenerative changes (28). Type C2 might also occur in the adult patient due to vigorous pathologic muscle contractions during a seizure or due to severe trauma, electrocution accidents, generating forced internal rotation, flexion, and adduction of the shoulder, resulting in locked posterior dislocation which, if left untreated, turns into a chronic condition (29, 30). Type C2 might also occur as a progression of an acute posterior shoulder instability event (A1 or A2) (26) or due to repetitive microtrauma and overload, for example, during excessive weight training (31).

The clinical presentation of group C may vary from asymptomatic during early stages, pain during shoulder-demanding activities, instability symptoms,

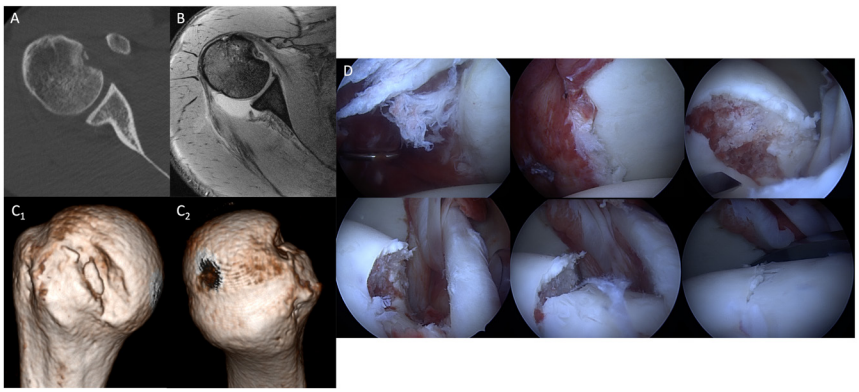

Figure 4

Axial posttraumatic CT (A) and MRI (B) scans after a bike accident with traumatic posterior shoulder dislocation and reposition under analgesia (type A2). (C_1, C_2) 3D anatomical reconstructions of the humeral head demonstrating a reverse Hill–Sachs defect. (D) Arthroscopic view of the retrograde disimpaction of the RHSL combined with posterior capsulolabral repair within a few days after the traumatic event.

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or joint stiffness to permanent pain when posterior cartilage damage and osteoarthritis progress (24). A conspicuous birth history or a severe traumatic posterior force, including a history of seizures or electrocution accidents, could hint at a type C2. These patients may report a limited range of motion or even blockage, restricting any further external rotation and causing shoulder pain (3).

Physical examination

The diagnosis of PSI is based on the medical history and targeted clinical examination of the patient (32). During visual inspection, the examiner looks for macroscopic changes in the shoulder contour, as well as snapping movement patterns in the shoulder area as an indication of pathological glenohumeral translation. Any signs of asymmetry, such as muscle atrophy or scapular dyskinesis, including abnormal scapular winging, protraction, or shoulder drooping, and general body posture should be noted (33). Further evaluation includes the comparison of active and passive range of motion, as well as accurate strength measurement of the injured shoulder compared to the contralateral side. In the evaluation of chronic locked posterior dislocation, limited external rotation might occur with preserved shoulder internal rotation and flexion.

The crucial part of the PSI examination involves performing specific instability tests. The Beighton score (34), Sulcus sign (35), Walch (36), Gagey test (37), and the load-and-shift test (38, 39) are used to analyze capsular and general ligamentous hyperlaxity and hypermobility, which have been associated with glenohumeral joint instability (40, 41). The O`Brien test (group B) and forced internal rotation (FIRO) test (group A) offer a high sensitivity and allow us to rule out the diagnosis of PSI more confidently with a negative result (42). The so-called show-me test seems to offer the best combination of specificity and sensitivity, as PSI patients with type B1 present with highly variable instability symptoms (positional or non-positional as well as controllable or non-controllable), which, however, are typically demonstrable in all cases (7, 12). For type B2, two main provocation tests, the Jerk test (43) and the Kim test (44), should be performed to assess posterior structural components such as the labrum and posterior capsule (7).

Finally, the clinical examination should be completed by evaluating potential coexisting structural lesions of rotator cuff muscles and anterior-superior anatomic shoulder components, for example, the biceps tendon.

Imaging

Radiographic workup for PSI should routinely include true anterior-posterior, axillary, and scapula Y views

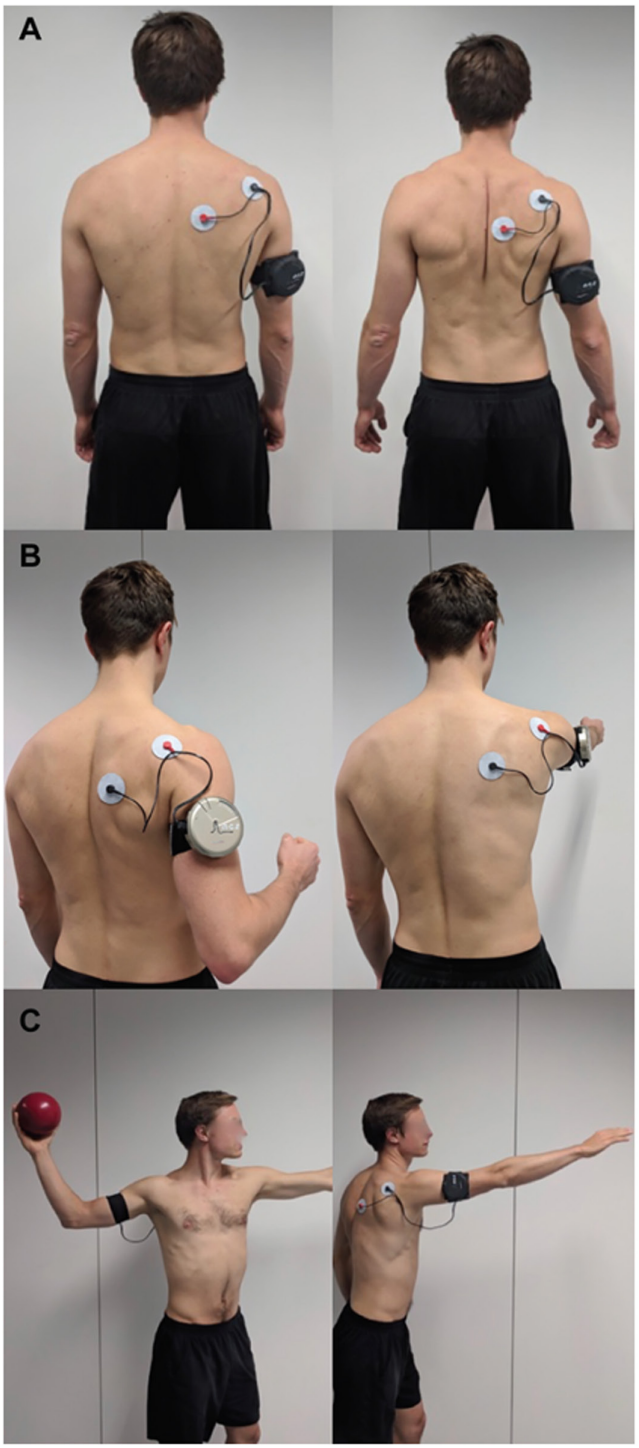

Figure 5

Example of motion-activated neuromuscular electric stimulation with shoulder pacemaker to activate hypoactive muscle groups during concentric (A), eccentric (B), and functional (C) training (e.g. throwing motion). Figure reproduced from Moroder *et al.* 2020 (15), 'Shoulder-Pacemaker Treatment Concept for Posterior Positional Functional Shoulder Instability: A Prospective Clinical Trial.' *American Journal of Sports Medicine* **48**: 2097–2104. (https://doi.org/10.1177/0363546520933841).

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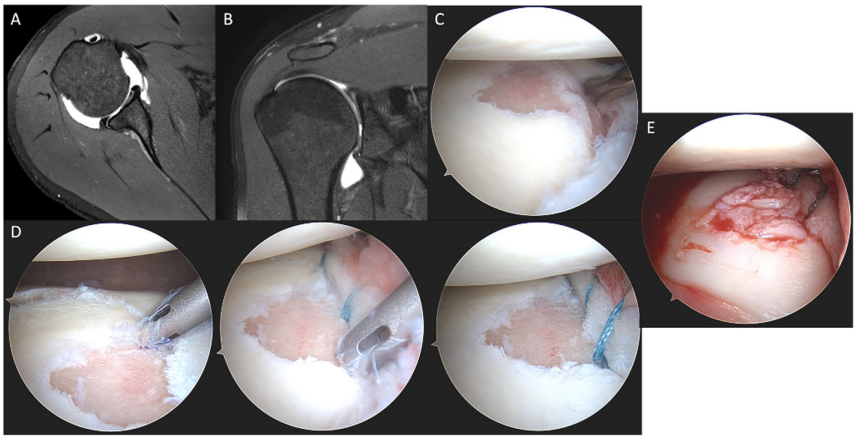

Figure 6

Axial (A) and coronal (B) MRI in a patient with structural dynamic posterior shoulder instability (type B2), showing a grade IV chondral defect of the posterior glenoid as well as a posterior labral lesion. Arthroscopy confirmed the lesions (C). First, a posterior capsulolabral repair was performed (D). Then, autologous cartilage was used for an arthroscopic repair using a minced cartilage procedure (E). (AutoCart, Arthrex, Naples, FL, USA).

of the affected shoulder. Standard radiographs are important to identify an acute or chronic locked dislocation, or fractures, or assess osteoarthritic changes.

CT imaging is very useful in the precise evaluation of bony humeral and glenoid defects, especially for aiding in surgical planning, assessing ongoing degenerative changes, and identifying associated fracture planes. Furthermore, this imaging modality offers a 3D reconstruction of the bony anatomy and the measurement of glenoid retroversion. Additionally, a CT scan can provide hints about atraumatic dynamic PSI with the presence of increased glenoid retroversion and deficiency of the posteroinferior glenoid rim, directing the clinician to the exact diagnosis if only subtle symptoms are present (45).

To evaluate the extent of soft tissue injuries, a MRI examination presents the gold standard. The most common soft tissue injuries are posterior labrum tears (reverse Bankart lesions), posterosuperior tears, and posterior labrocapsular periosteal sleeve avulsions (8). Cartilage defects and concurrent muscle–tendon tears may also be assessed, while bone marrow edemas may indicate acute bony lesions of the glenoid or the humeral head. The detection of small labrum lesions, estimated posteroinferior capsule volume, constitutional soft tissue deficiencies, and degenerative changes due to the increased posterior humeral head translation may be examined in more detail using intra-articular contrast agent application (46, 47) (Fig. 3).

Treatment

First-time acute PSI (group A)

Type A1 comprises young males with an acute traumatic first-time PSI event, occurring as a subluxation with immediate spontaneous recentering of the joint without the necessity for a reduction maneuver. As structural damages are typically limited to the posterior labrum

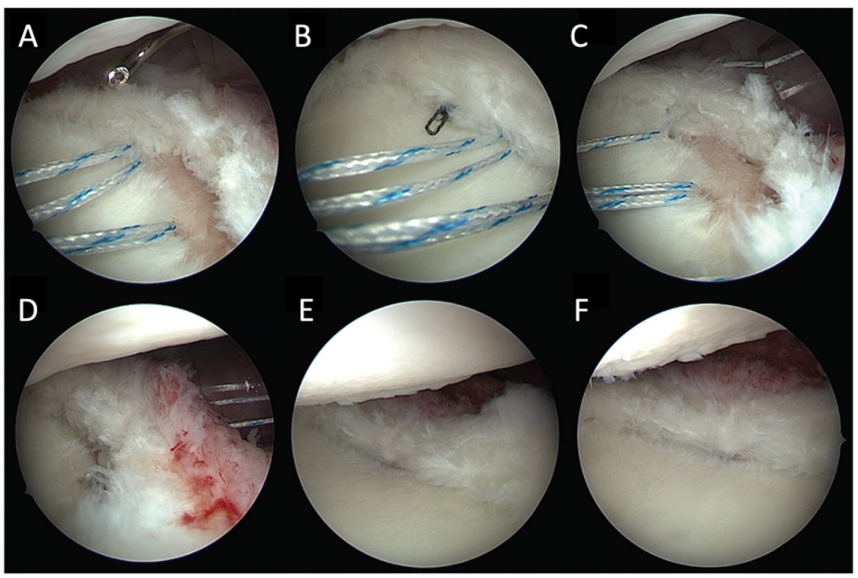

Figure 7

Arthroscopic views from the anterosuperior portal of a right shoulder with the patient in lateral decubitus position. (A–F) Both suture limbs of each anchor are passed through the posterior capsulolabral complex using a suture lasso and tied to form mattress stitches, reattaching and shifting the posterior capsulolabral complex anteriorly and at the same time covering the posterior cartilage defect. Figure reproduced from Moroder *et al.* 2022 (62), 'Arthroscopic Posterior Articular Coverage and Shift (PACS) Procedure for Treatment of Preosteoarthritic Constitutional Static Posterior Shoulder Instability (Type C1).' *American Journal of Sports Medicine* **50**:3617–3624. (https://doi.org/10.1177/03635465221124851).

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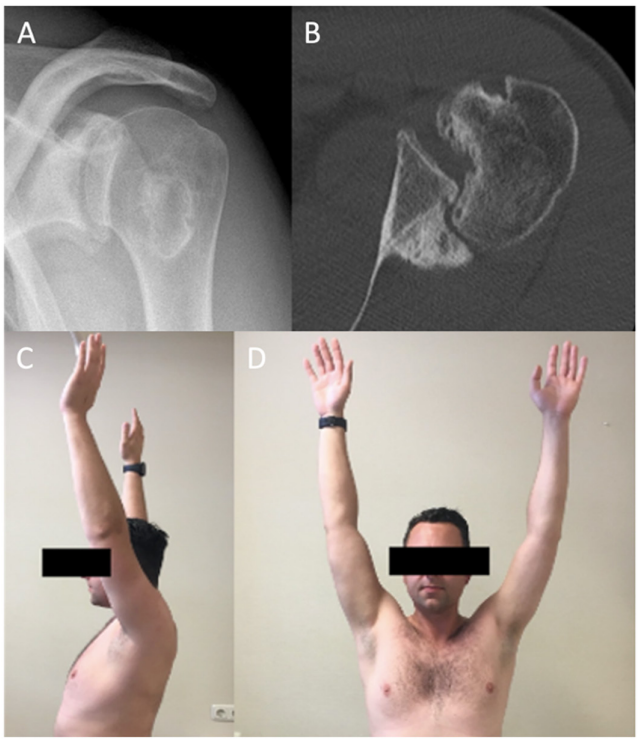

Figure 8

Chronic locked posterior shoulder dislocation (type C2) visible on AP radiograph (A) and axial CT scan (B) with preserved elevation capacity of the affected shoulder (C, D). © 2021 Thieme. Figure reproduced from Ruttershoff *et al.* 2023 (65), 'Joint Preserving Treatment of Chronic Locked Posterior Shoulder Dislocation by Means of Combined Humeral Allograft Reconstruction and Posterior Glenoid Autograft Augmentation.' *Zeitschrift für Orthopädie und Unfallchirurgie* **161**:290–296. (https://doi.org/10.1055/a-1651-0943).

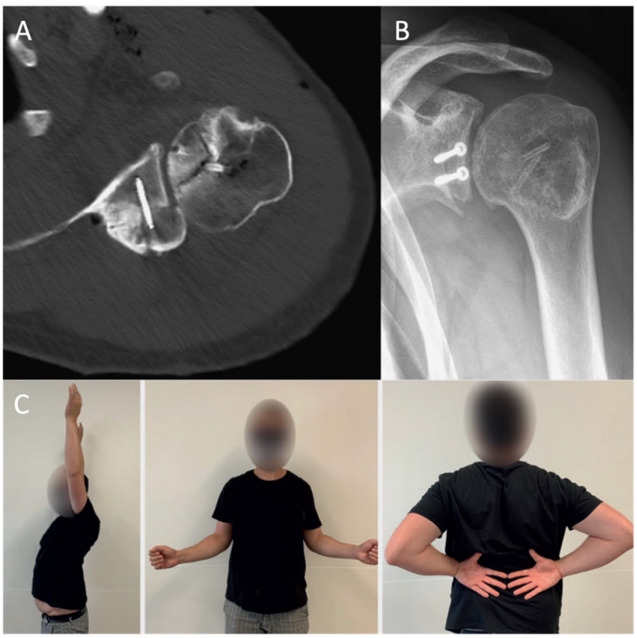

Figure 9

(A) Postoperative CT scan right after the surgery demonstrating anatomical reduction of the former chronic posterior locked humeral head after fresh-frozen femoral allograft reconstruction of the humeral defect and posterior glenoid augmentation with a harvested tricortical bone autograft from the ipsilateral iliac crest. One-year postoperative follow-up with radiographic (B) and clinical (C) evaluation. Figure modified from Ruttershoff *et al.* 2023 (65), 'Joint Preserving Treatment of Chronic Locked Posterior Shoulder Dislocation by Means of Combined Humeral Allograft Reconstruction and Posterior Glenoid Autograft Augmentation.' *Zeitschrift für Orthopädie und Unfallchirurgie* **161**:290–296. (https://doi.org/10.1055/a-1651-0943).

and small RHSLs, conservative treatment is indicated in most cases. Young age, posterior decentering of the humeral head, and a higher gamma angle (according to Moroder *et al.* (48)) are risk factors for recurrence of instability and a worse clinical outcome after non-operative treatment (26).

Type A2 includes middle-aged male patients with an acute traumatic first-time posterior shoulder dislocation requiring a self- or aided reduction maneuver or motion after a certain period of dislocation. In case of a locked dislocation, a closed or open reduction of the shoulder joint is warranted depending on the presence of a concomitant proximal humerus fracture (PHF) and the time passed since the dislocation. A posterior fracture dislocation should be reduced openly and internally fixed, mostly with the use of a plate system (49), while not forgetting to address the RHSL if necessary. The treatment choice in cases without a PHF depends on the severity of the structural defects. Conservative treatment of first-time posterior shoulder dislocation has been shown to result in good outcomes at mid- to long-term follow-up. However, young age, posterior humeral head decentering, and a higher gamma angle remain risk factors for the recurrence of instability and a poor outcome (26). Arthroscopic reconstruction of a diagnosed posterior capsulolabral tear can lead to good to excellent clinical results, especially in young patients with high functional demand (50). An RSHL with a gamma angle >90° should be addressed surgically to reduce the risk of instability recurrence (48), if possible, within a few days after the traumatic event with defect disimpaction combined with posterior capsulolabral repair (51) (Fig. 4). If more than two weeks after trauma have passed, a McLaughlin procedure (32), transposition of the lesser tuberosity (52), or filling the defect with allograft (53) can be performed to address an engaging RSHL in combination with posterior capsulolabral repair.

Dynamic PSI (Group B)

Type B1 demonstrates functional shoulder instability, which, in contrast to other subgroups of PSI, mostly affects female teenagers and young adults. Due to the lack of structural defects, non-surgical treatment involving physiotherapy is recommended (54), while surgical interventions should only be considered as

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a last resort, as their outcome is unpredictable and might even lead to worsened instability symptoms, pain, and early degenerative changes (14, 54, 55, 56). Recently, the so-called shoulder pacemaker treatment concept has proven effective against functional PSI (15). Motion-triggered/functional neuromuscular electrical stimulation is applied during conventional physiotherapy exercises to help affected patients regain physiological muscle activation patterns and thus stable shoulders via a feedforward learning effect (Fig. 5). The shoulder pacemaker concept has been shown to provide a better outcome than conventional physiotherapy alone in a randomized trial (57). While acquired structural defects are rare and mostly minor, our recently published study revealed a flattened or convex glenoid surface in 60% of the cases (7). This individual constitutional structural deficiency may enhance this type of instability and has been identified as a risk factor associated with decreased effectiveness of non-surgical treatment (15).

Type B2 often involves young males who either sustained trauma or were subjected to repetitive microtrauma during shoulder-demanding sports activities. The athroscopic posterior labral repair can lead to a good clinical outcome and a return to sports (50) (Fig. 6), irrespective of the fixation technique with hard body or all-suture anchors (58). However, the presence of posterior glenoid bone loss >11% might require posterior glenoid bone grafting (59), and a concomitant RHSL with a gamma angle >90° (48) might warrant a McLaughlin procedure (32), transposition of the lesser tuberosity (52), or filling the defect with allograft (53) to prevent engagement. Similar to the glenoid track concept in anterior shoulder instability, bipolar bone loss in type B2 can turn a non-engaging RHSL into an engaging RHSL (60).

Static PSI (group C)

Type C1 is mostly asymptomatic in the early stages and therefore only detected as an incidental finding. The available surgical treatment options include arthroscopic posterior articular coverage and capsular shift (PACS) (Fig. 7), posterior open-wedge osteotomy, and posterior bone block procedures (61, 62, 63, 64). While all mentioned surgical techniques have been reported to improve symptoms, at least temporarily, none of the techniques was able to recenter the joint reliably, which is why the treatment of this subgroup still remains a challenge. A stepwise treatment approach starting with non-operative management, followed by arthroscopic soft tissue procedures, and subsequently bone graft interventions have been suggested in an attempt to buy time in the typically rather young patients before arthroplasty might become necessary due to progressive eccentric wear (62).

Type C2 includes mostly males who sustained major trauma leading to acquired structural damage, including RHSL and posterior glenoid bony defects, and permanent posterior subluxation of the humeral head (Fig. 8). In cases where no relevant bony damage of the joint is present, soft tissue-based surgical interventions (e.g. PACS) can be attempted. However, in the case of bony structural damage, surgical treatment should focus on restoration of the articulating surfaces combined with soft tissue balancing to achieve better humeral head centering. Joint-preserving options include bone grafting (53) and transposition of the lesser tuberosity (52) in the case of a large RHSL or glenoid bone grafting in the presence of critical posterior glenoid bone loss, as well as a combination of both (65) (Fig. 9). In cases with advanced symptomatic osteoarthritis, either anatomical or reverse shoulder arthroplasty has to be considered depending on the degree of decentering of the humeral head (66, 67).

Summary

The ABC classification for PSI is comprehensive and allows distinguishing subgroups based on etiology, thus providing a basis for treatment decisions. The diagnosis is based on the medical history and targeted clinical examination, completed by MRI to detect structural lesions. In cases of extensive bone loss, CT-based quantification of the defect should be employed. While non-operative treatment is recommended in acute subluxation (A1), functional dynamic PSI (B1), and constitutional static PSI (C1), surgical intervention might be considered in acute dislocation (A2), structural dynamic PSI (B2), and acquired static PSI (C2) based on individual patient characteristics and functional demand.

ICMJE Conflict of Interest Statement
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Author contribution statement
All authors contributed to study conception and design. The first draft of the manuscript was written by AP. DA and PM critically reviewed and edited of the manuscript before submission.

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