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Special Issue: Shoulder Instability

Guest Editor: Amol Tambe

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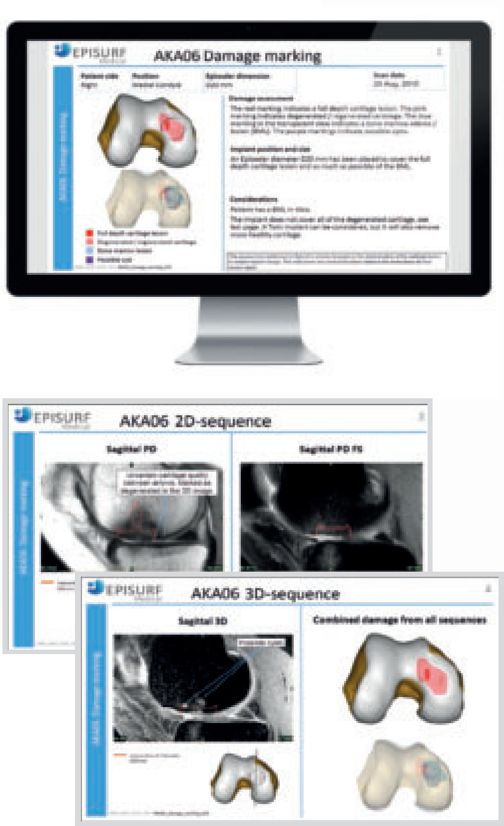

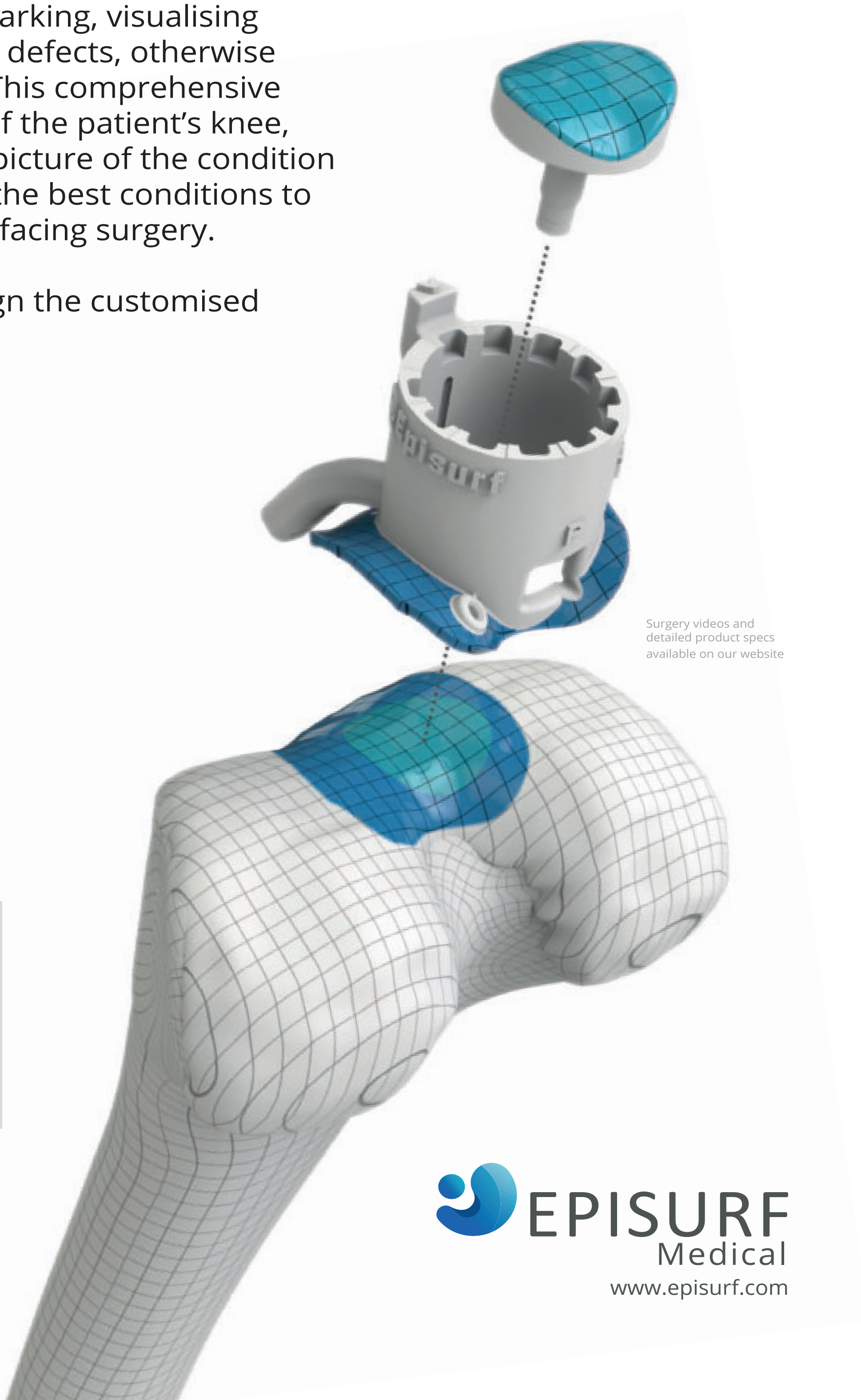

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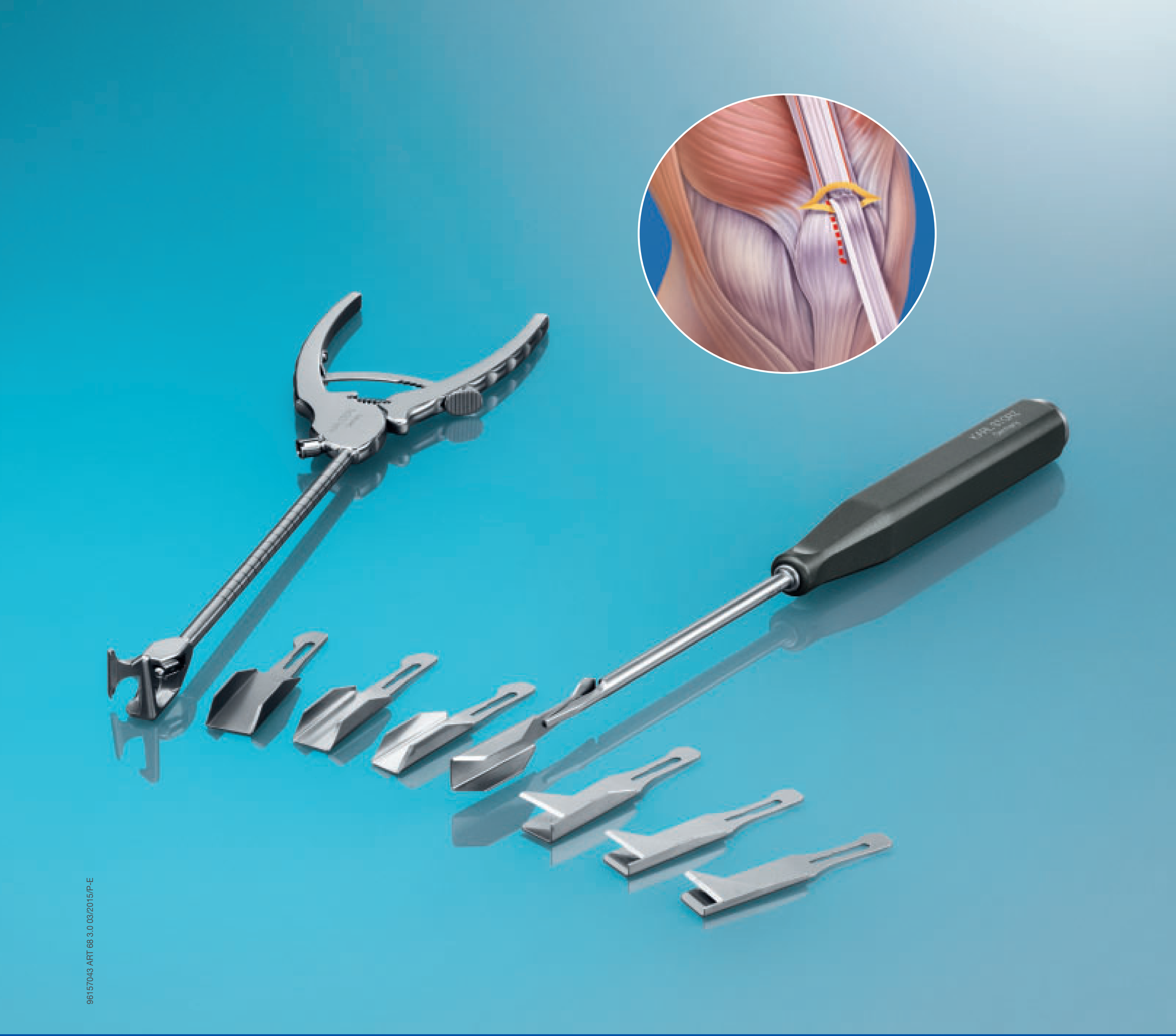

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Journal of Arthroscopy and Joint Surgery (JAJS) is committed to bring forth scientific manuscripts in the form of original research articles, current concept reviews, meta-analyses, case reports and letters to the editor. The focus of the Journal is to present wide-ranging, multi-disciplinary perspectives on the problems of the joints that are amenable with Arthroscopy and Arthroplasty. Though Arthroscopy and Arthroplasty entail surgical procedures, the Journal shall not restrict itself to these purely surgical procedures and will also encompass pharmacological, rehabilitative and physical measures that can prevent or postpone the execution of a surgical procedure. The Journal will also publish scientific research related to tissues other than joints that would ultimately have an effect on the joint function.

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Editorial

Taming the unstable shoulder – Are we there yet?

The unstable shoulder has a variety of presentations. While traumatic origin is common; the atraumatic type, habitual dislocators, and those uncommon types with abnormal neuro-proprioceptive mechanisms[1–3] which result in muscle patterning are also seen by most clinicians dealing with this problem.

One can easily get lost in the morass of nomenclature that goes with shoulder instability, we have however come a long way in defining and understanding the anatomic and structural lesions that underpin instability and the pathologic processes that propagate it.

The traditional TUBS/AMBRI[4] classification that is easy to understand but is often too simplistic a view of some complex instability situations appears to be gradually waning from use as systems that recognize the nuances and interplay between the instability types, namely the Stanmore Classification,[5] come into use.

The work done by a number of astute clinicians like Hovelius, Pascal Boileau, Giles Walch, Jo de Beer, Stephen Burkhart,[6–8] to name a few, has opened up vistas of knowledge that have improved our understanding and management of the unstable shoulder. There is increasing recognition of the large volume of instability work including complex instability that is undertaken in Asia and the Subcontinent. This work will be validated as surgeons and clinicians in the subcontinent focus not only on good quality work but also study their populations, research their methods and present their results in peer review publications.

There appears to be now a convergence of opinion amongst orthopaedic surgeons and shoulder specialists when dealing with bone loss,[6] addressing associated lesions[8,9] and understanding the risk factors for recurrence of instability.[10]

However, management of the first time dislocator, timing of surgery, approach to atraumatic instability[1,3] and approach to bipolar bony lesions[11] are some of the 'instability' related issues that still invoke differing views.

Our assessment of lesions that trigger instability has seen undoubtedly improved. The role of multi planar imaging and analysis of the glenoid bone loss with well described methods has enhanced surgical planning and eventual outcomes.[8,12–14] On the same count, newer concepts like the 'glenoid track' and the 'on track/off track' lesions[15] have not been fully embraced. The techniques to calculate these lesions remain complex and cannot be easily applied in routine clinical practice.

As a group, we are more aggressive in investigating shoulder instability. Surgical techniques have no doubt evolved to the extent that even complex procedures like arthroscopic coracoid transfer are becoming a thing of the routine. The question of how we assess our outcomes has however remained unchanged – a recurrence of dislocation is considered as 'failure'.

A number of studies, and more recently the review of Wasserstein et al., highlight this problem with recurrent dislocation. It is to be noted that the absence of repeat dislocation does not necessarily represent a good result after instability repair. Should loss of confidence when participating in sports or manual overhead work due to a feeling of insecurity in the shoulder also equate to failure despite the patient not having a frank repeat dislocation?[16,17]

The increasing volume of procedures and the ease of access afforded by arthroscopic surgery bring its own perils. We are on an 'upswing' as regards surgical management of shoulder instability. No doubt some of us will also be faced with failed instability repairs and the challenges it brings. It thus behooves the 'Shoulder Community' and senior clinicians to take on the mantle of teachers and trainers to the budding surgeons to give them the necessary insight and skills to see the bigger picture and do the best for their patients with unstable shoulders.

This special issue of has a focus on shoulder instability with well informed reviews from clinicians with expertise in this field. The idea is to cover a wider remit from assessment of instability to investigations, surgical techniques, tips and tricks along with a unique topic on sterno-clavicular joint instability management.

Approach to the contact athlete is presented and tricky topics like posterior labrum repair and the role of remplissage is addressed by experienced surgeons.

A comprehensive review tackles the issue of atraumatic instability and articles on management of failed instability repairs and locked posterior dislocation are the icing on the cake.

We hope that this treatise on shoulder instability will appraise the readers with current controversies while also reinforcing common ground. It will also serve as a guide book to the surgeon when facing some of the complexities of instability covered herein.

So . . . when talking about shoulder instability, one might ask – what's in and what's out? Without making a metaphor of it let us move towards comprehensive thinking and action that keep those tricky shoulders in. Out is definitely out!

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Review article

Diagnosing shoulder instability

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ABSTRACT

Shoulder instability can be challenging in diagnosis with a number of pathologies and causations at work. The four pillars of a structured consultation include history, examination, special tests and appropriate investigations. When findings in each of these domains are aligned then one can be confident in the diagnosis. This article focuses on the relevant points in each of these pillars to aid the practicing clinician in their diagnostic expertise.

Contents

1. Diagnosing shoulder instability

The shoulder is an inherently unstable joint, which is a natural consequence of the large degree of motion it achieves. A complex arrangement of both static and dynamic structures works in harmony to maintain its equilibrium of stability. Any disruption, malfunction or disharmony in the way these tissues act can result in shoulder instability, that can then present in clinic through a variety of manifestations- pain, weakness as well as the feeling of an unstable joint itself.

Shoulder instability can be classified through direction; anterior, posterior, multi-directional, as well as Cause. The Stanmore Classification has provided a comprehensive description of causation by taking into account both structural and non-structural causes. It gives a clear diagrammatic representation of the complex continuum and coexistence of these pathologies[1] (see Fig. 1).

Diagnosing shoulder instability is best made on a history of the problem, clinical examination, accurately performing pertinent special tests, and usually with the help of radiological investigations. The reliability of a diagnosis is high when these four key pillars are aligned, however, equally when they are not overlapping or are even divergent then confidence in the diagnosis will be lower.[2] This four pillar model of patient assessment is strongly advocated and practised in our department and as such this article will be broken down into these subcomponents.

The management of glenohumeral instability closely follows the description and definition of the type of instability (see Table 1). One needs to clearly categorise each patient into the relevant subtype prior to instigating treatment.

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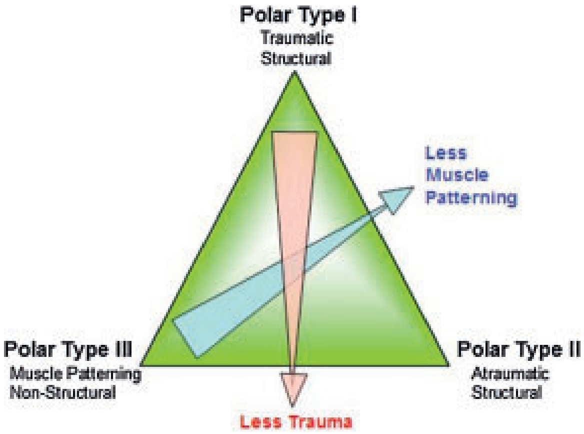

Fig. 1. The Stanmore triangle classification of instability.

Table 1
Types of Shoulder Instability.

Etiology	Traumatic	Single event
	Atraumatic	Repetitive Capsular Muscular Neurological
Direction	Anterior Posterior Global	
Pathology	Labral disruption Bony lesion Capsular pathology	
Duration	Acute Recurrent	

2. History

This should cover the direction of instability, duration and chronicity of symptoms. 96% of shoulder dislocations are associated with a traumatic episode[3] and therefore enquiry into a sentinel event, along with the nature and degree of energy involved are an important starting point. The position of the arm and direction of the force should be recorded, along with whether there was a subjective clunk or a frank dislocation that required medical reduction following confirmation by radiographs.

High-energy trauma is more commonly associated with structural changes to the joint. This is in comparison with an insidious onset ‘slipping out of joint’, which is more associated with non-structural changes. Symptom frequency during work, sport or even daily activities can inform on the severity of instability, it is also important to distinguish between subluxations experienced versus frank dislocations.

Almost half of patients do not experience instability at all and may only complain of pain or a ‘dead arm’ sensation. Associated pain with instability is important as it may indicate other injuries such as tuberosity fracture or rotator cuff tear, whilst progressive pain and stiffness following a long history of shoulder instability could herald the development of post-traumatic arthritic symptoms. It should also be noted that some young athletes can complain of pain alone, termed the “Unstable Painful Shoulder”. These patients may deny feeling unstable and complain of pain rather than apprehension in the relevant special tests.[4]

Age, occupation and any relevant leisure activities are important to document. In particular age of onset is a significant prognostic guide with an 80% risk of recurrence in patients younger than 20 years of age,[5] this can increase to 90% with a return to sport[6] or in patients involved in contact sports.[7] Conversely increasing age is protective of future dislocations with just a 16% risk of recurrence in those over 40 years of age.[8] Posterior instability can develop from the repetitive micro trauma associated with throwing, swimming or overhead racket sports. Patients may complain of a persistent ache towards the latter stages of their sport as muscles fatigue and dynamic stability is lost.[9]

Finally it is essential to enquire about other joints being dislocated, voluntary dislocation or generalised laxity. There may be systemic underlying conditions such as connective tissue disorders or other non-structural conditions that have resulted in instability.

3. Examination

The pattern of examination follows the mantra of “Look, Feel, Move” with several pertinent findings for instability patients. The initial observation of the patient may well be normal; particularly if it is a young and fit patient presenting once acute pathology has settled. However one can enquire about bruising around the shoulder at time of injury and occasionally in the modern day photographic evidence on a smart phone may be provided.

Look for wasting around the girdle, disuse, limb neglect or trophic changes secondary to CRPS. Shoulder girdle positioning and subsequent posture can also be observed, with painful or subluxed shoulders hanging downwardly rotated and depressed. This position can lead to traction on the lower portion of the brachial plexus and paraesthesia in C8 dermatomal distribution. Also patients with excessive capsular laxity tend to hang their shoulder at the limit of their hypermobile range, which can cause secondary compensatory thoracic kyphosis that can be observed. Astute observations may also show evidence of more systemic conditions such as Marfans Syndrome or evidence of Brachial Plexus injury.

Before touching the patient it is important to identify altered pain perception such as hyperalgesia, allodynia or CRPS, especially if suspecting atraumatic instability as a cause. Shoulder joint congruence, muscle contour and tone, asymmetry as well as tenderness of specific structures can all be identified by palpation.

Regarding movement, full active and passive range should be explored, with any excess or poor coordination in motion noted. Dynamic shoulder instability will often only be revealed when muscles are fatigued, therefore multiple repetitions of tasks may be required. The presence of scapula dyskinesia is a sensitive marker for muscle patterning behavious and this should be carefully noted.

Assessing Beightons score should also be routinely performed when patients present with instability.[10] This is helpful to gauge as well as give a clear record of global hyperlaxity.

4. Special tests

By this stage of a consultation the health care professional will often be testing their theory as to the type of instability the patient is experiencing. A wide variety of special tests have been described for each form of shoulder instability and here we describe those commonly used.

4.1. Anterior instability

An anterior apprehension test is performed whilst standing behind the patient. With the shoulder abducted to 90 degrees, the joint is then passively moved into maximal external rotation with an anteriorly directed force also applied to the posterior humeral head. A mirror is useful in this test to both observe apprehension in the patients face, as well as feeling reluctance in the muscles, particularly at the front of the joint with pectoralis major contraction[11] The Apprehension test can be combined with a relocation test, this involves placing a hand anterior to the humeral head which provides a posteriorly directed force. This should relieve apprehension and pain and may allow further external rotataion.[12]

The load and shift test is Hawkins modification of the anterior drawer test.[13] The patient should be seated and relaxed with the shoulder in 0–20° of forward elevation and 0–30° of external rotation. One hand is used to stabilise the scapula, the other holds the upper arm and loads the humeral head into the glenoid whilst attempting to translate the shoulder anteriorly. Increasing the abduction from 0 to 60°, to 60–80° and finally 90° will test the glenohumeral ligaments from superior to inferior respectively. The degree of translation felt can also be graded from 1 to 3 depending on its severity[14].

4.2. Posterior instability

The posterior apprehension test is performed best usually with the patient seated. One hand is used to stabilise the scapula with a thumb over the spine and index and middle fingers on the coracoid. The other hand creates a posteriorly directed force at the elbow with the shoulder internally rotated, flexed and abducted to 90°. Pain and apprehension represent a positive test.[13]

The Wrightington posterior instability test was developed to be particularly helpful in muscular contact athletes. This group of patients often complain more of pain and clicking posteriorly rather than instability itself. These patients have excessive posterior laxity and with translation, posterior joint pain may be experienced. The test involves a similar position to that described by O'Brien- the arm is placed in full adduction and internal rotation at 90° of flexion. A positive result is marked weakness and posterior pain on resisted forward flexion.[15]

4.3. Atraumatic instability

There is no singular test that diagnoses atraumatic instability itself. The above tests, if all positive, will often raise suspicion for global shoulder instability rather than specific anatomy disrupted. Beightons score will commonly be high and other signs of shoulder joint hyperlaxity should be sought. The sulcus sign involves passively pulling the arm downwards from the elbow whilst stabilising the scapula with the other hand. A positive result is shown if a significant step off from acromium to humeral head is seen. The Gagey sign asseses laxity in the inferior glenohumeral ligaments. Passively abducting the shoulder with one hand with the other placed over the acromium stabilising the scapula. A normal shoulder will usually abduct to 90 degrees with a positive sign seen if an angle of greater than 105 degrees is seen.

Finally Kibler's corkscrew test is helpful both for the professional as well as demonstrating for the patient poor core muscle stability.[16] The patient is asked to perform a single leg squat on the contralateral leg to the affected shoulder. A positive test is when the upper body "corkscrews" with rotation seen through the hip and knee.

5. Investigations

Plain radiographs are the first line investigation and are particularly useful if there has been a traumatic dislocation. The routine AP, axillary and scapular "Y" views should be analysed to ensure concentric reduction as well as looking for bony defects to the glenoid (bony Bankart lesions) and humeral head (standard or Reverse Hill Sachs). Further modified views can be useful to assess for these defects. The West Point view is a modified axillary image and is used to assess the anteroinferior glenoid for bony Bankart lesions.[17] The Stryker notch view is used to demonstrate the posterior humeral head for Hill Sachs lesions.[18] In current practive CT scans have substituted the role of such specialised views and can be used to give further information on the size and displacement of any bone fragments with 3D reformatted images particularly helpful. Clearly detailed assessment of bony injury can help to predict the likelihood of further instability as well as inform on the most appropriate treatment strategy.

Magnetic Resonance imaging enhanced with intra-articular gadolinium is the gold standard modality for assessing the myriad of intra-articular soft tissue injuries that are associated with shoulder instability. It has also be shown that positioning the arm in the apprehension position (abduction and external rotation) during the imaging can increase the sensitivity of the imaging for anterior labral pathology from 48 to 89%.[19] This form of imaging will also pick up damage to the biceps or rotator cuff that can be a source of ongoing pain and dysfunction in patients older than 40 years following traumatic dislocation.[20]

6. Conclusion

Assessing shoulder instability is best achieved through accurate application of each of the four pillars as detailed above. A detailed history with meticulous examination, appropriate special tests and supported by the correct imaging will reliably provide the diagnosis. While following the strategy set out above, not only will the type of shoulder instability be revealed but also significant information gained as to the likelihood of further episodes.

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Conflict of interest

All authors have seen and approved the final version of the manuscript being submitted. They warrant that the article is the authors' original work, hasn't received prior publication and isn't under consideration for publication elsewhere.

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Imaging in shoulder instability with focus on identifying and measuring bone loss: A narrative review

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ABSTRACT

Bone loss is a key burning issue in shoulder instability, mainly because its presence or the lack of it can significantly swing the surgical pendulum from an open bony augmentation to a standard arthroscopic soft tissue repair, respectively. Each of these surgeries has its own separate technical challenges and a separate recovery protocol hence, it behoves upon the surgeon to be able to precisely calculate the amount of bone loss pre-operatively, to assist in clinical decision making. We review the recent literature studying the commonly used imaging methods for calculating bone loss in shoulder instability, to enable the reader in integrating these concepts in their practice.

Contents

1. Introduction

The shoulder is the most commonly dislocated large joint of the human body, with an estimated incidence of 1–2% of the population and the majority of these, to the tune of 90–95%, are in the anterior direction.[1] Glenohumeral instability (GHI) is associated with a recurrence rate ranging from 30 to 90%.[2–4] Glenoid bone loss occurs in up to 90% of patients with recurrent GHI.[5] It is generally agreed upon that an anterior glenoid bone width loss in excess of 25% is a marker for poor results with just a soft tissue procedure.[6–12] It is important to note that the threshold for glenoid width loss and surface area loss as measured by the best fit circle are different, the latter amounting to 20%.[13] The Hill-Sachs lesion, first reported by Hill and Sachs in 1940,[14] is a depression created in the soft bone of the posterolateral aspect of the humeral head, when it collides against the hard anterior glenoid cortical rim.[15] The incidence of Hill-Sachs lesions has been reported to be as high as 93% in patients with recurrent GHI.[16] Bipolar lesions, involving both the anterior glenoid and the humeral head, occur in upto 62% of anterior GHI patients.[17]

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The less common posterior dislocation of the shoulder also presents with it's own unique spectrum of bone loss, the posterior glenoid bone loss and bone loss in the antero-superior portion of the humeral head, the Reverse Hill-Sachs Lesion (RHSL). RHSL's are also called Malgaigne lesions as they were first described by the 19th century French surgeon Joseph-Francois Malgaigne.[18] Bipolar bony defects are also described for posterior shoulder instability, with a reported incidence of 20-30%.[18,19]

2. Current popular methods of measurement

CT with 3D reconstruction is emerging as the method of choice for the pre-operative assessment of bone loss in a shoulder instability scenario. The commonly used methods for calculating glenoid bone loss are:

1) Calculating the percentage of glenoid width loss on a 2D view of the glenoid, known as the Griffiths index.[20]
2) Calculating the percentage of glenoid surface area loss on a 3D CT en face view of the glenoid using data from the opposite or ipsilateral glenoid, known as the Pico method.[21]
3) Calculating the percentage of glenoid width loss on a 3D CT en face view of the glenoid by drawing a circle on the inferior glenoid, known as the Sugaya method.[5]

The Sugaya method requires the presence of an intact posterior and inferior rim of the glenoid[5] and is thus prone to error compared to the Pico method that takes the size of the contralateral glenoid into account. In a shoulder CT scan, both shoulders are simultaneously irradiated and it is just a matter of requesting the radiologist to acquire data from the opposite shoulder for these measurements. The guidelines for measurements and the critical thresholds, for the Hill-Sachs lesions and RHSL's are less clear.[13]

Surgeons sometimes like to base their decisions on measurements obtained intra-operatively during arthroscopy. It is important to note that arthroscopic measurement of bone loss is prone to error.[13] One study estimated that the defect size measured arthroscopically, overestimated the actual size measured on a 3D CT by a whopping 55%.[22] Arthroscopic intraoperative measurements rely on the bare spot technique. The bare spot may not always be in the centre of the glenoid, in most instances it lies closer to the anterior edge, leading to overestimation of the defect size.[23–25] The spot may not actually be a discrete spot, but an area ranging in size from 2.4 to 9 mm, making centre point estimation difficult.[26]

3. Other emerging methods

The curvature of the glenoid has been studied as a causative factor for recurrent anterior instability. Unstable shoulders were found to have flatter glenoid profile than controls, in the anteroposterior and superoinferior directions as measured on a 3D CT reformatted image.[27] Glenoid version, especially excess retroversion has been associated with an increased incidence of posterior instability.[28,29] Excess retroversion has also been linked with contralateral shoulder posterior instability.[29]

4. X-ray

Plain radiography is the most popular and readily available modality of initial investigation for any orthopaedic condition. Standard X-ray views (true AP and axillary) were found to have lower accuracy and reliability in calculating glenoid bone loss.[10,30–32] Plain X-rays are useful as a good screening tool for suspecting bone loss, both at the glenoid and humeral ends.[13]

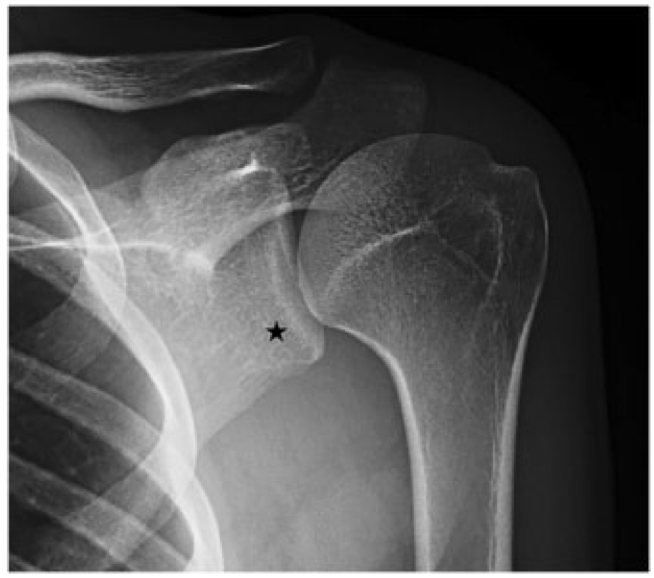

Fig. 1. Normal true AP view of the left shoulder of a recurrent anterior instability patient. Black asterix marks the normal double contour sclerotic line seen in the inferior portion of the glenoid in a situation where there is no bone loss.

4.1. Glenoid

In a true AP view, also known as the Grashey view (Fig. 1), loss of the sclerotic line of the glenoid, for more than 5 mm from the inferior glenoid edge (Fig. 2), has good predictive value in detecting significant anterior glenoid bone loss.[32] This was found to be independent of lesions of the posterior glenoid rim.[32] Specialised views like the Bernageau profile view had better accuracy and reliability scores in detecting and calculating glenoid bone loss.[13,33,34]

The true AP view is taken by positioning the patient's thorax at an angle of 35–45 degrees from the coronal plane, keeping the arm

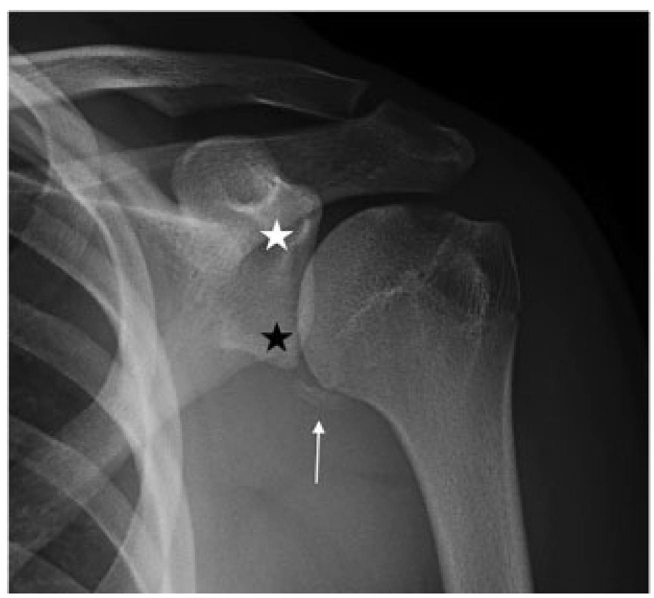

Fig. 2. True AP view showing loss of double cortical line at the glenoid of the left shoulder in a recurrent anterior instability patient. The black asterix marks the loss of the double contour of the normally sclerotic anteroinferior rim of the glenoid. The white asterix denotes the beginning of the normal cortical outline in the anterosuperior cortex. The white arrow points to a free floating bone piece in the axillary recess.

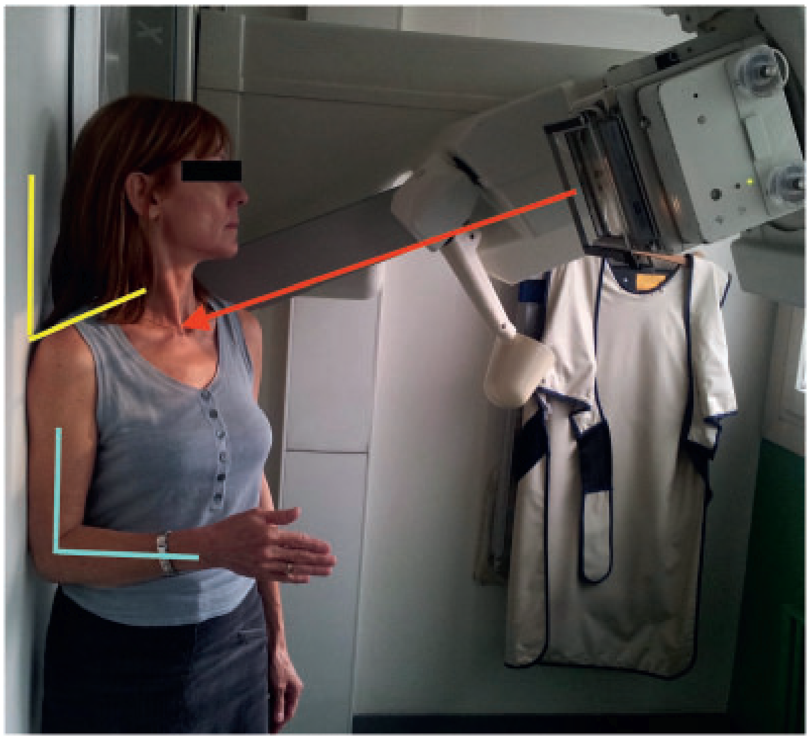

Fig. 3. Patient positioning for a true AP view of the right shoulder. The yellow angle denotes that the chest should be at an angle between 30-45° with respect to the X ray cassette kept at the back, with the left shoulder facing forward. The blue angle denotes the neutral hand positioning, with the forearm at an angle of 45°. The red arrow denotes a downward direction of the X-ray beam around 15–20°.

Fig. 4. The glenoid as seen in a Bernageau view of the left shoulder in a recurrent instability patient. The black asterix points to a large Hill-Sachs lesion. Note the truncation of the anterior glenoid cortex marked by the white asterix.

in neutral rotation with the X-ray beam and cassette perpendicular to the torso (Fig. 3).[35] The Bernageau profile view (Fig. 4) is a little difficult to obtain and standardise. Some authors have described it with the patient standing, flexing the arm 160° and maintaining a 70° angle with the X-ray cassette using a specially designed pillow, with the X-ray beam coming down at a craniocaudal angle of 30 degrees.[36] Another way of obtaining the Bernageau view is with the patient lying comfortably on his axilla, supporting his head in his hand (Fig. 5a and b). The X-ray cassette is placed below the axilla and the beam comes from the top, angled 15–20° from the vertical.[37] If bone loss is evident in either of these views, it is prudent to order a CT scan to confirm and measure it.

4.2. Humeral head

Internal rotation views are helpful to identify Hill-Sachs lesions.[13] Specialised views like the Stryker notch view can delineate the size and orientation of the Hill-Sachs lesions.[38] Hill-Sachs quotient is one method of quantifying this lesion, calculated by multiplying the depth and width of the lesion on an AP radiograph with the arm in 60° internal rotation and length as obtained on the Bernageau view.[39] Another popular and well validated measure is the ratio of the defect depth and humeral head radius (d/R) on a true AP view with the arm in internal rotation.[40]

4.3. Future trends

Stress radiography of the shoulder has been explored as a possible method for diagnosing anterior instability.[41] It has been reported that the normal cut off point for anterior humeral displacement is <3 mm in adults.[45] The Telos GA-IIE device with a special shoulder attachment was used to do the stress testing with the arm in neutral and 60° external rotation.[41] The exact force

Fig. 5. (a) Positioning for the modified Bernageau view. The patient is lying relaxed on his axilla. Note the 15–20° craniocaudal angle of the X-ray beam. (b) View from the head end, patient positioned for the modified Bernageau view. Note the angle of inclination from the vertical marked by the arrows. This angle may vary from 5 to 25 degrees depending on patient build to keep the opposite scapula from overlapping onto the X-ray.

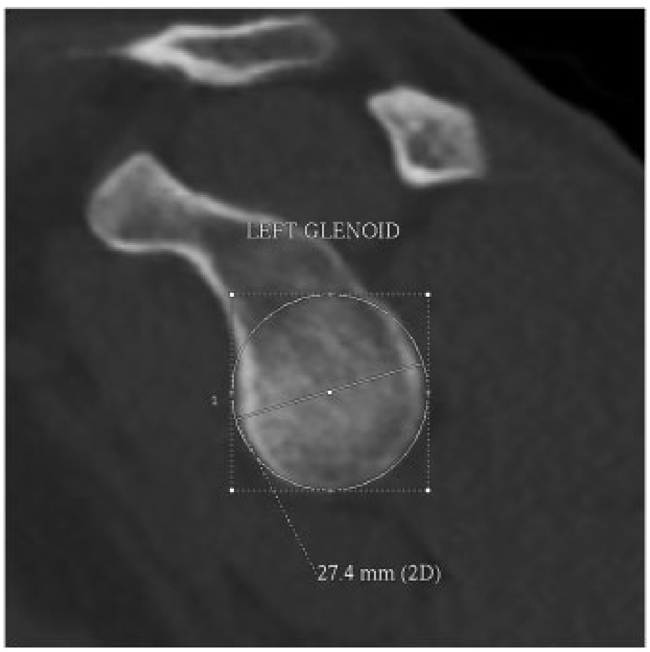

Fig. 6. Using the normal shoulder as a reference to measure glenoid bone loss. A circle is outlined over the inferior glenoid using the posterior and inferior cortices as the reference arc.

required to perform the stress test has been under question, the previously employed force of 15 daN (decanewton) was found to be inadequate.[41] Significant correlation was noted for anterior instability and anterior humeral head displacement exceeding 3 mm with the arm in zero degrees external rotation.[41]

5. CT

The Pico Method (using 2D and 3D CT) and Sugaya method (using volume rendered images) involve calculating the area of a circle centred on the inferior glenoid or the percentage loss in depth on a volume rendered image respectively. Normal contralateral glenoid is used as a reference based on the intact 3–9 o'clock margins, for the Pico method (Fig. 6).[21] This circle is superimposed onto the injured ipsilateral glenoid, and software based manual trace of the glenoid defect is used to calculate surface area bone loss (Fig. 7). Sugaya method uses the affected ipsilateral glenoid, to determine the pre-injury margins, provided the 6 o'clock–9 o'clock postero-inferior margin of the injured glenoid is intact, otherwise the results would be inaccurate (Fig. 8). Also the fractured osseous Bankart fragment can be manually traced and its surface area added to that of the glenoid for accurate assessment, as per this method.[5]

Essentially today three dimensional CT scan (3D CT) with volume rendering is used as a gold standard for preoperative imaging of an unstable shoulder. En face images of the glenoid cup are obtained, a line is drawn through the long axis of the glenoid (using oblique reformatted images) and a best fit circle is superimposed upon the widest part of the glenoid cup, assuming that the posterior and inferior aspect of the glenoid traces a curved arc. Surface area can be easily measured using on the DICOM console or using freely available proprietary computer software like the Image J.[42] Also the diameter or width of glenoid is measured, perpendicular to the glenoid long axis, 3–9 o clock position. CT offers the advantage of acquiring information of both shoulders, hence the normal contralateral shoulder glenoid width (D) is calculated, and the pathological ipsilateral glenoid width subtracted from it to obtain their difference (d).

The glenoid track concept has changed the way we now think about bone loss in shoulder instability. Glenoid track, simply put, is the contact zone between the humeral head and the glenoid cup, or more accurately, the supero-lateral portion of the posterior humeral head that impacts the inferomedial margin of the anterior glenoid in the position of athletic function and this is no more than 83% of the maximum glenoid width. In other words glenoid track is $0.83 \times$ maximum glenoid width.[43]

Imaging wise, and as discussed above, we acquire the width of normal contralateral glenoid (D) (Fig. 9a), calculate difference (d)

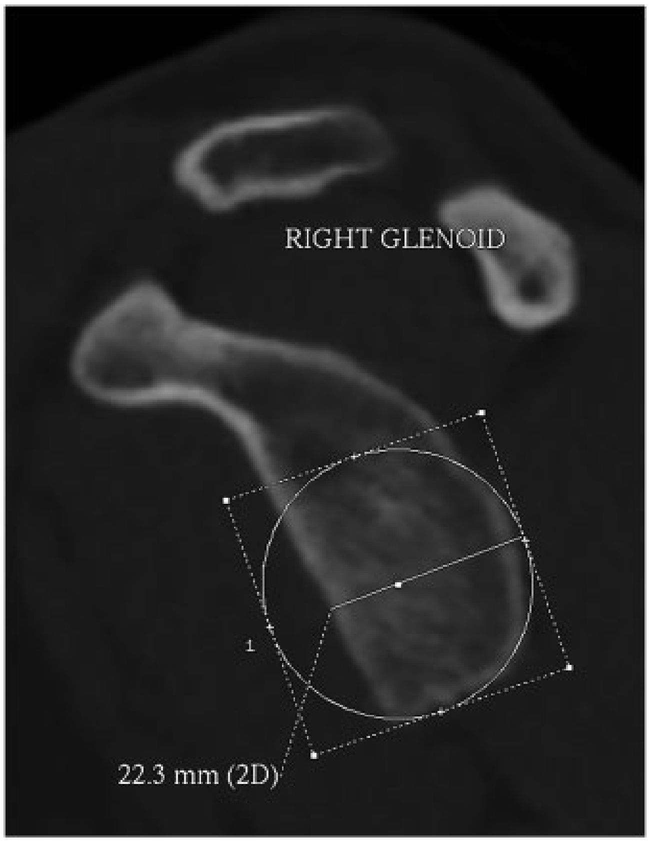

Fig. 7. Circle based on contralateral glenoid measurement is drawn on the affected glenoid. The percentage surface area bone loss in this case was calculated as 18.7% using Image *J* as per the Pico method. The percentage diameter based bone loss measurement in this case was 18.6% using Image *J* as per the Griffith index.

(Fig. 9b) which is the bone deficiency width, and calculate the proposed glenoid track for the ipsilateral deficient glenoid as (0.83 × D)- d. Percentage of bone loss is d/D × 100. A parallel calculation of Hill Sachs Index (HSI) is made by using volume rendered images, the widest width of the humeral head defect (HS) is measured including the bone bridge (BB) of the rotator cuff (HSI = HS + BB) (Fig. 10).[43]

If the HSI is wider than the measured glenoid track, the lesion is engaging or off-track, meaning that the Hill-Sachs lesion will contact the anterior glenoid rim in the maximally abducted and externally rotated position even after arthroscopic/ open soft tissue fixation. It is noteworthy to mention that the length of the coracoid process can be calculated by CT from its base (fat stripe carrying the attachment of coraco-clavicular ligament) to its tip, so that the surgeon may have a pre-operative idea of the coracoid dimensions, in case a bony augmentation is planned using the coracoid (Fig. 11).

6. Role of magnetic resonance imaging (MRI)

The MRI is a more commonly used modality of imaging in any given musculoskeletal scenario and is an excellent modality to image the soft tissue lesions in shoulder instability like the Bankart lesion and its multiple variants (Fig. 12). The soft tissue lesions, however do not impact management strategies as much as the bony lesions do and are pretty much standardised and straight forward. They can also be accurately assessed intra-operatively and only require arthroscopic/open refixation.

Using multiplanar 3D acquisition sequences, MRI can measure the glenoid bone loss, percentage, and glenoid track, using ipsilateral glenoid with best fit circle extrapolation of the expected normal glenoid width (D). MRI however suffers from its largest disadvantage, lack of volume rendering to reliably assess the Hill Sachs index, and hence is an inadequate study for complete pre-operative imaging.

Fig. 8. Glenoid bone loss calculation by the Sugaya method. In this example using the Image *J* software, bone loss was calculated as being 24.3%.

7. Discussion

Recently, the discussion points in recurrent shoulder instability have swung from the soft tissue to the bone. The soft tissue findings in recurrent instability are well known and do not impact management strategies as much as the bony findings do. In particular loss of bone, especially from the glenoid, has generated a lot of clinical interest in deciding the best management strategy for the instability patient. Humeral bone loss is less well defined and one area of recent interest is the complex interplay between the glenoid and humeral bone defects, the so called bipolar bone loss. Currently, arthroscopic Bankart repair using modern suture anchor techniques has failure rates ranging from 4 to 17%.[46] Burkhart et al. concluded that significant glenoid bone loss, approximately 25–45% of glenoid width loss, was associated with higher failure rates of arthroscopic Bankart repairs.[6]

Plain radiography has a good screening value for glenoid bone loss.[13] For the quantification of glenoid bone loss, the Bernageau view emerged as the clear choice in terms of accuracy and reproducibility.[40] Ikemoto et al. described a reliable method of measuring anterior and posterior glenoid rim distance and comparing it to the contralateral shoulder, and concluded that this Xray view showed accuracy comparable with CT in evaluation of degree of bone erosion.[33] Itoi et al. compared x-rays (West point and axillary views) and 2DCT (width of inferior fourths of glenoid rim measured in a single axial plane) to assess reliability and concluded that CT was superior.[10] For the Hill-Sachs lesions, plain radiography is less clear in terms of assessment.[13] The most clinically relevant technique was that of Sommaire et al. who calculated the depth/ radius ratio. This was found to be reliable and reproducible.[34] Overall X-rays are much cheaper, widely available,

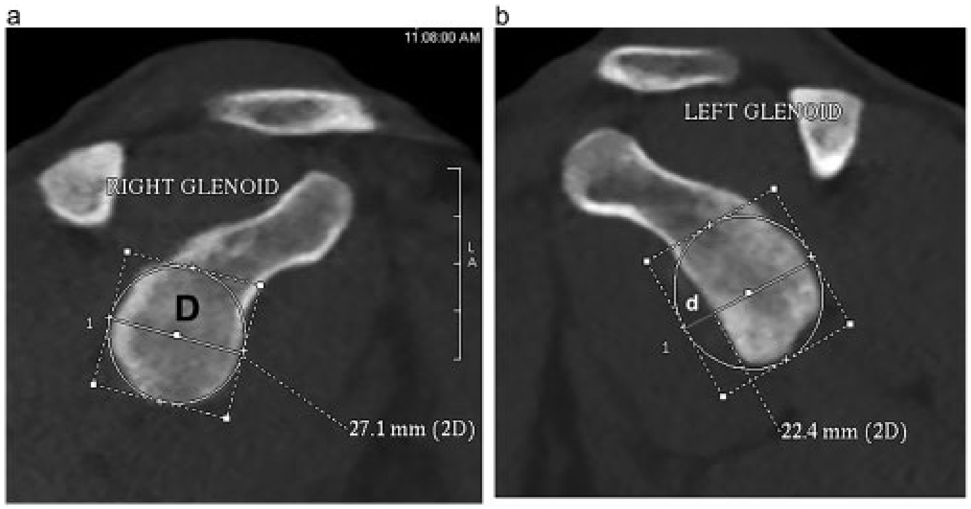

Fig. 9. (a) Multiplanar reformatted image (MPR) in sagittal oblique plane, shows the best fit circle method of glenoid width calculation. The diameter of the circle is calculated (D) at the normal glenoid. (b) The pathological left glenoid width (22.4 mm) was subtracted from normal right glenoid (D = 27.1 mm) to obtain a difference of d = 4.7. The bone loss is d/D x 100 = 17.3% and glenoid track is 17.7 mm (D x 0.83 -d).

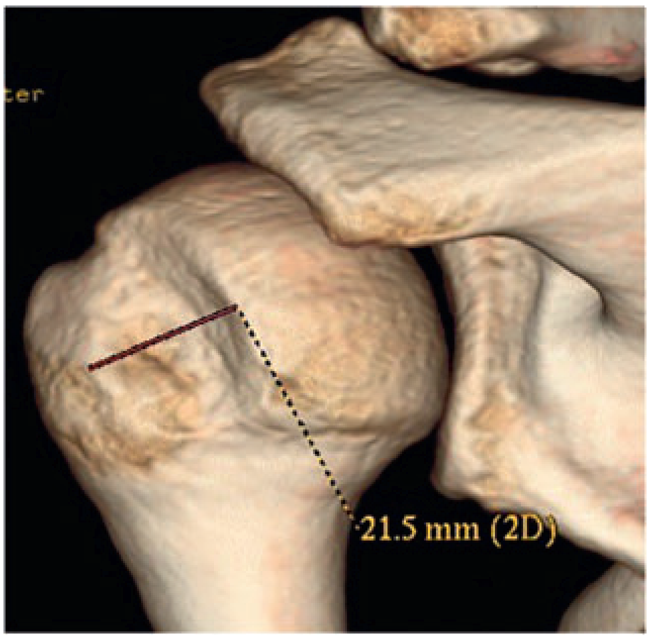

Fig. 10. In the same patient as shown in Fig. 9 (a) and (b), the Hill-Sach index (HSI) on a 3D volume rendered image measures 21.5 mm, which is greater than the calculated glenoid track (17.7 mm) and is thus likely to engage with the anterior glenoid rim even after a successful soft tissue repair.

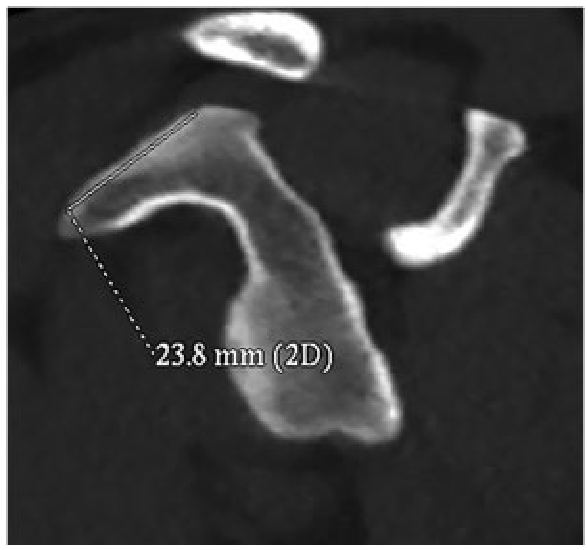

Fig. 11. Measuring the length of the coracoid on the CT.

but suffer from disadvantages such as inconsistent performance, and difficult positioning in patients with shoulder pain or dislocation, leading to suboptimal interpretation.

Computed tomography with 3 dimensional acquisition and volume rendering capabilities is now the widely accepted imaging modality to evaluate bone loss, especially the bipolar variant. CT scan offers various advantages like rapid acquisition, excellent bony resolution, comparison with contralateral shoulder and is more acceptable to claustrophobic patients with reduced metal artefacts in post-operative patients. Volume rendered CT with humeral head subtraction, clearly exhibits the glenoid cup in end-on view as pear shaped morphology with curved anteroposterior and inferior margins. Measurements of glenoid bone loss revolves around either the width method or surface area method. Several authors have found both methods to be reliable and clinically reproducible.[5,44]

Griffith et al. were the first to introduce glenoid width loss technique as an accurate method comparing both shoulders and introduced the Griffith index.[20] Chuang et al. introduced the Glenoid index, which was the ratio of injured glenoid width/ normal glenoid width, calculated perpendicular to the long axis of glenoid on 3D CT. This index was 96% accurate in predicting patients who had significant bone loss and needed a Latarjet procedure.[47] It is important to note that the threshold for critical bone loss as calculated by the width methods like Griffith index and the area methods like the 'best fit circle method' are different.

Itoi et al. introduced a paradigm shift with their concept of bipolar bone loss in anterior instability. This concept centres on the engagement of the hill sach's lesion with the anterior glenoid rim in the maximally abducted and externally rotated position; the so called "sports position". Di Giacomo et al. published several useful measurements based on a new concept called the glenoid track, that helps predict humeral head engagement in recurrent anterior instability.[46] The term "Glenoid track" was first introduced by

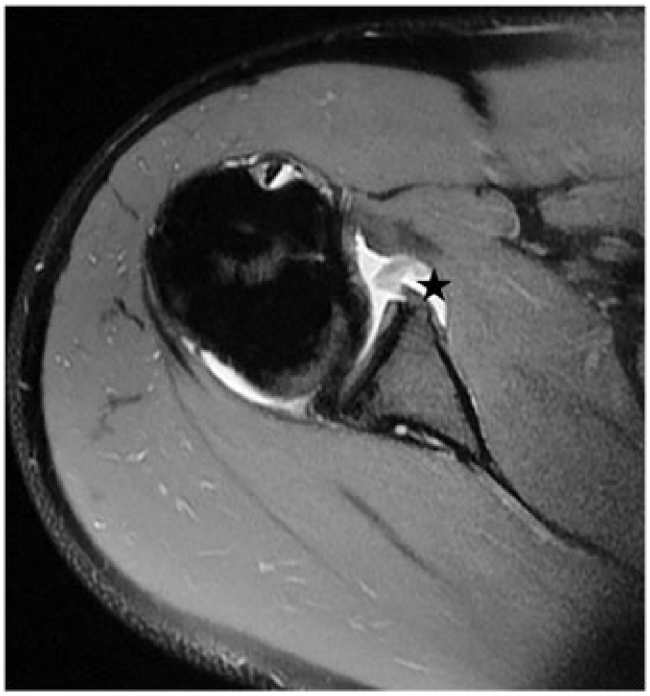

Fig. 12. Axial proton density fat saturation image showing a classic non-osseous Bankart lesion. The asterix points to a combination of full thickness labrochondral tear through the anteroinferior labrum with periosteal stripping, and no distinct bone fragment. Note the attritional bone loss and truncation of the osseous glenoid rim in this patient of recurrent dislocation (more than 40 episodes).

Yamamoto et al.[48] It refers to the width of glenoid articular surface covering the humeral head in the maximally abducted, externally rotated position. In this position the glenoid pushes the cuff by 16% of its width, leaving the remaining 84% (the glenoid track) covering the articular surface of the humeral head.[47] In glenoids with bone loss the width of the glenoid track is less, thereby making the hill-sach lesion more likely to engage with the anterior glenoid rim, especially if it is placed medially (off track).[46,47] This has helped generate specialised treatment algorithms for managing gleno-humeral instability after factoring in bone loss from both ends.[46]

Moroder et al. made similar observations in posterior instability, wherein non critical reverse Hill-Sachs lesions were converted to critical engaging ones (based on gamma angle measurements) in the presence of a posterior glenoid bone defect.[49] The same set of authors in a different paper identified three different types of reverse hill-sach lesions, each with a different potential for engaging with the posterior glenoid rim in a bipolar bone loss scenario.[18] This brings to light the importance of looking at glenoid and humeral bone defects in unision rather than separately. The role of curved multiplanar CT is emerging due to the inherent concave curved surface of the glenoid that makes the area measurements by the Pico and Sugaya method inaccurate.[13]

MRI is a very popular imaging modality for shoulder instability. Apart from being a non ionising radiation modality, it offers the advantage of excellent examination of osseous, labral, capsuloligamentous and myotendinous structures, with multiplanar capabilities. It suffers from certain disadvantages like claustrophobia, longer acquisition time, breathing artefacts, lack of volume rendering and contralateral evaluation. The jury is still out on its usefulness in calculating bone loss both at the glenoid and humeral ends. Gyftopoulos et al. in a cadaveric study, demonstrated that the best fit circle width method, based on ipsilateral glenoid, had equal concordance correlation coefficient (CCC) using 2DCT, 3DCT and MRI.[44] Tian et al. in their prospective cohort study derived similar results, they used MRI acquisition methods like VIBE (volume interpolated breath hold examination) to attain better resolution.[50] In comparative studies however, CT was found to be superior to MRI in assessment of bone loss.[51]

8. Conclusions

1) Plain radiography is useful as a screening tool for suspecting bone loss. A true AP view and the modified Bernageau view are the two best views for detecting bone loss. An attempt can be made along published guidelines to calculate glenoid and humeral bone loss from the modified Bernageau view[33,34]
2) CT scan with it's 3D volume rendering capacities remains the modality of choice for detecting and measuring glenoid and humeral bone loss. Free proprietary software like the Image J^{TM} simplify these measurements. It is highly recommended to look at these defects in unison as per the glenoid track concept both for anterior and the less common posterior instability to understand best management guidelines.
3) MRI sequences are evolving to help measure bone loss, especially for the Hill-Sachs lesion, its biggest limitation so far. Till the time this becomes standardised and reproducible, CT shall remain the imaging modality of choice from the perspective of measuring bone loss.

Conflict of interest

None.

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Review article

Diagnosis and management of atraumatic shoulder instability

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ABSTRACT

Shoulder dislocation is usually as a result of trauma, although some individuals experience episodes of instability in the absence of injury. In this paper we highlight the classification of shoulder instability and describe clinical assessment before discussing the evidence behind managing this often complex problem both from a conservative and surgical perspective.

Contents

1. Introduction

The shoulder is the most frequently dislocated joint in the human body and whilst most occurrences are due to trauma, a subset of patients experience recurrent episodes of subluxation or dislocation in the absence of injury. The epidemiology of atraumatic instability is

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not well documented but estimates are in the region of 5% compared to 95% following trauma.[1] In this paper we describe the classification of shoulder instability and methods of clinical assessment before discussing the rationale behind treatment strategies and the evidence for different methods of treatment.

2. Classification

Shoulder instability can be classified in different ways and this helps to guide the clinician towards the most suitable treatment approach.[2] Two common methods of are the TUBS & AMBRI classification and the Stanmore Triangle classification.

2.1. The TUBS & AMBRI classification

First described by Thomas & Matsen in 1989, this method simply divides patients into two groups: those with a history of trauma resulting in a structural lesion of the glenohumeral joint, and those without a traumatic onset.[3] The acronym TUBS stands for Traumatic, Unidirectional, Bankart, Surgery describing the traumatic nature of onset with instability in a single direction resulting usually in a Bankart lesion (tear of the capsule & labrum complex)[4,5] that requires surgical repair. AMBRI stands for Atraumatic, Multi-directional, Bilateral, Rehabilitation and Inferior describing the absence of injury, instability in more than one direction that can apply to both shoulder and usually is managed with rehabilitation before considering an inferior capsular shift procedure if the patient fails to improve.

2.2. The Stanmore Triangle classification

Named after the Royal National Orthopaedic Hospital in Stanmore, London, UK, and sometimes referred to the Bayley's Triangle after the senior author Professor Ian Bayley,[2] this classification system is more detailed than Thomas & Matsen's. This method recognises that patients do not always fit into the two distinct categories of TUBS & AMBRI. Take, for example, a patient who has sustained trauma but has no structural lesion on imaging or arthroscopy. Conversely there may be patients who have no history of trauma but may have developed a structural lesion due to repeated microtrauma. The Stanmore classification (see Fig. 1) adds a third category of patients without a history of significant trauma but with a structural lesion of the glenohumeral joint. It allows for distinct cases that fit one of these three polar groups and also a sub-classification between the poles in cases that are less clear-cut. The characteristics of the polar groups are shown in Table 1. Type 1 corresponds to the TUBS group. Types 2 and 3 having the common components of underlying capsular laxity without trauma but type 2 has structural pathology of the glenohumeral joint whereas type 3 does not. Using the example of the patient who described significant trauma but no structural injury on imaging or arthroscopy this person would be sub-categorised as type 3(1). The authors' recommendation for treatment is guided by the presence of structural instability defects which are more likely to require surgical repair however if the patient displays additional abnormal muscle-patterning then this should be addressed first with rehabilitation. Those patients with no structural defect and abnormal muscle-patterning are advised to be managed non-operatively with rehabilitation. The difficulty however with this approach is the diagnosis of abnormal muscle-patterning which is difficult to assess clinically with poor specificity of just 11% compared to EMG testing.[6]

2.3. Multidirectional instability

In clinical practice and in the literature the term 'Multi-directional Instability' or MDI is often incorrectly used synonymously to describe atraumatic instability.[7–12] True MDI has a combination of anterior, posterior and inferior instability[2] where instability is defined as a symptom such as subluxation rather than a clinical assessment of laxity.[12] Patients may therefore be over-diagnosed as having MDI when in fact their symptoms are of atraumatic instability in a single direction: either antero-inferior or postero-inferior.

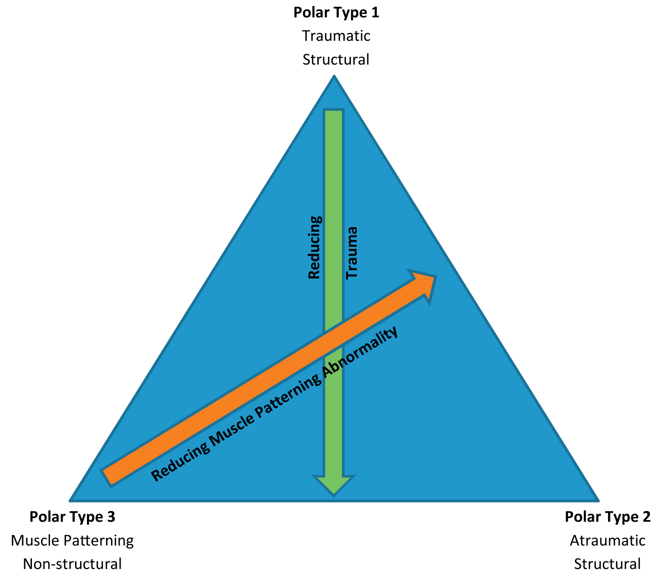

Fig. 1. The Stanmore Triangle. Reproduced and updated with permission of Mr Angus Lewis. Reprinted from Current Orthopaedics, Vol 18, Angus Lewis, T. Kitamura, J.I.L. Bayley, The classification of shoulder instability: new light through old windows!, Pages 97–108, Copyright 2004, with permission from Elsevier.

3. Clinical assessment

3.1. Subjective assessment

A thorough history is vital when establishing the classification of shoulder instability. The presence or absence of a significant traumatic event is key to determining the likelihood of structural pathology and the subsequent treatment pathway. Patients should be questioned regarding shoulder subluxation, either voluntary or involuntary, during childhood and whether there are other symptomatic joints such as clicking hips or patello-femoral joint instability to establish a possible background of joint laxity.

Age is also an important factor. Typically patients with atraumatic instability begin to experience symptoms in their teens or early 20 s rather than later in life.[2]

The characteristics of the symptoms including the direction(s) of instability, frequency of episodes and ease of dislocation are useful to guide treatment planning and aid prognosis. Those patients with infrequent episodes that only occur with the shoulder in extreme positions of their normal range of movement or with powerful activities such as throwing are likely to respond faster and more effectively to intervention than those patients whose shoulders sublux repeatedly every day with simple shoulder movements such as flexion to 90° or whilst they are just sitting still.

Occupation or sports may be an important factor in the case of patients that acquire laxity due to the repeated microtrauma of fast or powerful movements at the extremes of range, such as with throwing sports.

3.2. Objective assessment

This should begin with exposure of the limb and observation of the overall appearance to assess for deformity such as muscle

Table 1
Explaining the features of the different polar types in the Stanmore Classification. Updated with permission of Mr Angus Lewis. Reprinted from Current Orthopaedics, Vol 18, Angus Lewis, T. Kitamura, J.I.L. Bayley, The classification of shoulder instability: new light through old windows!, Pages 97–108, Copyright 2004, with permission from Elsevier.

	Polar Type 1	Polar Type 2	Polar Type 3
Trauma	Yes	No	No
Articular surface damage	Yes	Yes	No
Capsular problem	Bankart lesion	Dysfunctional	Dysfunctional
Laxity	Unilateral	Unilateral or bilateral	Often bilateral
Muscle patterning	Normal	Normal	Abnormal

wasting, subluxation at rest or winging of the scapula. Further observations can be made during movements of the shoulder and may highlight repeated subluxation or dynamic scapula winging.

Active range of shoulder movement should be assessed into flexion, abduction, internal and external rotation. Some patients may have limited range, typically in overhead positions, due to pain or apprehension of further dislocation. Many patients display a full range of motion but it is important to note whether this may be considered hypermobile. Beighton's score [13] is typically used to assess for generalised joint hypermobility (see Fig. 2) but does not contain a measure of shoulder range. We consider external rotation of 90° or abduction of 180° or greater to be hypermobile in the shoulder.

Some patients can voluntarily sublux their shoulder on request. This ability in itself is not considered to be problematic unless associated with pain or if in some cases it becomes involuntary over time. This is because of the subtle but important difference between laxity and instability where laxity is excessive movement in a joint that is not symptomatic (i.e. no pain and normal function) whereas instability is excessive movement that is symptomatic. These symptoms may include uncontrollable subluxation or dislocation, pain and loss of function.

Laxity of the shoulder can be assessed clinically using tests such as the sulcus sign or load & shift test but relies on the patient being able to relax the shoulder muscles and is rather subjective in terms of how much movement is considered to be excessive. Laxity can be assessed more easily with the patient or limb anaesthetised allowing full relaxation of the muscles.

Perhaps of more relevance are instability provocation tests such as the apprehension test or posterior jerk test. The apprehension test assesses the patient's reaction to the shoulder being passively externally rotated at 90° of abduction to the extreme of the range. If the patient is fearful of dislocation this suggests that the joint is unstable anteriorly. This finding is reinforced if the movement is repeated whilst the assessor applies firm pressure anteriorly over the humeral head and the patient feels the instability sensation improve. During the posterior jerk test the shoulder is flexed to 90° and adducted with internal rotation. The assessor applies a force at the flexed elbow so that the humeral head is pushed posteriorly in the glenoid. This may reproduce apprehension or a painful clunk within the joint indicative of posterior instability.

Isometric muscle power of the shoulder should be assessed and compared to the contralateral side. As axillary nerve lesions are commonly associated with shoulder instability the deltoid should be tested against abduction, flexion and extension. The rotator cuff muscles are the key stabilisers of the glenohumeral joint so should be assessed for weakness. Rotator cuff tears are rare in patients with atraumatic instability but can be seen in patients over 40 years old. If recurrent instability has precipitated the formation of a paralabral cyst that compresses the suprascapular nerve then

Fig. 2. The Beighton Score. A patient with a total of 5 points or more is considered hypermobile. One point is allocated for hyperextension at each elbow and knee; a point for each wrist flexed so that the thumb is parallel to the forearm; a point for each little finger achieving 90° extension and one final point for palms flat on the floor with knees extended. A maximum score is 9.

weakness is identified on isometric external rotation or on the ‘full can’ and ‘empty can’ tests. The subscapularis can be tested on the ‘bear hug’, ‘belly press’ and ‘lift off’ tests but is rarely found to be deficient.

For patients whose shoulders sublux every time they elevate the arm, simple movement adaptations that modify the activity of the rotator cuff [14–16] may reduce or abolish the subluxation and increase the active range of movement if it was limited by apprehension. Such techniques include asking the patient to:

- maintain their sitting balance on a large Swiss ball
- maintain single (contralateral) single leg standing balance
- grip their fist (ipsilateral)

. . . whilst elevating the arm. If such techniques do reduce the symptoms then this suggests that conservative treatment directed at improving muscle function of the rotator cuff and trunk stabilisers is likely to be successful and also is helpful in motivating the patient.

3.3. Imaging

Plain film radiography (AP and axial views) is useful to exclude an inherent structural abnormality such as glenoid dysplasia. A more useful form of imaging is magnetic resonance arthrogram (MRA) to assess for instability related lesions such as a labral tear. This helps to classify within the Stanmore criteria whether the issue is Type 2 or Type 3. In the case of a Type 2 instability with a structural lesion this gives greater justification for surgery should there be limited progress with conservative management.

In the absence of trauma bony defects are unlikely so computerised tomography (CT) is of little value in this category of instability. Taking into account the less than 100% sensitivity and specificity of MRA, diagnostic arthroscopy may be appropriate for patients with severe symptoms and normal MRA findings and this provides a dynamic assessment tool for the treating clinician.

4. Conservative treatment

4.1. General principles

It is important after a recent dislocation to mobilise the shoulder as soon as possible to prevent muscle atrophy. Sling immobilisation is shown to provide no benefit in reducing future episodes of instability.[17] The patient should be encouraged to actively use the arm for functional tasks as soon as they feel able and this should be supplemented with specific rehabilitation exercises. It is important to counsel the patient that disuse is likely to perpetuate the problem so return to normal activities including sports is recommended provided that the specified activity does not result in frequent recurrent subluxation. This may mean modifying activities initially so that they can be performed in such a way that the glenohumeral joint remains congruent. The therapist should take time to discuss and assess functional movements of the shoulder relevant to the patient’s activities to facilitate this. Apprehension of further instability episodes is often a key concern of patients so early return to activity may be useful in overcoming such fears.

4.2. Evidence-based rehabilitation methods

Whilst conservative management is recommended as the mainstay of treatment for patients with atraumatic shoulder instability, there is a surprising lack of evidence on the subject.[7] Until recently the only reproducible exercise programme with evidence of efficacy was from Burkhead & Rockwood. [18] This programme guided the patient through a series of shoulder exercises to strengthen the shoulder in movements of flexion, extension, abduction, internal rotation and external rotation. Patients began with elastic exercise bands performing five repetitions of five second holds of each movement two or three times a day. After two to three weeks the grade of elastic was increased until all six grades had been completed. The patient then progressed to the same exercises using a weight and pulley system to increase the load, with the addition of push-ups initially in standing, then kneeling and finally in a full horizontal position. At a minimum follow up of two years 83% of 66 patients had a good or excellent result based on the modified Rowe Score. The Rowe Score however is a very basic measure with just four variables and in this study there was no baseline measure to compare against.

Recently an alternative rehabilitation strategy has been published by Watson et al.[19,20] This provides a detailed and complex treatment algorithm beginning with a focus on correction of scapula posture, then strengthening of the posterior shoulder muscles, before the addition of through range flexion and abduction strengthening, finishing with occupational and sport related functional strengthening. The outcomes for this regime have been reported from a service evaluation at minimum three month follow up in 39 patients with significant improvements in the validated outcome measures of the Oxford Instability Shoulder Score (OISS), Western Ontario Shoulder Index (WOSI) and Melbourne Instability Shoulder Score (MISS).[21] Mean OISS improved from 35.76 to 20.67 points and mean WOSI improved from 39.78% to 77.04%. A randomised controlled trial of the Watson Program versus the Burkhead & Rockwood regime has also been completed.[22] At 24 week follow up the 17 patients completing the Watson Program had significantly better outcomes in terms of WOSI and MISS compared with the 20 completing the Burkhead & Rockwood regime. Both groups however gained significant improvements from baseline with similar numbers in each group achieving the minimal clinically important difference in the WOSI and MISS outcomes.

4.3. Weight-bearing proprioception and plyometric exercises

The Burkhead & Rockwood regime focusses on shoulder strengthening whereas the Watson Program includes strengthening alongside targeted scapula stability exercises. Neither method includes many exercises specifically focussed on fast plyometric activities or weight-bearing proprioception. Indeed the Watson Program specifically suggests avoiding weight-bearing exercises in the presence of posterior instability and generally they are only used as an end stage sport specific exercise if the patient’s activities require it.

Evidence suggests that patients with MDI have significantly greater hand position error (i.e. reduced proprioception) compared to healthy controls.[9] A potential explanation for this was proposed by Lephart & Henry (see Fig. 3).[23] Each instability episode may cause a decline in proprioception that in turn results in decreased neuromuscular control and increased likelihood of recurrent instability episodes causing the cycle to perpetuate. It is however possible to improve shoulder proprioception after dislocation with weight-bearing exercises on uneven surfaces such as a wobble-board or ball.[24] Likewise proprioception improves with plyometric exercise training[25] suggesting the incorporation of these exercise methods is important for patients with atraumatic instability.

A treatment regime that incorporates these elements into a rehabilitation programme for patients with shoulder instability is the Derby Shoulder Instability Rehabilitation Programme.[26] In addition to fast plyometric exercises and weight bearing proprioception exercises from the outset (even for those patients with posterior instability), other factors set this programme apart from

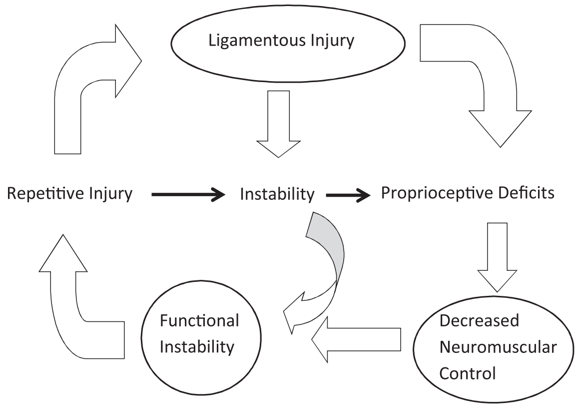

Fig. 3. Reprinted and updated by permission, from Lephart SM and Henry TJ: Restoration of proprioception and neuromuscular control of the unstable shoulder, in Lephart SM, Fu FH (eds): Proprioception and Neuromuscular Control in Joint Stability. Champaign, IL, Human Kinetics, 2000, p 407.).

the others. Patients are progressed through two categories of exercises with only two prescribed at any one time. Each exercise has a target number of repetitions or specified performance time to achieve comfortably and with good technique prior to progression to the next. This means that progression is only made with increased performance and not just expected over time. As such there is an inherent measure of adherence. The focus of the patient's attention is directed to the activity of the hand rather than posture or the position of the scapula and shoulder girdle. Each exercise therefore is designed to restore normal shoulder muscle activity through a functional approach, negating some of the potential negative effects of over-medicalisation of postural asymmetries that are often a normal incidental finding.[27] It is acknowledged that different patients will spend more time practicing the exercises than others and will improve at different rates so follow up times are decided by the patient based upon realistic timescales for achieving the required target for each exercise prescribed. There is only currently published short term outcome data from 18 patients for this programme with significant benefit seen in both OISS and WOSI measures[26] but data monitoring is on-going. As of December 2017 40 patients with atraumatic instability had completed the programme with mean follow up 26 weeks. Mean improvements in WOSI were from 42.04% to 82.91% and mean improvements in OISS from 39.27 to 23.47 points. The improvements were similar to the Watson Program[21] however a mean of only six appointments were required compared to twelve with the Watson Program. The Derby Shoulder Instability Programme instructions are freely available for use via www.DerbyShoulderUnit.co.uk.

4.4. Pain during exercise

Pain can be an emotive topic amongst therapists however it is important to understand that fear of pain can be a huge barrier for patients.[28] Rehabilitation strategies that focus on the avoidance of pain either by complete avoidance of certain activities or by therapist-initiated subtle movement corrections have the potential to further compound the issue. Pain perception is complex[29] but patients should be continually supported to understand that simple exercises such as lifting a light weight or weight-bearing through their arm are not causing any structural harm to the tissues despite sometimes feeling unpleasant. A focus on the functional task or goal rather than instant reduction in pain or clicking should help to reduce this fear. Patients should therefore be encouraged to return to normal activities and persist with rehabilitation exercises even in the presence of pain. Evidence would suggest that this approach paradoxically has a greater effect of reducing pain than a pain-free exercise approach.[30]

5. Surgical management

Surgical management should be considered in patients who continue to suffer from debilitating instability symptoms despite completion of a suitable rehabilitation program. Surgery should be individualized to correct any anatomical cause of instability.[31] Soft tissue techniques most often used are open and arthroscopic capsular shift, thermal capsulorraphy and rotator interval closure.

5.1. Open capsular shift

Inferior capsular shift surgery was popularized by Neer and Foster.[32] The aim of the surgery was to tighten up the hyperlax inferior capsule and reduce joint volume. They described the humerus based capsular shift procedure and reported satisfactory result in 39 out of 40 shoulders.[32] Biomechanical studies have shown that anterior-inferior capsular shift improves joint stability while preserving external rotation with no significant loss of maximum elevation compare to anterior tightening procedures.[33] Volume reduction achieved by capsular shift procedure is directly related to amount of release and shift performed.[34]

Cadaveric studies comparing humeral vs glenoid based capsular shift have reported contrasting results with regards to limitation of rotation. Deutsch et al reported significantly greater reduction of external rotation with glenoid-based shift.[35] However, Remia et al reported significant reduction of rotation with humeral-based shift.[36] Clinically both shift techniques have reported good results. Recurrence rates after both shifts average 5%–10%, whereas loss of external rotation has ranged between 2° and 18°.[37–41] Pollock reported long term results of open capsular shift surgery. At mean follow up of 61 months, 2 out of 49 shoulder re-dislocated.[41]

However, return to sport rates remains sub optimal. Altchek et al[37] reported 40 patients with MDI treated with Bankart procedure combined with glenoid based capsular shift. In their series, 33 of 40 patients (83%) returned to full sport at a mean of 3 years. But, all throwing athletes experienced decreased velocity.

Pollock et al reported that 31 of 36 athletes returned to sports.[41] However, only 25 of these (69%) were able to return to the premorbid level of participation following an inferior capsular shift procedure. Athletic patients should be appropriately counselled regarding return to sports after this procedure.

5.2. Arthroscopic capsular plication

Advances in arthroscopic techniques have made inroads in management of atraumatic instability. Arthroscopic approach has advantages of decreased morbidity, visual confirmation of decreased capsular laxity, and avoidance of subscapularis detachment. In addition, redundancy in the anteroinferior and posteroinferior capsule can be addressed using a single approach or by selectively addressing deficiencies in each capsular region. It also allows for visualization and reconstruction of posterior labrum, deficiency of which can contribute to instability.[31]

Arthroscopic technique involves creating pleats of capsular tissue, which is sutured back to either intact labrum or suture anchors placed at the glenoid margin. Provencher et al in a cadaveric study reported similar load to failure with use of suture anchors and plication through intact labrum.[42] However, labral displacement at failure was higher with plication alone. The authors recommended use of anchors if labral tissue was deficient.

In cadaveric studies, arthroscopic capsular plication has been shown to reduce capsular volume and magnitude of reduction depends upon amount of plication. Flanigan et al demonstrated

16.2% mean volume reduction at 5 mm of capsular plication and 33.7% volume reduction for 10 mm of capsular plication.[43] Multiple pleat techniques have been shown to reduce capsular volume more than open inferior capsular shift.[44] Jacobson et al reported collective results of 7 studies including 219 shoulders.[45] They reported no difference between open and arthroscopic techniques with regard to recurrent instability, return to sport, loss of external rotation, and overall complication. Chen et al in a meta- analysis reported similar outcomes between open and arthroscopic surgery for MDI.[46]

Other studies have reported high patient satisfaction and more than 85% return to sports after arthroscopic management of MDI in properly selected patients.[47–49]

However, over-aggressive plication can result in stiffness, particularly in external rotation. Magnitude of plication is a subjective determination and is individualized to each patient. In general, subluxation of the proximal humerus over the glenoid rim should not be possible after capsular plication is complete and range of motion should not be markedly limited as compared with the contralateral side.[31]

5.3. Thermal shrinkage

Arthroscopic thermal shrinkage is not universally recommended for management of atraumatic instability and has fallen out of favour after studies reporting high recurrence rates, chondrolysis and thermal nerve injury.[46],[50],[51] However, a recent multicentre randomized trial reported 2 year results comparing thermal shrinkage with open inferior capsular shift. Authors reported comparable quality of life and functional outcomes between two groups. Thermal shrinkage group had fewer complications and episodes of recurrence compared with open surgery.[52] Further evidence is required to define its role as a primary procedure or in combination with other procedures in management of atraumatic instability.

5.4. Rotator interval closure

Rotator interval closure remains controversial for managing atraumatic instability. Neer described closing the gap between SGHL and MGHL it as part of open capsular shift surgery.[32] Harryman et al in a cadaveric study described open medial to lateral imbrication of rotator interval. Posterior translation was reduced in neutral, abduction and abduction external rotation position. Inferior translation was reduced in neutral position.[53] This study is cited as a basis of arthroscopic rotator interval closure as a treatment option for MDI.[31] Traditional arthroscopic techniques close the interval in superior- inferior direction by approximation SGHL and MGHL. This technique however has failed to replicate findings of Harryman et al.[54],[55] Using a cadaveric MDI model, Farber et al compared a superior-inferior closure with arthroscopic medial-lateral rotator interval closure.[56] Medial-lateral closure was done to replicate Harryman's technique.[53] The medial-lateral closure resulted in better restoration of motion to the intact state than the superior-inferior closure, and it improved posterior stability.[56] However, the authors were unable to reproduce all of the findings of Harryman et al.[53]

Interval closure alone in cadaveric studies have shown improve anterior stability but evidence of improvement in posterior and inferior stability is inconsistent.[55] Shafer et al reported that in the setting of capsular plication, rotator interval closure may be important to limit anterior-posterior laxity in the abducted position and superior- inferior translation in both the neutral and abducted position.[57] Remia et al have reported that rotator interval closure combined with inferior capsular shift decreased inferior translation in the apprehension position.[36]

Studies have shown good result with or without interval closure.[48] There is no clear clinical evidence of additional stability provided by rotator interval closure in patients with multidirectional instability. Based on biomechanical studies interval closure may be indicated if laxity persists after adequate capsular shift procedure. However, this decision should be taken in consideration with complication of restriction of external rotation. It is recommended that procedure be performed in 30° of external rotation to prevent loss of external rotation.[31]

6. Summary

Whilst atraumatic shoulder instability is much less common than traumatic instability it is often more complex. The history and clinical assessment is key to determining the management strategy but rehabilitation should form the mainstay of treatment. Effective rehabilitation should not only focus on strengthening but also weight bearing and plyometric exercises to aid return to higher function and sport. Surgical options are available when conservative management fails but should not be considered a panacea.

Conflicts of interest

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Management of first time shoulder dislocation

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ABSTRACT

Glenohumeral dislocation is a common emergency department presentation. It is most often a traumatic anterior dislocation and occurs most frequently in young, active male patients. Shoulder instability and further dislocations may occur following primary dislocation, and these are associated with shoulder joint pathology and loss of function. Younger patients are more likely to experience further instability events, while shoulder dislocation is more often a singular event in older patients. There is debate regarding whether first time dislocators should be managed surgically or conservatively. This article discusses the evidence in the literature and current guidelines for the management of first time shoulder dislocation, proposing surgical management for young active patients following a first-time dislocation, most often an arthroscopic labral repair.

Contents

1. Introduction

The glenohumeral joint has the greatest range of movement of all joints in the body. This comes at the expense of being unstable.[1] This is reflected by the frequency of emergency department presentations: glenohumeral joint dislocations are the most common of all large joints with an incidence of 17 per 100,000 per year.[2] The vast majority are anterior dislocations, and follow a traumatic injury.[3] Overall, shoulder dislocations are most commonly seen in athletes, males and in the young.[4,5]

A first-time anterior dislocation can cause damage to a number of different anatomical components of the shoulder joint. ‘Bankart’ lesions are the most common: first described in 1923,[6] this avulsion of the glenoid labrum from the scapular periosteum increases the probability of further anterior instability, and occurs in 86–100% of young patients.[7] Further damage to the ligaments, specifically the middle and superior glenohumeral ligaments, and labrum may occur as isolated injuries or more frequently in combination with Bankart lesions.[2] Damage to the posterolateral humeral head caused by impaction with the harder anterior glenoid causes a ‘Hill-Sachs lesion’ which is seen in 54% of anterior dislocations,[8] and occurrence

Abbreviations: BESS, British Elbow and Shoulder Society; BOA, British Orthopaedics Association; EFORT, European Federation of National Associations of Orthopaedics and Traumatology.

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increases with frequency of dislocation. Glenoid bone loss is seen in 8%,[9] and rotator cuff and greater tuberosity fracture are seen more frequently in older patients.[8] Nerve injury is relatively common with the axillary nerve being affected in 10%, while vascular injury occurs in approximately 2% of anterior dislocations.[8]

Reduced joint stability may result following dislocation, and is especially problematic in young active patients. In 2006, Robinson et al followed 252 patients managed conservatively and demonstrated that 87% of those aged under 20 will re-dislocate, but only 30% of those over 30 years old.[10] A comparison of findings in patients with recurrent instability as compared with primary dislocators demonstrated increased frequency of rotator cuff tears, anterior labral periosteal sleeve avulsion (ALPSA) lesions, intra-articular loose bodies and capsular laxity,[11] while another study compared acute instability patients with those with six or more instability events and demonstrated increased rates of glenoid bone loss and ALPSA lesions.[12] These findings suggest that further instability events after primary dislocation may cause progressive damage to the glenohumeral joint.

For patients with ongoing instability unresponsive to conservative management following traumatic dislocation, surgery can be considered. However, there remains a debate in the literature regarding the best management following the first episode of shoulder dislocation. This review aims to examine the literature for current evidence for best practice in the management of first time dislocators, examining national and international guidelines, and briefly discussing our own practice.

2. Initial management of glenohumeral joint dislocation

Initial management of shoulder dislocation is generally regarded to be a closed reduction as soon as is feasible. The combined British Elbow and Shoulder Society (BESS) and British Orthopaedics Association (BOA) guidelines for traumatic anterior shoulder instability describe a pathway for management.[3] Dislocations should be reduced in a hospital environment, with no attempt at pre-hospital reduction unless in specific controlled circumstances. Plain radiographs in two views should be acquired before reduction; one should be in the anterior-posterior plane and one in the axial if possible. A Wallace[13] or lateral scapular view is adequate if an axial view is prevented by pain. This is to confirm the type of dislocation (e.g. anterior/posterior), and also to ensure no other associated bony injury can be seen, such as a tuberosity fracture or humeral neck fracture.

If closed reduction is appropriate, neurovascular status should be recorded beforehand. Reduction is often first attempted under sedation in the emergency department but may require general anaesthesia. Two radiographic views are then acquired post-reduction, and neurovascular status recorded again.

In our practice, closed reduction is performed under sedation in the Emergency department. The reduction technique preferred is longitudinal traction in the line of the scapula, with counter traction used if necessary. We do not recommend the use of any rotational movement in reduction, especially in patients over 50 years old, due to the possibility of causing an iatrogenic injury, such as a proximal humeral fracture.

3. Post-Reduction management

Following glenohumeral joint relocation there is debate regarding the appropriateness of surgical management: who requires surgery, and when, as well as the type of surgery performed.

3.1. Conservative management

There is some debate about whether immobilisation in external or internal rotation is most effective. Itoi et al. demonstrated that when the shoulder was placed in external rotation, the displacement of a labral tear was reduced on MRI, suggesting this could cause improved healing and therefore reduce recurrence.[14] However, other studies have demonstrated that the position of immobilisation to be equivocal, with no significant reduction in recurrence in external versus internal rotation.[15]

A survey of BESS members in 2009 showed that time scale of immobilisation of a young patient (aged less than 25 years old) varies considerably between surgeons, who opted for 0–6 weeks.[16] This brings into question the efficacy of immobilisation following dislocation, and some studies have investigated recurrence rates and found no difference when comparing immobilisation for a prescribed period of time as opposed to immobilising to patient comfort.[17,18] Further, older patients achieve maximal recovery when mobilised as soon as possible following an instability event.[7]

In our own practice, patients are often sent home in a simple shoulder immobilising sling in internal rotation from the Emergency Department following uncomplicated closed reduction. The images of all patients sustaining dislocations are then reviewed by a consultant orthopaedic surgeon, and triaged to appropriate follow-up, be this primary physiotherapy or to see a shoulder specialist surgeon within the next week. At this point, a decision is made about the requirement of a sling, and the management of the dislocation.

3.2. Surgical management

There has been a recent trend in the literature towards offering surgery to young first time dislocators. In 2002, 35% of British upper limb surgeons would offer surgery to these patients if aged 17–25 years old, while in 2009 68% would offer this surgery.[16]

A longitudinal study of over 250 first time dislocators in Sweden over the course of 25 years was conducted by Hovelius et al.[9] They demonstrated that patients under 22 have 51% recurrence within 2 years, while only 33% felt their shoulder had become stable. In patients over 40 years of age shoulder dislocation is more likely to be a singular event.[8] Sachs et al studied a population of 131 dislocators for an average of four years.[19] Of the 90 patients aged under 40 years old, 39 (43%) had one or more further dislocations, with 37 (95%) of these involved in contact sports or jobs requiring reaching above the chest or head. Only four (10%) of those aged over 40 years old had a recurrent dislocation, none requiring surgery. It has therefore been suggested that a “watch and wait” strategy may be justified in the older population, and given their different sequelae of injuries they require a different treatment pathway as compared with the younger dislocators.[7] While older patients are less likely to have further instability, rotator cuff injuries are far more common.

Repair of these concomitant injuries in the acute setting in this group of patients can improve pain, stability and patient satisfaction.[20] BESS guidelines reflect this, suggesting urgent radiological assessment using ultrasound or magnetic resonance imaging if appropriate in patients over 40 years old.[3]

The impact of further instability events in young patients must be taken into account when considering the possibility of surgical management. Hovelius et al argue that 65% of their young (up to 22 years old) patients with dislocations had spontaneously stabilised by 25 year follow-up, and suggested that stabilising all patients under the age of 25 would result in at least 30% being unnecessary operations.[9] However, this only takes into account shoulder stability as an outcome. Other studies show faster return to baseline activity for both civilians and to service for military personnel.[21]

First time dislocators are more likely to undergo arthroscopic labral repairs and/or capsular plication, while recurrent dislocators are more likely to require open Bristow-Laterjet procedures and treatment of biceps pathology; fewer first time dislocators have bony defects or biceps pathology as compared with those with recurrent dislocation.[22] The evidence for operating on young dislocators was further investigated by a systematic review in 2010 by Godin et al, who found five randomised controlled trials comparing outcomes of operative management of first time shoulder dislocations. The oldest patient was 40 years old, the mean age was 23, and 87% were male. The authors concluded that while evidence was limited, operative stabilisation should be offered for primary acute shoulder dislocation in young active adults participating in highly demanding physical activities.[21]

A radiographic study of the same Swedish cohort of primary shoulder dislocation patients aged 12–40 years by Hovelius et al. 25 years post dislocation showed that moderate to severe arthropathy was evident in 18% of those with no recurrence, but in 39% of those with one or more recurrence. Those surgically stabilised had a lower prevalence (26%), and showed lower rates of moderate to severe arthropathy than those which had stabilised over time, suggesting surgery may reduce rates of arthropathy in primary dislocators if performed before further recurrences, even if the shoulders would subsequently stabilise themselves.[23]

Due to the evidence suggesting that young active first time dislocators could have better outcomes with surgical treatment, the European Federation of National Associations of Orthopaedics and Traumatology (EFORT) sanctioned a review on this topic, and BESS and BOA have recently developed joint guidelines. BESS and BOA recommend that for patients aged under 25 years of age, there should be discussion with a shoulder surgeon within six weeks for clinical examination, risk assessment and shared decision making. If appropriate, diagnostic imaging should be offered, with surgery advised if necessary.[3] As male patients are more likely to have further instability, the guidelines suggest they are more likely to be appropriate of surgical management. For patients aged 25–40 years old they suggest re-review three to six months following the event, to determine if symptomatic instability remains. The EFORT review does not define a specific age nor comment on gender, but suggest surgery in "young" patients performing highly demanding physical activities.[24]

A caveat is that patients with multidirectional instability or hyperlaxity are more likely to fail with an arthroscopic labral repair,[25] and these patients should be recommended for conservative therapy first.[26]

4. Operative technique

For those first time dislocators in whom surgical management is deemed most appropriate, anatomical repairs provide the best outcome with respect to function and complication rates.[27,28] Attitude to operative management for dislocators has shifted. In Britain there was a marked change in operative choice in the previous decade from open to arthroscopic management.[16] This is possibly reflective of a combination of an evolution in skills, techniques and equipment; earlier studies found open procedures to have better outcomes,[25] but more recently have been shown to be equivocal with regards to re-dislocation rates.[29] Shorter recovery times and reduced complication rates in arthroscopic surgery is an obvious advantage.

The most common pathology found in young first time dislocators is a Bankart lesion of the glenoid labrum.[22] In a multicentre unblended study in 1999, Wintzell et al. suggested lavage alone is effective as a treatment for primary anterior shoulder dislocation in sixty 16–30 year olds followed for 1 year following dislocation.[30] However, more recent studies show arthroscopic labral repair more effective than immobilisation or lavage treatment alone. In 2012, Chahal et al performed a systematic review of the literature for randomised and quasi-randomised trials.[31] Two compared labral repair with immobilisation, two compared labral repair with arthroscopic lavage. The rate of recurrent instability was reduced by labral repair as compared with both controls.

In terms of the method of labral repair used, transosseous sutures and resorbable bio-tacks have been used previously, but with high rates of recurrent instability.[32,33] Arthroscopic placement of suture anchors was first described by Wolf in 1993,[34] and was shown separately by Kim et al and by Cole and Romeo to have very low rates of recurrence (0–4%) and excellent rates of return of function.[35,36]

In a patient with a clear history, examination and plain radiograph consistent with an uncomplicated first-time shoulder dislocation without bone pathologies, first line surgical management is usually a labral repair. In a patient with features suggestive of further pathology, CT arthrogram is most accurate to detect bone loss, but MR arthrogram is often sufficient and is better for detection of soft tissue pathology.[3] Bony pathologies are more common in recurrent dislocators,[22,37] and are associated with increased failure rate of labral repairs,[38,39] further suggesting a need for early repair before more complex surgery is required.

5. Summary

Anterior traumatic glenohumeral joint dislocation is a common injury, especially in young, male active patients. Recurrence and associated instability after a primary dislocation is especially high in patients initially at risk of dislocation. The consequences are of short term lack of function during recovery from instability events, and in the long term possible joint damage and associated reduction in function and quality of life. Patients of all age groups with chronic instability without response to conservative management are usually offered surgical stabilisation. Older first-time dislocators often do not experience further instability and so are initially treated conservatively. Debate remains regarding treatment of young first time dislocators. The literature suggests that young (under 25 years old), active male patients, especially those involved in overhead activities, should be treated surgically following their first dislocation. This is reflected in national guidelines, and in our local practice.

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SWK: Manuscript preparation.
PDC: Manuscript editing, project development.

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Review article

A treatment algorithm for locked posterior dislocation of shoulder

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ABSTRACT

Aim: The purpose of the study was to analyse features of locked posterior shoulder dislocation and provide a surgical algorithm to facilitate optimal results in this complex condition and present the results in 31 consecutive cases in 30 patients.

Materials and methods: We present a retrospective series of 39 patients with locked posterior shoulder dislocation. 31 locked posterior dislocations (one bilateral posterior locked dislocation) in 30 consecutive patients of the 39 are included. Patients were classified according to the measured reverse Hill Sachs defect and critical fragment. UCLA scores were measured at a minimum of six months post-operative follow up and radiographs were taken at yearly follow up.

Results: The average UCLA score was 28.61 (minimum 15 and maximum of 35). 17 cases had a good to excellent result with 11 of these 17 achieving a score of 35. There were 9 fair and 5 poor results amongst the 31 cases.

Conclusion: Early diagnosis is desirable to avoid invasive non-anatomical procedures. Assessment of critical fragment and reverse Hill Sachs will achieve a more accurate osteotomy. A native cartilage transfer is much better than iliac crest grafting. The derotation osteotomy is reserved for malunited fracture dislocation patients presenting late and less than optimal results are likely. All the patients presenting late beyond a year inevitably required a hemiarthroplasty.

Contents

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1. Introduction

Even in this modern age, posterior dislocation of the shoulder joint is commonly missed at the first instance, leading to a complex condition of persistent dislocation, chronicity, pain and dysfunction. Although the incidence of posterior dislocations amongst shoulder injuries is less than 2%,[1] worldwide reported rates for missed posterior dislocation vary from 50% to 79%.[2–6] All aspects of locked posterior dislocation are convoluted as compared to anterior shoulder dislocation that is more common, overt and obvious. The rarity of this condition along with its typical manifestations often eludes a prompt diagnosis, further complicating its treatment. The condition is variably named as locked, chronic, missed, persistent or even locked posterior dislocation. The patient not only presents with limited range of movement but pain in the initial stages. While the incidence of nerve injury in locked posterior dislocation of shoulder has not been highlighted in the literature, it is possible that the incidence of nerve injury is more common. In addition to the physical dysfunction, patients of locked posterior dislocation suffer emotionally as their diagnosis was missed and the delay compromises recovery and causes at least temporary disability. Recurrence after surgery is also not unknown and addressing a failed surgery for the same can be an intricate issue. Since there are several variables influencing the outcome of surgery in locked posterior dislocation of the shoulder it has been difficult to give a treatment algorithm for locked Posterior dislocation of shoulder. Due to the paucity of reports and rarity of the condition, it is often difficult to advocate a standard regimen. Age of the patient, duration of the dislocation, extent of the reverse Hill Sach's defect, status of the cartilage, size of the critical fragment (CF) (ref 1.4.a section), neurological status are important factors that need to be considered in decision making. With the wide number of surgical options available, it is the aim of this study to provide a guideline for the treatment of locked dislocation of the shoulder. In addition, after analysis of the factors studied, we have devised a classification system that would make surgical decision making simpler. There is sparse literature on the subject and even fewer papers that provide an algorithm for treatment except for Cicak[1] and Jochen et al.[7]

2. Clinical features

The mechanism of injury is adduction, internal rotation and flexion. Bulky posterior shoulder musculature and the natural lie of the shoulder in internal rotation further mask the deformity. Even then simple understanding of the pathology and awareness of the condition can prevent a misdiagnosis. This is probably the only condition where the affected arm is steeply fixed in internal rotation. In the initial few weeks, pain is disproportionately severe to the apparent lack of deformity. The patient is unable to externally rotate even a few degrees but seldom may reach just short of neutral. If the arm can rotate beyond neutral position it is highly unlikely to be a posterior dislocation of shoulder. The axial movements of forward flexion and abduction are deceptively impressive, though not full. With such robust clinical findings, there is no true differential for this condition. Few conditions mimic a locked posterior dislocation of shoulder closely and an experienced surgeon should easily discern between the two. Rowe & Zarins test[8] demonstrates the inability to fully supinate the affected side forearm, in the presence of a locked posterior dislocation. Although forearm supination is unrelated to shoulder dislocation biomechanically, it is the steeply internally rotated shoulder that causes the lack of supination. (Fig. 1)

A severe frozen shoulder with loss of rotation can imitate a locked posterior dislocation of shoulder. However, in frozen shoulder, both internal and external rotations are affected unlike

Fig. 1. Rowe Zarins test.

a locked posterior dislocation of shoulder where external rotation is predominantly affected. A malunited proximal humerus with restricted rotations comes a close second but again there is usually a global loss in the range of movement, including the axial movements. Apart from these two conditions a locked posterior dislocation of shoulder is unlike any other disorder but is still a dilemma for the surgeon who has never seen one.

In late presentations and lean patients, the humeral head may be felt posteriorly along with some amount of wasting of the posterior muscles. Unlike a chronic anterior dislocation, however, the round contour of the shoulder is retained. To the attentive eye, the coracoid process may be prominent on the affected side.

Seldom locked posterior dislocation of shoulder is associated with neuropathy, usually of the Suprascapular and Axillary nerves. Locked posterior dislocation with an associated neuropathy would complicate an already difficult problem.

AP Radiographs add to the predicament, as they appear spuriously normal. On the AP radiograph the head of humerus appears to be in steep internal rotation; an **inverted light bulb appearance** (Figs. 2 and 3). On a closer look, the glenoid also appears empty; a **vacant glenoid sign**. Like a standard anterior instability, there is a humerus defect, albeit positioned anterior-medially; a reverse hill Sachs defect. This can also appear as a **double trough sign** on an AP radiograph. A lateral radiograph is crucial to the diagnosis and if the surgeon were to take a lateral radiograph, it would be diagnostic. The lateral radiograph provides important information about the humeral head position and the size of the reverse Hill Sachs defect.[9] The humeral head can be seen clearly posterior to glenoid and would also reveal the reverse Hill Sachs, which is typically medial to the long head of biceps groove, in the antero-medial sector of the humeral head. Unfortunately, often, an axial radiograph is not done - partly as it is difficult and also because a primary clinical diagnosis was never made. Whenever the surgeon is perplexed at the presence of disproportionate symptoms compared to the clinical appearance, he should not hesitate to seek a CT Scan that would clinch the diagnosis. A CT scan is recommended even if a correct diagnosis has been made on radiographs. The CT will allow classification of the dislocation and

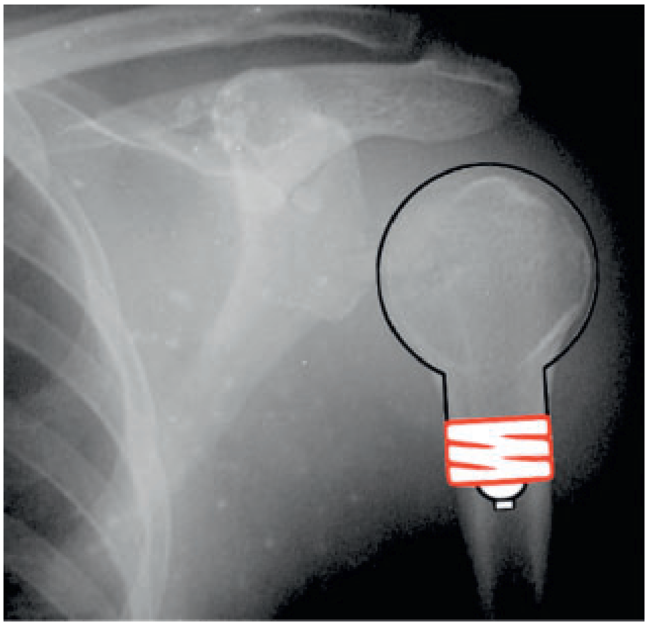


Fig. 2. Inverted light bulb sign comparison.

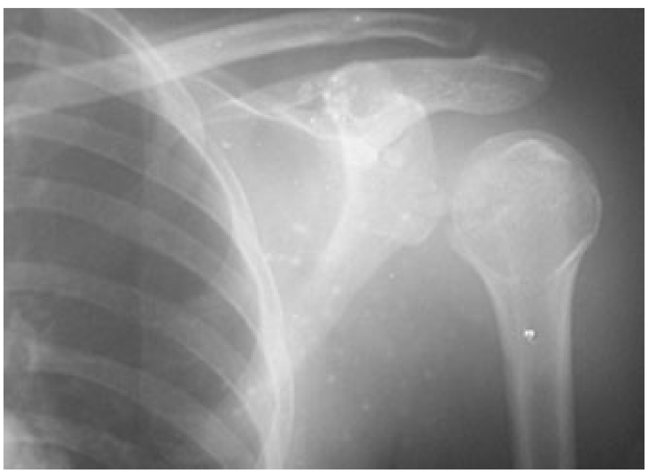


Fig. 3. Inverted light bulb sign comparison.

measure the size of the reverse hill Sachs defect and the critical fragment (to be discussed below).

3. Material & methods

The study included patients from June 2004 to July 2016 with a minimum of six months post-operative follow up.

A retrospective study of a series of 31 patients with 39 locked posterior dislocations was undertaken. Three patients did not under go surgery either because they were well compensated and pain free or it was expected that surgical intervention would not improve on their current shoulder function. Five patients were lost to follow up. All patients were scored post-operatively with a UCLA score. Causal injury, duration since injury and extent of reverse Hill Sachs defect were measured. Three patients had been operated on previously, re-dislocated & had presented to our unit for revision surgery. Of these three patients, two were primarily operated without a correct diagnosis of locked posterior dislocation and the third was operated for a locked posterior dislocation of shoulder but re-dislocated within six weeks of the index surgery. A classification was devised to facilitate the treatment strategy.

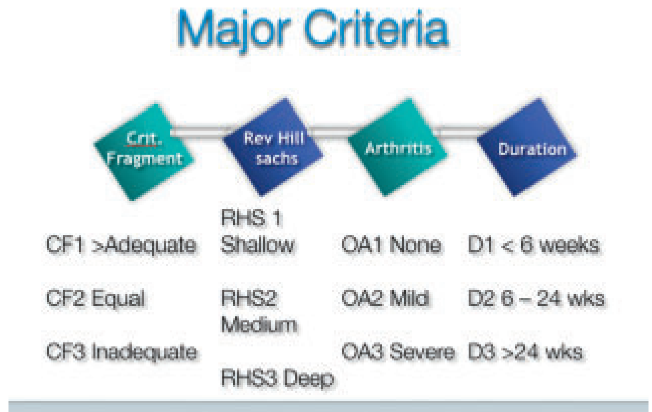


Fig. 4. Major criteria.

3.1. Classification

Major and minor criteria were relevant in planning the surgical option. Four major criteria were considered and quantified accordingly. Minor criteria included whether previous surgery was performed, age and neuropathy of the patient. The classification of each criteria was serially categorised from lesser to major or simpler to more complex as per conventional wisdom (Fig. 4 and Table 1).

3.2. Size of the critical fragment – CF 1, 2, 3

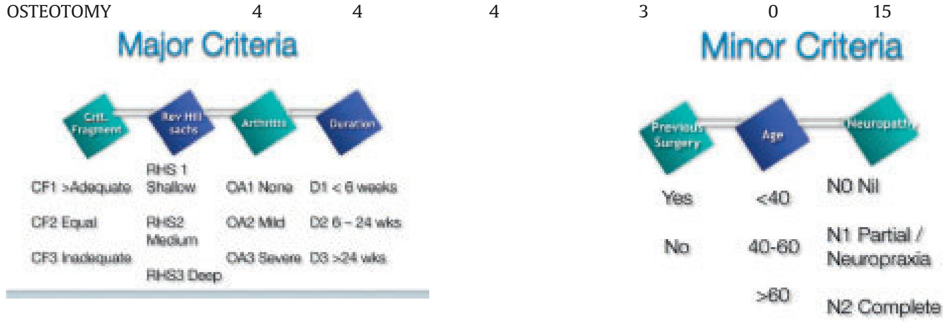
The *critical fragment* (Fig. 5) was defined as the size of the available osteotomy starting medially from the intertubercular sulcus to the lateral margin of the reverse Hill Sach's defect. The subscapularis inserts on the lesser tuberosity, which constitutes majority of the *critical fragment*. When the size of the *critical fragment* was larger than the reverse Hill Sach's defect then it was classified as CF1. A CF1 (Fig. 6) suggests a more than adequate lesser tuberosity osteotomy, if it was required. A critical fragment that was equal to the reverse Hill Sach's defect was classified as CF2 (Fig. 7). In a CF2 critical fragment, the surgeon must design the osteotomy to fill the defect accurately. CF3 (Fig. 8) denoted a non-existent or miniscule critical fragment, such as when the reverse Hill Sach's defect extended into the inter-tubercular groove. A CF3 critical fragment meant a functioning osteotomy was not feasible and either an osteochondral allograft or a shoulder replacement should be planned.

3.3. Extent of the reverse Hill Sachs defect – RHS1, 2 & 3

If the size of the reverse Hill Sachs defect was negligible or less than 20% the size of the head of Humerus it was classified as RHS1. Such a defect could be left alone provided post reduction the shoulder joint was stable on rotation under anaesthesia. However, after reducing the dislocated head, one should evaluate the stability in all provocative positions and confirm stability of the joint. If a RHS1 defect is even marginally unstable after reduction a lesser tuberosity osteotomy ought to be done. This is exceedingly rare and since the defect is small, an osteotomy may not have sufficient area for two screws. We believe two 4 mm screws are mandatory to prevent rotation of the fragment. A defect between 20% & 50% the size of the head of the Humerus was classified as RHS2 and requires a lesser tuberosity osteotomy to fill up the defect. In effect, not only does the lesser tuberosity transfer fill the reverse Hill Sachs defect, but it also transfers native cartilage to the defect. A reverse Hill Sach's defect more than 50% of the head of the Humerus diameter would be impossible to fill up with the lesser tuberosity osteotomy and be classified as RHS3. It is possible that in the presence of a large Hill Sach's defect an osteochondral allograft could be preferred. Unlike a

Table 1

Procedure	Pain	Function	Active FFx	Strength	Satisf	UCLA	Duration since Wks	Age of Pt @ Surgery
Modified Maclaughlin	10	10	5	5	5	35	6	52
Modified Maclaughlin	2	8	4	6	5	25	6	34
Modified Maclaughlin	10	10	5	5	5	35	6	45
Modified Maclaughlin	8	8	3			19	2	31
Modified Maclaughlin	10	10	5	5	5	35	3	55
Modified Maclaughlin	8	6	4	4	5	27	12	32
Modified Maclaughlin	10	8	3	4	5	30	12	28
Modified Maclaughlin	10	10	5	5	5	35	8	57
Modified Maclaughlin	6	6	3	3	0	18	13	31
Modified Maclaughlin	10	10	5	5	5	35	2	50
Modified Maclaughlin	8	6	3	4	5	26	10	26
Modified Maclaughlin	6	6	3	4	5	24	4	48
Modified Maclaughlin	8	8	3	4	5	28	9	30
Modified Maclaughlin	6	6	3	4	5	24	3	43
Modified Maclaughlin	10	10	5	5	5	35	3	41
Modified Maclaughlin	6	6	2	3	0	17	3	52
Modified Maclaughlin	6	6	2	5	5	24	2	40
Modified Maclaughlin	8	6	3	4	5	26	10	31
Modified Maclaughlin	6	6	3	5	5	25	4	57
Modified Maclaughlin	10	10	5	5	5	35	3	42
Modified Maclaughlin	10	10	5	5	5	35	6	26
Hemiarthroplasty	8	10	5	5	5	33	260	51
Hemiarthroplasty	8	8	5	5	5	31	4	52
Hemiarthroplasty	8	10	5	5	5	33	4	52
Hemiarthroplasty	6	6	3	4	0	19	12	28
Hemiarthroplasty	8	10	5	5	5	33	6	54
Hemiarthroplasty	10	10	5	5	5	35	104	38
Hemiarthroplasty	10	10	5	5	5	35	52	35
CR	10	10	5	5	5	35	0	43
OSTEOTOMY	8	6	3	3	5	25	7	31
OSTEOTOMY	4	4	4	3	0	15	7	42



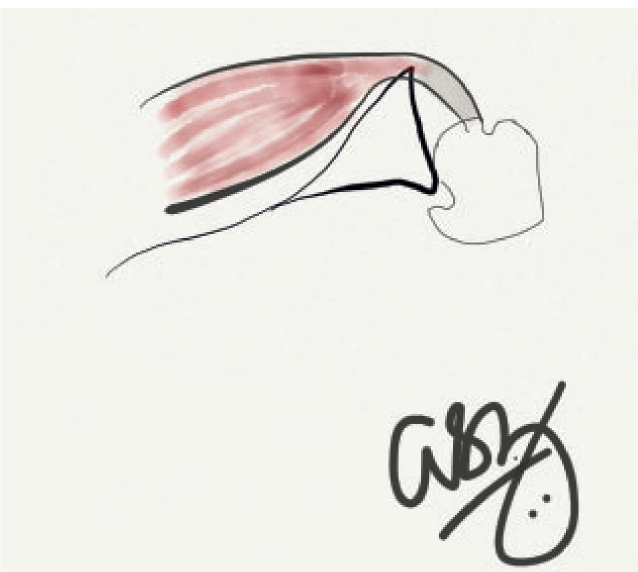


Fig. 5. Reverse HilL Sachs & Critical fragment.

conventional Hill Sachs, a reverse Hill Sachs is an intra-articular defect. We believe an iliac crest bone graft being non-articular surface may not be the most ideal graft for articulation with glenoid. Alternatively, if the patient is elderly and the dislocation is chronic, then a shoulder replacement may be the ideal solution.

3.4. Arthritis OA 1,2 & 3

OA1 arthritis was classified provided there was no cartilage injury or minor cartilage wear that was negligible. OA2 arthritis suggested type II/III cartilage (Outerbridge classification) wear or focal defects of cartilage (apart from the reverse Hill Sachs defect) that was still compatible with salvage. Such a patient needs a long term follow up for progression of osteoarthritis. OA3 arthritis meant there was extensive cartilage wear and overt arthritis and in such a situation, salvage of the joint was not possible and only arthroplasty or arthrodesis was viable.

3.5. Duration since injury D 1,2 & 3

There is no doubt that any persistent dislocation of shoulder is an emergency and should not entail any delay in treatment. Within 48 h of dislocation, we have performed a closed reduction safely

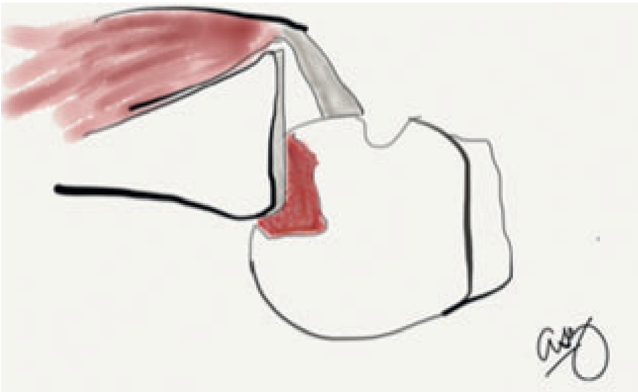


Fig. 6. RHS 1 & CF1.

Fig. 7. RHS 2 & CF2.

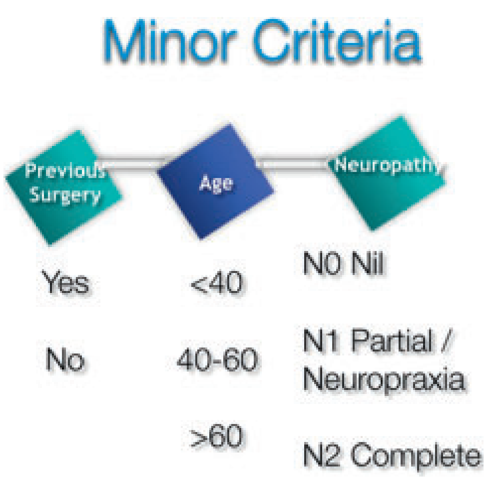


Fig. 8. RHS3 & CF3.

and avoided complex surgery. Beyond 48 h it is perhaps best to safely perform open reduction in most cases. Provided history is less than three weeks and two attempts at closed reduction under anaesthesia have failed, then a decision to open reduce must be taken. Any persistent posterior dislocation within six weeks of injury was considered viable and primary salvage of the joint planned. Between 6 weeks to 24 weeks it is expected that some degree of cartilage damage has happend but if pre-operative MRI or radiographs are normal then a primary salvage should be prioritised. When it is more than 24 weeks of persistent dislocation, cartilage is likely to be necrotic and perhaps pointless to perform primary salvage of the joint. An arthroplasty or arthrodesis are viable options. Any attempt to reduce a four-month-old dislocation is fraught with risk of neurological injury and also likely to convert a patient from a painless unstable joint to a painful stable joint. It is not unusual to leave these patients untreated, as a few of these patients with chronic dislocation of the shoulder may retain a painless functional range of movement. Needless to say, the axial movements are impressively good but the rotations tend to be very poor and restricted.

The most chronic posterior shoulder dislocation operated in this series was five years from index injury and underwent a shoulder replacement.

3.6. Minor criteria

The presence of axillary or Suprascapular nerve injury did not directly influence the type of surgery but helped prognosticate the improvement in function for the patient. Once the shoulder was reduced, it is expected that the neuropathy will start improving. If there was a complete nerve injury it was classified as N0 and if the nerve injury was partial or recovering it was labelled N1. In the absence of nerve injury, it was classified as N0.

Amongst the three patients that had undergone previous surgery, two had to be operated with a derotation osteotomy. The third patient had a plate in situ with a non-salvageable head fragment and hence underwent shoulder replacement. Revision surgery adds a whole new dimension due to scarring, delay and wasting of deltoid and rotator cuff muscles.

Age was of relative importance. Naturally every effort should be made in the young patient to salvage the head of the Humerus. Occasionally when the reverse Hill Sachs was classified as RHS3, it was impossible to avoid shoulder replacement. Even in the elderly if the head was viable and the Hill Sach's defect RHS1 or RHS2, the surgeon must endeavour to avoid shoulder replacement. However, in the elderly patient the speed of muscle wasting, stiffness and cartilage degeneration may compel the surgeon to choose arthroplasty over joint salvage.

3.7. Surgical planning

Before the actual surgery, a proper pre-operative plan must be in place based on the above classification. According to the plan, the surgeon must counsel the patient about the surgery and future outcome that may be expected. It is usual in our practice to give detailed knowledge of the open reduction and osteotomy requiring two screws for fixation followed by a post operative immobilisation in a custom made external rotation splint for a period of 4–6 weeks. This is followed by a supervised rehab programme for two weeks and a phase II home programme for six to eight weeks. It is rarely possible that the critical fragment may be insufficient in size to restore adequate stability and in such a case the surgeon may need to resort to a derotation osteotomy. In such a scenario, the post-operative outcome and plan is likely to change. A derotation osteotomy will require a more elaborate plate fixation and hence the external rotation splint becomes unnecessary. However due to the nature of the derotation, the patient will lose out on external rotation. In our experience, the one movement that the patient of locked posterior dislocation desires is external rotation. Hence pre-op counselling is important to prepare the patients mind in such an event. For any complicated locked posterior dislocation, it is our opinion that the surgeon must keep a shoulder prosthesis for shoulder arthroplasty at hand.

True AP & Axial radiographs are standard practice. The presence of proximal migration of head of humerus, joint space reduction and osteophytes are noted along with deformity of head of humerus. The CT scan confirms the extent of posterior migration, presence of OA and irregularity of humeral head. Most importantly the Reverse Hill Sachs (RHS) defect and critical fragment (CF) are calculated to classify the defect into RHS1,2 or 3.

A small RHS1 defect and large CF1 may simply require a simple close reduction under anaesthesia and the resultant joint is likely to be stable. This stability should be checked by rotating the shoulder through a provocative arc – especially forward flexion, adduction and internal rotation. A RHS2 medium sized defect with an adequate critical fragment CF2 is tailor made for a lesser tuberosity osteotomy, which would be a cartilage transfer along with filling up the reverse Hill Sachs defect. A word of caution here to emphasise, the need to separately measure the reverse Hill Sachs defect and critical fragment. A medium Hill Sachs defect need not necessarily accompany an adequate critical fragment. If the RHS is placed more laterally, then it will be closer to the biceps groove and leave a much smaller CF, inadequate to fill the RHS. The position of the RHS depends on the mechanism of injury and the degree of adduction, internal rotation during the event. Our experience is the epileptic patients tend to have a much more compromised critical fragment. Hence it is important to measure the RHS & CF independently.

3.8. Procedure

Preoperatively it should be established that there is no evidence of osteoarthritis and no permanent neuropathy. The patient is positioned in a semi-beach chair position with a horse-shoe support to secure the head and neck of the patient. An image intensifier is positioned behind the operation table to plan the osteotomy and screw length and direction. A standard delto-pectoral approach is recommended in all cases. We have never had to resort to a posterior approach in any of the patients in this series. After identifying the cephalic vein followed by the conjoined tendon originating from the coracoid, a modular retractor is placed. Next the subscapularis is identified but **not cut,** as the critical fragment would then be rendered avascular after the osteotomy. It is important that the subscapularis be retained on the lesser tuberosity not only to keep the osteotomised fragment vascular but also to maintain a dynamic element to the transfer. Cicak[1] suggested that the upper subscapularis with the capsule may be divided to improve the exposure but we have preferred not to resort to handling the subscapularis. To evaluate the position of the RHS and plane of the osteotomy the surgeon must work through the resected rotator interval. Creating a window in the rotator interval allows the surgeon to place a blunt bone lever or Fukuda retractor between the head of the Humerus and the glenoid and gently lever the head into the glenoid. In addition to the lever, one needs to apply finger pressure on the posterior aspect of the dislocated head of the Humerus to help guide the head of humerus into the glenoid. In the absence of any fracture of the neck of the Humerus this is not a complicated step. In chronic cases, when one has difficulty in reducing the shoulder,[1] a blunt bone lever may be inserted into the RHS and distract the humeral head laterally. Only after the humeral head disimpacts, should the surgeon perform a gentle external rotation manoeuvre to reduce the dislocated head into the joint. However, in the presence of a fracture neck Humerus, which maybe malunited, often the head of the Humerus unites in retroversion and locks the head of humerus behind the glenoid. This can lead to a great difficulty in reducing the head back into the glenoid. This probably is the most difficult part of the procedure and the longer the duration since injury, the more difficult is the reduction. At this point if the injury is classified as RHS 1 & CF1 then that is all that is required. It is imperative to put the joint through provocative movements to ensure the shoulder is stable (Fig. 9).

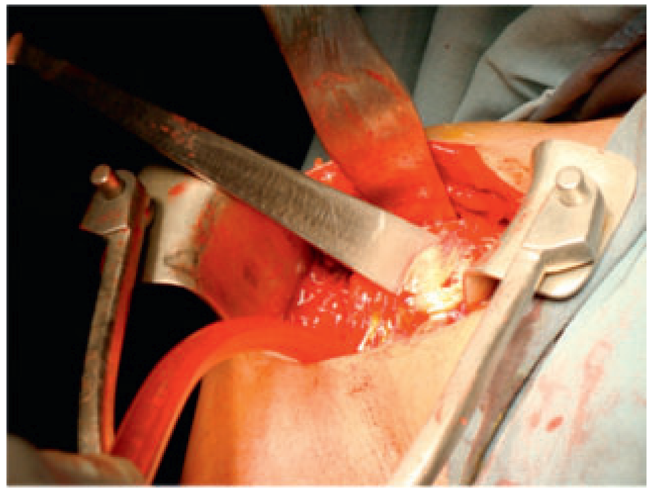


Fig. 9. Lt Osteotomy.

In case a lesser tuberosity osteotomy is required, It is imperative to perform the osteotomy, after relocating the shoulder joint. Once reduced, provided the RHS & CF are adequate, the surgeon should plan the lesser tuberosity (LT) osteotomy. Place a 20 mm osteotome (Figs. 10 and 11) just medial to the biceps groove and direct the same to the floor of the defect and double check this under the image intensifier in the axial plane. Once a clean osteotomy is completed, the exposure of the joint is improved dramatically. The joint should be inspected for glenoid cartilage defects, loose fragments and a thorough lavage is performed. The critical fragment is now transferred more medially into the RHS defect. If the osteotomy is well planned this will match the RHS defect imperceptibly and this is then fixed with two low profile cannulated cancellous 4 mm screws. The direction and length of the screws are gauged on the image intensifier. The stability of the construct and shoulder joint are checked by taking the joint through rotational movements. After a through lavage and suction drain, the wound is closed in layers. It is not mandatory to close the rotator interval, as this will make the joint stiff. Post-operative radiographs are taken in both planes. The shoulder is immobilised in an external rotation brace that is sized for the patient. (Figs. 12 and 13).

3.9. Humeral rotational osteotomy

Rotational osteotomy of the proximal humerus has also been described in literature[10,11] but other authors do not recommend this technique because of the technical difficulty, a high percentage of osteoarthritis progression, and the risk of humeral head necrosis.[10,11] We recommend this only as a salvage procedure, if there are no viable alternatives. The rotational osteotomy of humerus involves a transverse osteotomy exactly at the surgical neck of humerus, where the vessels and axillary nerve winds around the humerus. The shaft of humerus is then internally rotated in relation to the humeral head and fixed with a locked

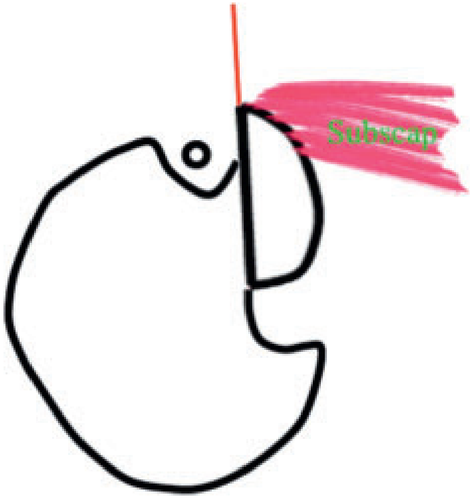


Fig. 10. LT Osteotomy.

Fig. 11. Pre op inverted light bulb sign.

Fig. 12. Post o.p Two screws mandatory.

Fig. 13. External rotation brace.

plate.[13] Although the RHS is not addressed, the internal rotation of distal fragment, ensures the RHS does not engage the glenoid. The biggest disadvantage of the humeral rotational osteotomy is reduction in effective external rotation. We believe, the one movement that a patient of chronic locked posterior dislocation desperately needs, is in fact, external rotation. Hence we reserve the rotational osteotomy to the revision surgery group of patients or patients of chronic locked posterior dislocation associated with a malunited proximal humerus fracture.

4. Results

30 of 39 patients were available for review and complete data collection. Patients diagnosed with a locked posterior dislocation from Jan 2003 up until Aug 2016 were included in the study. Four patients suffered an electric shock, three (Four shoulders as one was bilateral) were epileptic and twenty-three were road traffic accidents. Patients varied in duration of injury, to time of final surgery, from a minimum of one day to five years late. Five of these thirty-one cases required revision surgery after the previous surgeon had either missed the diagnosis or even after diagnosis, posterior instability had recurred. Two of these five patients had at least two surgeries elsewhere, prior to the final surgery. There were 26 males and 4 females. The patients average age was 41.8 (range 26–57 years). Average delay in presentation at hospital was 18.8 weeks (range 0 to 260 weeks).

Patients were regularly followed up with a minimum follow up of 14 months and a maximum follow up of 15 years (mean 26 months). Modified McLaughlin procedure, without cutting the subscapularis, was performed on 21, hemiarthroplasty on 7,

Table 2

Modified McLaughlin	21
Hemiarthroplasty	7
Osteotomy	2
Closed Reduction	1

Table 3

UCLA		
Excellent	34–35	**11**
Good	28–33	**6**
Fair	21–27	**9**
Poor	0–20	**5**

	PAIN	Function	Active Forward flexion	Strength	Satisfaction	UCLA
Average	8	8.1	4	4.5	4	28.61
S D	2.06	1.96	1.06	0.75	1.81	6.456.46

derotation osteotomy on 2 with one patient having successful closed reduction (Table 2).

Practically all the five patients who presented a year after injury underwent hemiarthroplasty; we were unable to salvage the native humeral head in these late presenting cases as the cartilage was extensively damaged. The other patient who underwent bilateral single stage hemiarthroplasty had a massive RHS, background of renal failure who was elderly and epileptic. There were no redislocations, infections or arthritis up until the last follow up in any of the 31 cases. One patient of derotation osteotomy underwent an implant removal 18 months after her index surgery. Only one patient who presented 24 h after his injury, could be treated by closed reduction successfully. Both patients who underwent derotation osteotomy had malunited posterior fracture dislocation with complex non-anatomical deformities of the proximal humerus, in addition to the posterior dislocation. Amongst the seven cases where hemiarthroplasty was undertaken, two had a locked plate fixation and had to undergo revision surgery along with implant removal. One patient of hemiarthroplasty had undergone a manipulation of his shoulder after an unrecognised locked posterior dislocation and thereby sustained a fracture of the proximal humerus with the humeral head still locked posteriorly.

The average UCLA score was 29 (minimum 15 and maximum of 35). 17 had a good to excellent result with 11 of these 17 achieving a score of 35. There were 9 fair and 5 poor results amongst the 31 cases (Table 3).

5. Discussion

Locked posterior dislocation of the shoulder is without doubt a complex condition, further complicated by a delay in the diagnosis. Due to rarity of the condition and paucity of literature, there are few evidence based articles. As there are several variables, it is almost impossible to provide a cookbook recipe for the condition. Variables like duration since injury, extent of CF & RHS, presence of osteoarthritis, neuropathy, age differences and underlying co-morbidities like epilepsy & electric burns, create a challenging situation to define an algorithm. Cicak[1] and Jochen et al[7] have mentioned a treatment path for this confounding condition. We have probably for the first time, described a critical fragment and included the important variables, to provide an algorithm. We admit this algorithm may be imperfect, due to the number of variables. However, we have tried to provide guidance for most combinations of RHS & CF. We have categorised the vital factors as major variables which will dictate the surgery of choice and other factors which also need to be accounted for but may not impact the choice of surgery directly. With our series of 31 cases, a substantial number for an uncommon condition, we have learnt, analysed and presented guidelines which could provide a methodology for addressing this complex condition. This series does not include the patients that were self-reduced and were not locked in situ. Almost 25–44% of these self-relocating patients have a residual posterior instability and may need an arthroscopic subscapularis tenodesis &/or posterior labral repair.[7] We think these are a different set of patients, whose treatment goal is uncomplicated and these could be categorised as recurrent posterior instability. This paper restricts the study and discussion to the unreduced posterior dislocation that remains a veritable challenge.

Most published articles are a mix of case reports and diverse cases of posterior dislocation. Surprisingly very few articles have mentioned associated neuropathy. 10 of our 30 patients had suprascapular neuropathy and two had partial Axillary neuropathy that were recovering. These two nerves are most commonly affected. These are the shortest nerves around the brachial plexus and poorly tolerate stretch and injury. Patients that had neuropathy or poor rotator cuff tissue had the worst results of the cohort.

Few surgeons have advocated addressing the RHS with iliac crest graft.[14,15,9] Gerber[14] reported four patients with a 68 month follow up, with three of four patients achieving minimum restriction and fourth patient developing symptomatic necrosis of humeral head six years later. Martinez[15] reported six patients with allograft reconstruction of the RHS with four of the six patients with almost normal function. Two patients developed collapse of the allograft with osteoarthritis. None of the published cases of Iliac crest grafting of the RHS have reported long term incidence of osteoarthritis due to the non-articular surface of the graft. We believe that since the RHS is an intra articular lesion that articulates with the glenoid, it is inappropriate to have a non-cartilagenous graft for the long-term survival of the joint. Even an osteochondral allograft will heal as fibrous cartilage and is likely to be less than perfect though better than an iliac crest graft. In this context Bock et al[16] reported technique of elevating the cartilage and bone grafting the subchondral bone looks ideal and perhaps feasible for focal defects, that can be addressed by small tunnel to elevate the defect. A posterior surgical approach with glenoid osteotomy and bone grafting has been suggested[17,18] but these are for complex posterior recurrent instability patients and Randelli[18] cautioned about post op osteoarthritis after posterior glenoid osteotomy. The posterior approach is futile for a locked posterior dislocation. The main pathology is the reverse Hill Sachs defect which should be addressed through the delto-pectoral approach, which is familiar to most surgeons. Relocating the dislocated humeral head is more difficult as the duration of dislocation becomes chronic but is not impossible through the delto-pectoral approach. Except for the two patients of derotation osteotomy, we immobilised all the patients in an external rotation brace for four to six weeks.

6. Conclusion

A locked posterior dislocation of shoulder remains a complex problem, whatever the duration of injury. A prompt correct diagnosis would avoid the complications and suboptimal results and prevent a shoulder replacement in the late presenting patients. It is imperative that we spread awareness and learn to diagnose the condition in the first instant. Correct planning and mapping of the RHS & CF will facilitate an accurate lesser tuberosity osteotomy, which not only fills the RHS defect and also transfers native cartilage into the articular defect. All the chronic cases beyond a year ended up with a hemiarthroplasty. We firmly believe that the derotation osteotomy is a salvage surgery with a compromise in result, reserved for complex malunited fracture dislocations. Our results were gratifying, the principle aim of surgical intervention being to achieve a stable, congruous jont that is functional and pain free shoulder.

Conflict of interest

None.

Acknowledgements

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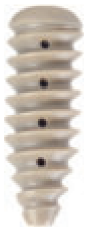

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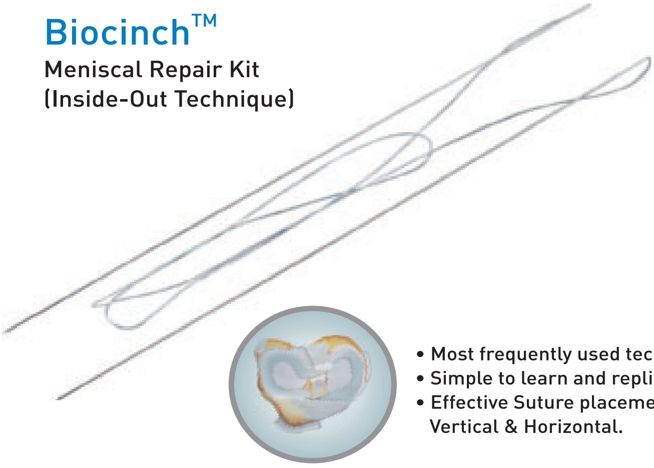

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Anterior shoulder instability in collision and contact athletes

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ABSTRACT

Glenohumeral instability is a common problem for the collision and contact athlete. An understanding of the pathoanatomy, aetiology and injury mechanisms can help guide the clinical examination and appropriate investigations. This in turn can help guide appropriate management of patients, with the aim to return them to pre-injury levels of sport.

Contents

1. Introduction

Glenohumeral instability is a common disability for the collision and contact athlete. It accounts for 23% of shoulder injuries in American Collegiate Athletes,[1] and in Australian professional rugby union players was the soft tissue injury that led to the greatest time off.[2] Similar results were found in the UK professional rugby competition where glenohumeral instability results in the highest rate of absence from playing and training and has the highest recurrence of all shoulder injuries.[3] Rugby league, Australian Rules football, lacrosse and ice hockey all had similar high rates of shoulder injuries and instability.[4–7]

Collision and contact sports are often treated as the same patient group. Collision sports are best described as sports where the athletes purposely and repeatedly collide at high force with each other or inanimate objects, such as the ground. Sports such as rugby union, rugby league, lacrosse, American football and boxing are typical collision sports. In contrast, in contact sports whilst collisions still occur regularly during the game, such as soccer and basketball[8],[9] they usually involve lower levels of force. It may be important to differentiate between these two groups, as we know that a sport such as rugby union has a specific and different high velocity injury pattern to many other sports.[10],[11]

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2. Pathoanatomy of anterior instability

The glenoid labrum improves the stability and depth of the glenohumeral articulation[12] but also act as the insertion of the glenohumeral ligaments to the glenoid. Removal of the labrum in cadaveric specimens leads to easier dislocation in the anterior and inferior direction.[13] Avulsion of the labral attachment of the antero-inferior glenohumeral ligament (AIGHL), between the 3 and 5 o'clock position, in traumatic anterior shoulder instability, was described in detail by Blundell Bankart in 1938[14] (Fig. 1). This Bankart lesion is the most common lesion seen in first time traumatic shoulder dislocations.[15],[16] However, we now understand that the soft-tissue injury not only involves avulsion of the AIGHL from the glenoid but the ligament may also avulse from the humeral side (HAGL), mid-substance capsular injuries or in combination.

The soft tissue Bankart tear can occur with an associated avulsion fracture of the anterior glenoid rim (bony Bankart lesion), particularly with the higher energy dislocations seen in collision and contact sports. Further erosive glenoid bone loss can occur in recurrent shoulder instability and has been reported as affecting up to 90% of patients.[17] Higher rates of glenoid bone defects have been found following traumatic dislocations in younger aged patients.[18] Bone loss decreases the safe zone that the humerus can pass through before becoming reliant on the AIGHL for restraint.[19] Itoi et al in 2000 found in a cadaveric model, that in the presence of glenoid bone loss of greater than 21%, repair of a soft tissue Bankart lesion failed to provide sufficient stability in translation and external rotation.[20]

Bone defects can also occur on the humerus, as a pathological impaction fracture of the posterior humeral head as described by Hill and Sachs in 1940.[21] The arthroscopic appearance of the Hill Sachs lesion was well described in 1989 by Calandra et al, who found it present in a high proportion of their instability cases.[22] Burkhart and De Beer reported a 100% failure rate for arthroscopic stabilization procedures in patients who had an engaging Hill Sachs lesion, which they defined as a defect that engages with the glenoid in a functional position of abduction and external rotation.[23] However, it is the combined relationship of bone loss on both the humerus and glenoid that determines the implications on instability. Yamamoto quantified this bipolar loss via the "glenoid track" concept. The glenoid track was defined as the contact zone between the humerus and the glenoid during maximal external rotation and increasing degrees of abduction. In a cadaveric model they mapped out this contact zone and deemed this the glenoid track. When the Hill Sachs lesion fell medial to this, they were defined as "off track" and therefore more likely to engage during physiological range. "On-track" lesions were contained within the track and therefore should not engage in physiological movement. The measurement from the posterior rotator cuff footprint to the medial margin of the normal glenoid track measured 84% of the width of the glenoid, with glenoid bone loss leading to a narrower glenoid track and potentially more "off track" lesions[24] (Fig. 1). Kurokawa et al found that "off track"" humeral lesions were associated with glenoid bone loss of at least 12%, with more than half of the patients having glenoid bone loss of greater than 20%. [25]

3. Aetiology and injury mechanism

The mechanism of injury pattern can guide the clinician to the injury and commonly occurring associated lesions. Within rugby, the injury mechanisms can be broken down into an injury involving the tackler, try-scoring injury, direct impact injury and flexed fall injury[10],[26] (Fig. 2). However, these positions are frequently replicated in all collision and contact sports.

The tackler has a posteriorly directed force applied to the abducted, externally rotated arm, usually leading to an anterior shoulder dislocation. This will often lead to the standard Bankart lesion, with SLAP tears and HAGL injuries also common.

The try-scorer has a posterior force applied with the arm in flexion rather than abduction. Whilst both Bankart tears and SLAP tears are common, rotator cuff tears are more common with this mechanism (Fig. 3).

The direct injury mechanism is when the athlete falls directly onto the lateral side of the shoulder with the arm held by the side, often in internal rotation. This exerts a large compressive force across the glenohumeral joint leading to a higher rate of bony glenoid lesions as well as complex labral tears. Fractures around the shoulder girdle as well as acromio-clavicular injuries are also common.

Flexed fall injury is the last common mechanism, where the athlete falls onto the elbow with the arm held in a flexed posture. This results in a posteriorly directed force across the shoulder joint causing higher rates of injury to the posterior shoulder, including posterior labral and glenoid damage, posterior HAGL tears and posterior rotator cuff injuries.

4. History & clinical examination

A detailed description of the mechanism of injury is crucial, as this will frequently provide much of the information required. However, shoulder instability can occur as subluxation events

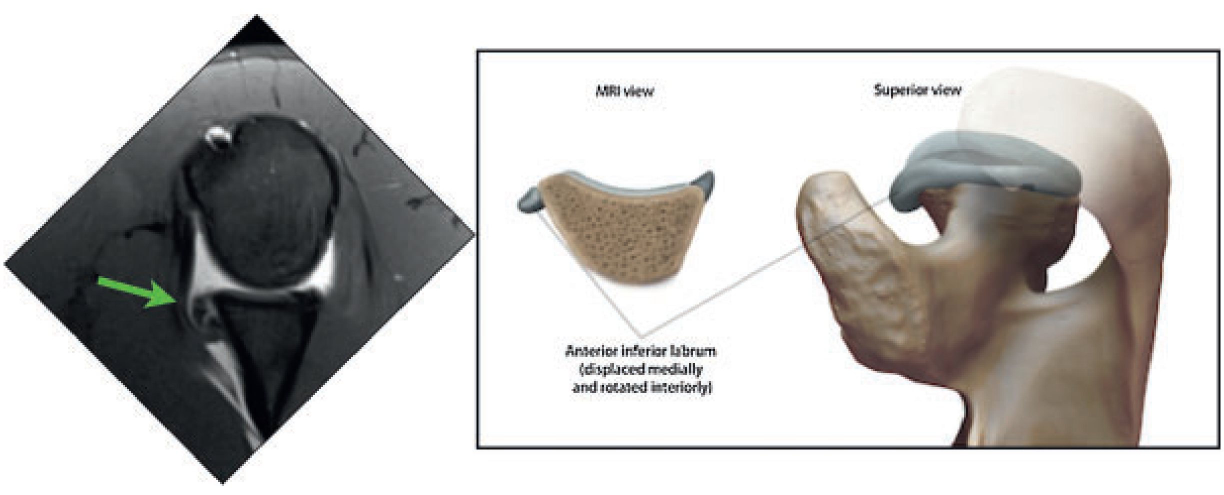

Fig. 1. Labral tear.

Antero-inferior labral tear as described by Bankart.

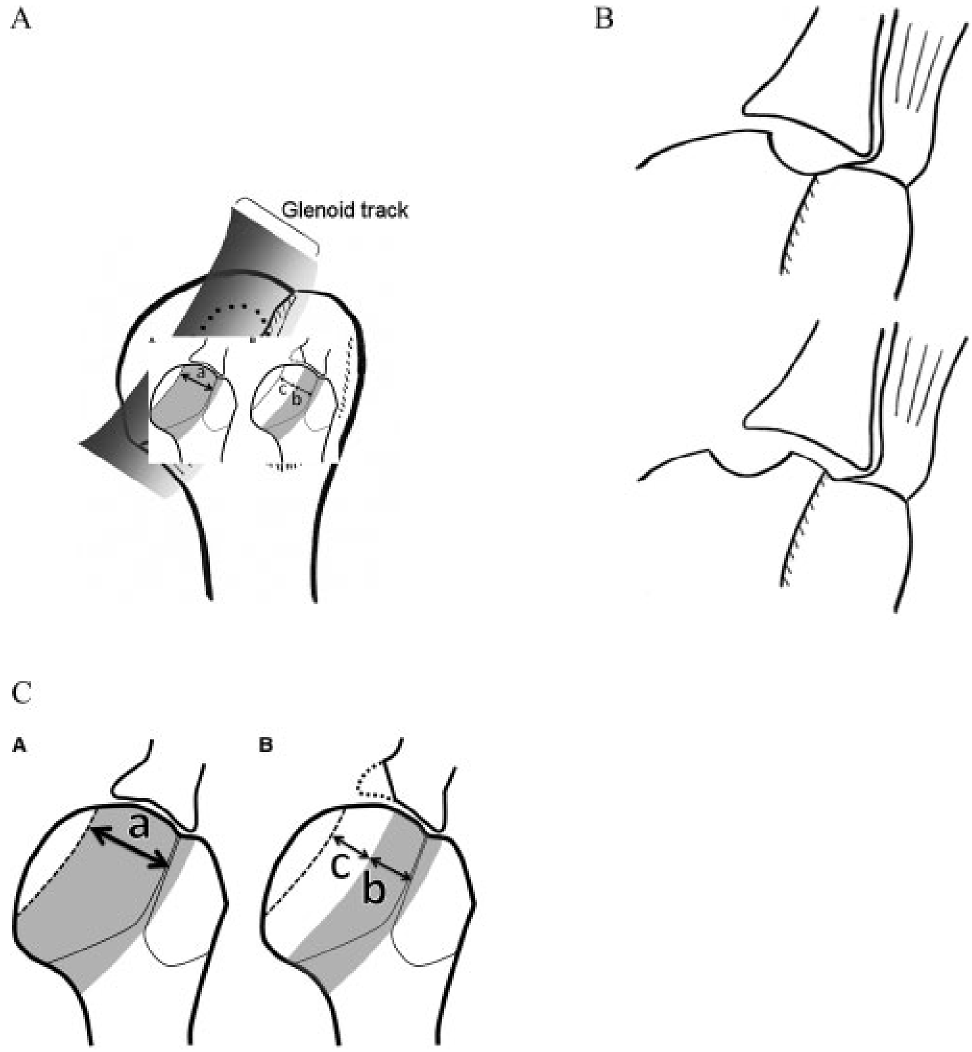

Fig. 2. A–C: Glenoid Track.
A: The glenoid track as mapped through the elevation and external rotation. B: If the Hill Sachs lesion falls within the glenoid track, in is an "on track" lesion and unlikely to engage in range of motion whereas those medial to the glenoid track, are "off track" lesion and likely to engage. With permission.[24] C: With glenoid bone loss, the glenoid track becomes narrower, and therefore a Hill Sachs lesion that was considered "on track" with no glenoid bone loss could become "off track" and more likely to engage. With permission.[25]

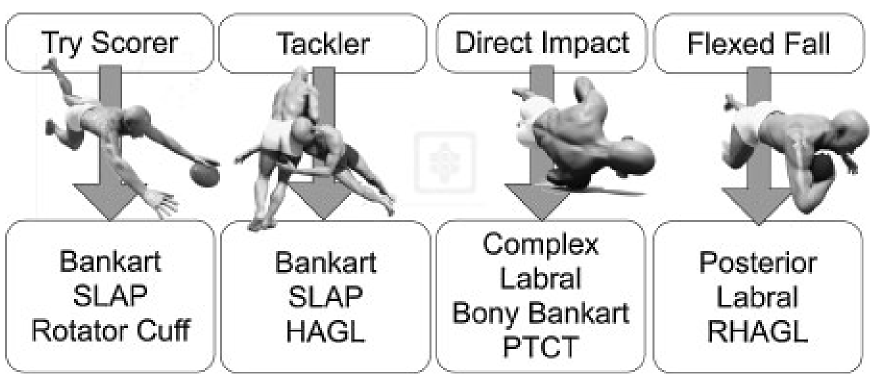

Fig. 3. Injury mechanism.
The common mechanisms of injury in collision sports, demonstrating the injury patterns that often follow.

rather than frank dislocations and the absence of an index injury does not preclude a diagnosis of instability. A subluxation is best described as translation of the glenohumeral joint beyond physiological limits not requiring manual relocation, or subluxation events may have been dislocations that self-reduced rather than required a manual reduction.[16] Subluxation events may be reported as a dead arm symptoms, pain, or weakness that would not necessarily stop them from completing a match.[27] Athletes can often play the season, but be troubled during training and often report pain after the game.[26]

Patients should be assessed for degree of generalised ligamentous laxity. This can be assessed by the criteria described by Wynne Davies[28] or by Beighton.[29] Laxity is a clinical sign of increased joint translation and is different to instability, which is a patient reported symptom. Laxity around the shoulder can be clinically assessed using the load and shift test and the sulcus sign.[30] The integrity and laxity of the inferior glenohumeral ligament can be assessed by the hyperabduction test. Gagey and Gagey found that passive hyperabduction beyond 105°, especially with passive abduction less than 90° on the unaffected side correlated with increased laxity of the inferior glenohumeral ligament complex.[31]

Anterior instability is assessed via the apprehension test, relocation and anterior release tests. These tests were found to have improved sensitivity and specificity when apprehension rather than pain was used as the definition for a positive test.[32],[33] If the apprehension test is positive in midrange abduction, from 30 to 90°, and less external rotation than the standard test, 90° abduction and external rotation, this is often indicates significant glenoid bone loss.[34] Gerber and Ganz advocated the anterior draw test, assessing the degree of anterior displacement of the humeral head compared to the fixed scapula in the supine position to grade the degree of subluxation.[35]

Posterior instability in these athletes is assessed by the Kim test,[37] the Wrightington Posterior Instability Test (WPIT)[38] and the posterior apprehension test. The dynamic labral shear test[36] is also useful in athletes, in combination with the tests above for superior and posterior labral pathology. Standard assessment of the rotator cuff is vital, along with cervical spine and neurological assessment of the upper limb.

5. Investigations

Plain radiographs still have a role in the investigation of anterior shoulder instability. They have been shown to be able to detect glenoid and humeral defects, as well as provide accurate measurements of these defects.[36],[37] Specific views for glenoid bone loss such as the Bernageau, West Point and Didieé[38] have been described. CT scans, which include three-dimensional reconstructions with humeral subtraction, have now become the commonly accepted standard for assessment of the glenoid in respect to fracture and bone loss[39–42] (Fig. 4).

The assessment of the capsulo-labral complex is best performed with an MRI. An MRI arthrogram (MRA), with an intra-articular injection of gadolinium, has been shown to improve the accuracy, however a recent meta-analysis has found the difference between the two modalities to be “marginal”.[43] MRI accuracy of the anterior band of the inferior glenohumeral ligament (AIGHL) can be improved by imaging the shoulder in abduction and external rotation (ABER). This ABER view was shown to have a sensitivity and specificity of 94% and 82% respectively in detecting injury to the AIGHL.[44]

6. Management

The management of anterior shoulder instability in the contact and collision athlete must take a number of issues into account, with the goal of any treatment being the restoration of stability, maintenance of function and return to sport.

6.1. Non-surgical

Initial non-surgical treatment can vary, with the position and length of immobilisation controversial. Radiological studies have shown that the anterior labral structures are reduced better with the arm in external rotation compared to internal rotation,[45],[46] however the clinical outcomes of external rotation slings compared to internal rotation slings have yielded mixed results.[47],[48] A recent review by Whelan et al in failed to find a difference in the recurrence rate between those in a standard internal rotation sling and an external rotation sling.[49] The period of immobilisation has also not been clarified, with 2 year recurrence rates being no different between those immobilised for extended periods of time compared to early mobilisation.[50]

The risk of recurrent instability of the shoulder after non-operative treatment has been shown to be much higher in young, male contact and collision athletes.[51–55] Three systematic reviews have shown that non-surgical options have yielded high failure rates in contact and collision male athletes,[56],[57] with this failure rate above 70% in those under the age of 18.[58]

Non-operative treatment might be preferred for the management of the athlete suffering an in-season shoulder dislocation. Some patients can successfully return to sport to complete the season, but in one study 37% of them suffered a repeat instability event.[59] Indications for non-operative versus operative intervention in this situation have been proposed based on the risk of recurrence and the ability for the athlete to perform sport-specific drills.[60],[61] The aim of the management in this situation is to minimise further instability events until the off-season when potential surgery could occur.

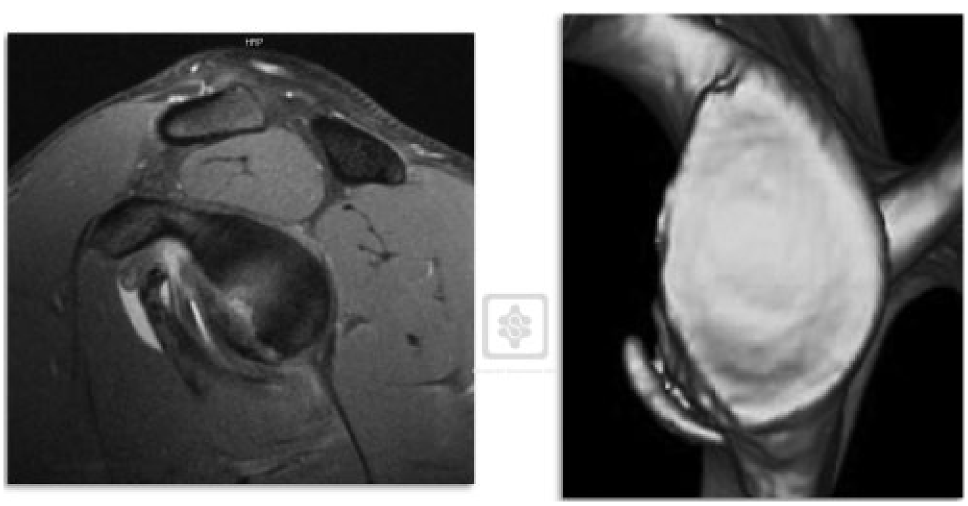

Fig. 4. Bone loss.
MRI arthrogram and CT demonstrating anterior glenoid bone loss commonly encountered in collision atheletes.

6.2. Surgical

Surgical intervention has been shown to lower the high recurrence rate and therefore surgery is often the preferred option for this patient group. Surgical intervention broadly consists of anatomical Bankart repair, bone transfers to the anterior glenoid and surgery to address the Hill Sachs lesion. Each of these procedures can be performed arthroscopically or open.

6.2.1. Bankart repair

Traditionally soft tissue labral repairs were performed using open techniques and required an approach through subscapularis.

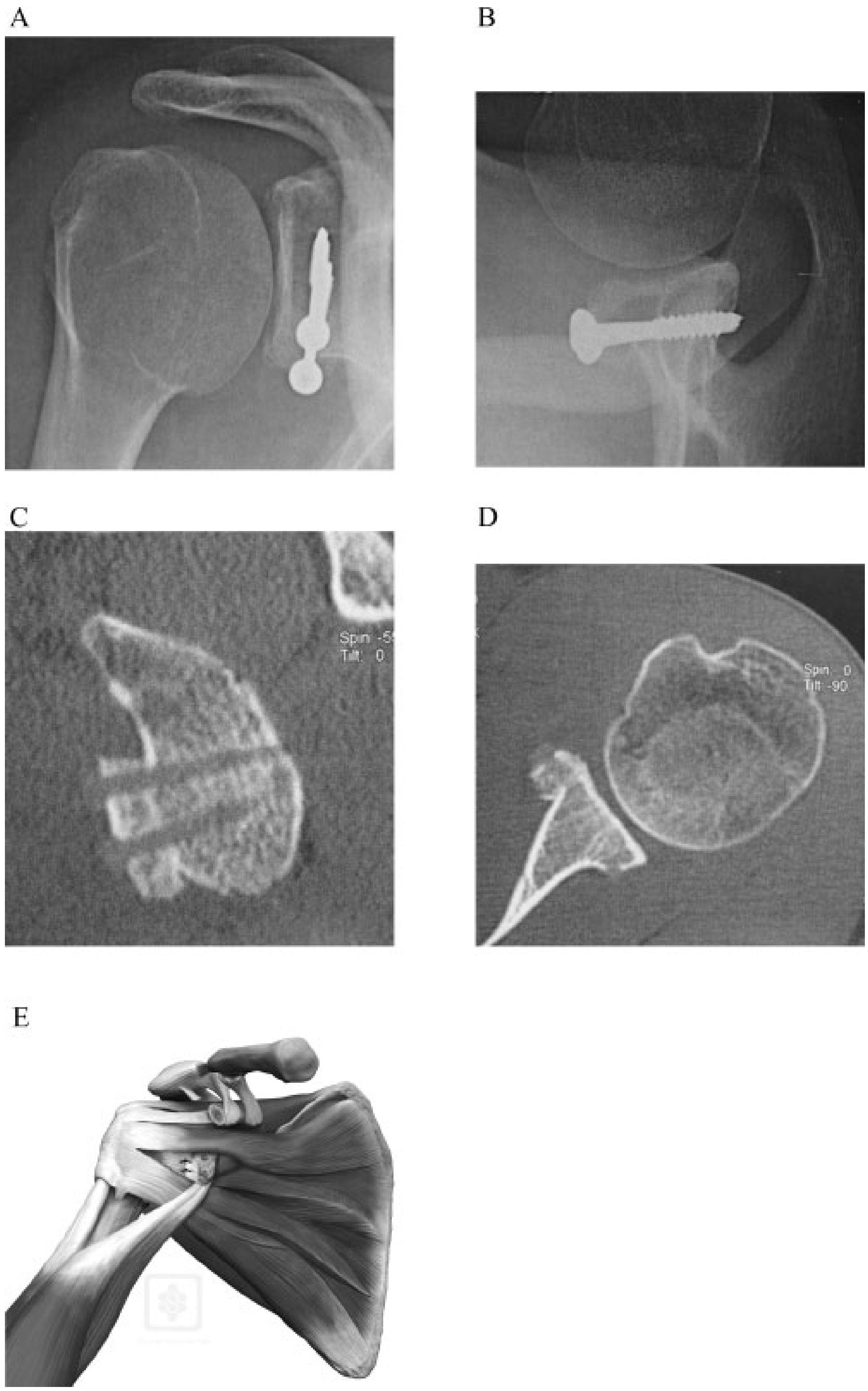

Fig. 5. A–E: Latarjet and principle of action.
A-D: Radiographs and CT showing a united Latarjet procedure and the "bony effect" of the procedure E: The coracoid graft positioned with capsular repair, demonstrating the "sling effect" via the conjoint tendon and subscapularis as well as the "capsule effect" via repair of the capsule to the graft.

The better visualisation and limited soft tissue compromise made the procedure well suited to arthroscopic surgery. However early arthroscopic techniques had a high failure rate compared to open repairs.[62,63] Technological advancements have facilitated stronger and more anatomical repairs with suture anchors and now show comparable results to open repairs in most series.[64,65]

Risk factors for failure of an arthroscopic Bankart repair include male patients, age less than 22, more than 3 previous dislocations, surgery performed in the beach chair position compared to lateral decubitus, use of less than 3 suture anchors and engaging Hill Sachs lesions or significant glenoid bone loss.[65–71] Balg and Boileau tried to aide surgical decision making with the Instability Severity Index Score, or ISIS.[72] By assigning points based on the patient's age, degree and type of sport participation, hyperlaxity and glenoid bone loss or humeral head defect, they found a score of 6 or less led to recurrence rates of 10% with scores of more than 6 leading to recurrence rates of greater than 70%. They concluded that patients with an ISIS score of greater than 6 are better managed with the Latarjet procedure. However, a follow-up study was not able to confirm its accuracy in detecting failure of arthroscopic Bankart repairs.[73]

In a comparison between contact and collision athletes who underwent arthroscopic stabilisation after the first shoulder dislocation Ranalletta et al found a 0% recurrence rate at 2 years for the contact group, but a 14.7% recurrence in the collision group, made up primarily of rugby players. Cho et al also found a recurrence rate of 28.6% in collision athletes compared to 6.7% in the non-collision group.[74] They also found that contact athletes returned to sport quicker and with more returning to their pre-injury athletic level compared to collision athletes.[75] The difference in specific sports is also shown in the adolescent population, where a study of contact and collision athletes, not including rugby, had a recurrence rate of 10.3% at over 6 years follow-up,[76] whereas a series of primarily adolescent rugby players found a recurrence rate of 31% at 4 years.[77] Another series found a 21% recurrence in contact and overhead adolescent patients at 5 years follow-up.[78]

Glenoid bone loss has been repeatedly shown to be associated with high recurrence rates after arthroscopic Bankart repairs. De Beer and Burkhart found that in their patients with bone loss >25% they had a recurrence rate of 67%, and 89% in contact athletes, with an arthroscopic Bankart repair compared to 4.9% with an open Latarjet procedure.[19,23] Glenoid bone loss has been shown to be more common in patients with recurrent dislocations and in those who have their first dislocation at a younger age.[18] Different contact sports may also tolerate different amounts of bone loss, with Nakagawa et al finding rugby athletes had a high recurrence rates after arthroscopic Bankart repairs with any bone loss, whereas other contact and collision athletes they could tolerate 10% bone loss before the recurrence rate significantly increased.[79]

6.2.2. Bone procedures – Latarjet, Bristow and variants

Anterior glenoid bone block procedures have been shown to have a low recurrence rates in contact and collision athletes, including rugby.[80–82] The most commonly used is the Latarjet coracoid transfer but similar procedures such as the Bristow (coracoid tip) or Eden-Hybinette (iliac crest graft) are also used with good results. Anterior glenoid bone blocks have been shown to be effective in the setting of glenoid bone loss and Hill Sachs lesions by Burkhart and De Beer.[23] The "triple blocking effect" and the biomechanics of the Latarjet have been described in detail[83,84] (Fig. 5). The Latarjet being found to be superior to the Bristow in the setting of glenoid bone loss[85] as well as being effective in the management of Hill Sachs lesions.[86] A recent meta-analysis comparing the bone block procedures to an arthroscopic Bankart repair found a significantly lower recurrence, 11.6% compared to 21%, and redislocation rate, 9.5% for Bankart procedures compared to 5% with the bone block procedures.[87] Whilst another review of the published Latarjet results found a repeat subluxation or dislocation occurring in 7.5% of patients, with a reported range of 0–19.1%.[80] In our own group of contact athletes we found a recurrence rate of 3% and return to sport of >95% treated with the Latarjet.[88]

There have been concerns raised by the high number of complications associated with these procedures in some studies,[89,90] however this has not been borne out in all review articles.[80] Indications, patient selections and surgical techniques have been discussed in various articles, aimed at decreasing the complication rate and improving outcomes for the open Latarjet procedure.[91,92] Graft malposition is also a key step at avoiding complications, with grafts positioned too medial leading to higher redislocation rates and too lateral leading to glenohumeral arthropathy.[93,94]

6.2.3. Other procedures

There has been an increase in the interest in the management of the Hill Sachs lesion, and especially "off track" lesion, in the humeral head.[95,96] Whilst the Latarjet has been used for this lesion, other options include allograft reconstruction, partial resurfacing arthroplasty, hemiarthroplasty or remplissage.[97] Giacomo has proposed that patients with "off track" lesions and no bone loss should be treated by a remplissage,[96] however the role of this in the contact athlete is unclear.

Other injuries that may need to be assessed include a humeral avulsion of the glenohumeral ligament, or HAGL, which may be anterior or posterior, as well as capsular ruptures and tears. HAGL's have been reported to have high recurrence rates with non-operative treatment but can be successfully treated by both open and arthroscopic techniques.[98,99] With capsular tears however, some authors have found high recurrence rates even with arthroscopic repair, leading to concerns about the best way to treat these lesions.[100]

Rotator cuff tears can also occur in the setting of shoulder instability, and they always require surgical repair. Some surgeons report good results with a two-staged procedure to address the pathology, with the rotator cuff treated first and the instability addressed at a later stage.[101] Others have managed to treat the patient's instability with an arthroscopic Bankart repair to allow them to return to sport and performed a rotator cuff tear in the off-season.[102] We prefer to address both pathologies at the same time, to minimise the time off sport and aide recovery.

7. Summary

Anterior shoulder instability is a complex issue in the contact and collision athlete that requires an individualised approach, taking into account the patients, level and type of sport, time of the season and structural injuries around the shoulder. of choice as well as assessing their risk of recurrence is vital to ensure the correct management decision is made. Surgery should address all the relevant pathologies. This approach, with multidisciplinary input, is vital to ensure the correct management decision is made to ensure a safe return to sport and minimise the risk of recurrence and complications.

Conflict of interest

Dr. Mattern has nothing to disclose.

Dr. Funk has nothing to disclose.

Dr. walton reports personal fees from Arthrex, personal fees from LIMA, personal fees from Medarits, outside the submitted work.

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Latarjet procedure: Current concepts and review

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ABSTRACT

The Latarjet technique[1] sometimes also referred to as the Bristow- Latarjet, or Latarjet -Patte procedure is a well-known for treating anterior shoulder instability.

This procedure involves transfer of coracoid with attached conjoint tendon to anterior inferior aspect of glenoid rim via a split in the subscapularis muscle.

There is renewed interest in this procedure due to failure of soft tissue stabilization techniques in the long term, improved understanding of chronic shoulder instability, improved techniques involving fixation as well as doing it arthroscopically. Over the years, the original Latarjet technique has undergone various modifications and adaptations.

This article traces its history, technique and evolution over the years. It also provides the indications, contraindications and complications. It also highlights the trends of latest developments and current literature.

Contents

1. Introduction

In 1954, Latarjet, first described this technique for recurrent anterior shoulder dislocation. He treated them by transferring and fixing coracoid process with conjoint tendons (detaching upper part of subscapularis muscle) to the anterior margin of glenoid. He used a screw for fixation. Around the same time, Helfet described a similar procedure called the Bristow procedure where the transferred coracoid was only sutured to the subscapular tissue. The modification of Bristow procedure to use a split in the subscapularis and screw fixation was the same as Laterjet's. Hovelius et al popularized it in Sweden in late 70 s and published early to mid term results in their series of 112 cases with 90% excellent results. They used two screws for fixation[2] Patte explained the reasons for successfully stabilizing the shoulder joint by virtue of 'triple blocking effect'. They were

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1 Physical Extra articular bone graft increasing the glenoid surface area, which provides stability in midrange of motion.
2 The dynamic sling of the conjoint tendon attached to coracoid to support the humeral head during overhead abduction, which stabilizes the shoulder in mid range and end range glenohumeral abduction and external rotation.
3 The Bankart effect of repairing capsulolabral complex to the bone, which provides end of range of abduction and external rotation stability.

1.1. Indications of Latarjet procedure

The indications for the Latarjet Procedure have evolved since it was first described. Giacomo et al[3] suggest Latarjet+/- humeral procedure as a recommended treatment for patients with >=25% of glenoid defect with an on or off track Hill-Sach's lesion.

Similarly, Balg, Boileau et al[4] described the Instability severity Index Score (ISI Score) to help in preoperative determination of arthroscopic vs open shoulder stabilization. They recommend, Latarjet procedure in patients with a preoperative ISI Score of >3 with isolated glenoid or combined glenoid and humeral bone defects.

In high impact elite athletes it can be used as a primary reconstruction procedure with minimal bone loss[5]

2. Contra indications of Latarjet procedure[6]

1 1 Recurrent Anterior instability associated with massive irreparable cuff tear in the >50 year age group. The authors caution against the potentially increased risks of graft fracture and non union in the older population. They have also observed the complication of static instability of humeral head and progressive osteonecrosis due to contact of humeral head with the bone block. They also observed another complication of irreducible inferior humeral head subluxation due to the non elastic part of the subscapularis being pulled down by the transferred coracoid with conjoint tendon.
2 Traumatic dislocations in the older population with or without large glenoid rim fracture. This is also a relative contra indication due to reasons above. If the Humeral head is centered over the glenoid, conservative management is successful even with a displaced glenoid fragment.
3 Uncontrolled epileptic patients. With uncontrolled seizure disorders, there is a risk of fracture of the transferred coracoid, or displacement leading to non union or failure of surgery.
4 Habitual anterior dislocators/subluxators. The tissue laxity in this patient subgroup may not be controllable by the Latarjet procedure and the literature reports poor results.
5 Anterior instability of Prosthesis. There is no evidence in the literature to support Latarjet in this situation, and Reverse Shoulder gives a more reliable outcome.
6 Young athlete with painful shoulder and micro instability. This scenario can exist in a throwing athlete with hyperlaxity. The Anterior apprehension is reproducible in the ABER (Abduction External Rotation) position. Usually a very careful evaluation and tests are essential to ensure correct diagnosis and arthroscopic soft tissue stabilisation procedures should suffice.
7 Chronically locked anterior dislocation. There is a risk of osteonecrosis of humeral head and osteoarthritis. These cases are best treated non operatively or using a reverse shoulder replacement if symptomatic.

3. Key steps in operative technique[7]

Many steps and pearls have been authored in the literature according to the preferred technique of the surgeon.

Attention to detail during essential steps of the procedure helps achieve an excellent outcome while avoiding the pitfalls

Positioning: Beach chair position with free draping of the shoulder and arm helps the maneuverability of shoulder. It also helps with intraop access to imaging during screw placement. An arm holder or a Mayo table to support the arm is an additional armamentarium in the surgeon's arsenal

Approach: Deltopectoral approach with meticulous hemostasis and preservation of coracoid blood supply (along the medial aspect of conjoint tendon), appropriate use of retractors and having good anaesthesia to facilitate the surgery in frequently muscular patients.

Coracoid Graft Harvest: It is important to visualize Coracoid well with a superior retractor. This will help plan the osteotomy anterior to the coraco-clavicular ligaments near the base. The CA ligament also needs identification and transected leaving 1–2 cm of it attached to the Coracoid, to facilitate capsular repair. Pectoralis minor release subperiosteally helps mobilise the Coracoid after harvest. Digital palpation of axillary and musculocutaneous nerves throughout helps avoid inadvertent damage. Marking the osteotomy site and measuring with a ruler ensures adequate length (approximately 2–2.5 cm). Protecting the base of coracoid medially, laterally and inferiorly subperiosteally with chandler retractors help during osteotomy. A 90 degree oscillating saw helps harvest the graft without risking glenoid fracture. Gentle release of adhesions of soft tissue and around the musculocutaneous nerve with swab or digital palpation helps mobilise the graft. Prepare the inferior surface of the graft by decorticating it so that it will incorporate well with the glenoid post transfer.

3.1. Glenoid exposure and preparation of graft bed

Transverse dissection of superior 2/3 and inferior 1/3 of subscapularis to gain access to the anterior glenoid. Single prong Gelpi retractors to retract subscapularis and visualize capsule. T or L shaped capsulotomy as per surgeon preference. Tagging of capsule with suture for ease of repair later helps. After inferior labrum is retracted, the anterior glenoid neck comes into view. Fukuda retractors will help safely retract humeral head to improve access. Abrasion of bone bed over the antero-inferior neck of glenoid is done to bleeding bone to help with incorporation of graft.

3.2. Coracoid transfer and fixation

Placing the coracoid flush to the anterior glenoid rim. It is placed longitudinally ideally from the 3 to 5' o clock position. Some commercially available holders (for eg from Arthrex, Naples, FL) help hold the coracoid securely and orient it correctly with access for pre-drilling the screw holes within the graft at an adequate distance. Definite screw fixation using cannulated or solid 3.5 or 4.0 mm lag screws with bicortical purchase is achieved as per surgeon preference with image intensifier guidance and direct visualization to avoid intra articular screw placement. Avoid over tightening of screw to prevent splitting of graft. Check for proper graft position to avoid medialisation or lateralization. Burr can be used to smoothen any sharp prominent edges of the graft.

3.3. Capsule and subscapularis repair

A pre loaded suture anchor could be used at the graft, glenoid interface to repair the capsule apex of the T or L incision and the graft becomes extracapsular. The Stump of CA Ligament also helps provide tissue for securing the repair. Positioning arm in a degree of external rotation during suturing avoids over tightening of repair and post op rehabilitation without loss of external rotation.

4. Rehabilitation following latarjet procedure

Since Latarjet procedure is becoming a procedure of choice by surgeons for patients with bony lesions or for revision surgery it is paramount to understand the anatomic aspects of surgical intervention, outcomes, complications and post-surgical rehabilitation regime. The Latarjet procedure is significantly different in terms of rehabilitation following a Bankart repair. The bony union of coracoid is essential before the shoulder can be subjected to stressful environment. The subscapularis muscle suffers significant surgical trauma and subsequent immobilisation can lead to significant rotator cuff atrophy and weakness. The post-operative regime and rehabilitation programme should involve multiple disciplines including Physiotherapy, Pain management and Shoulder surgeon. Most rehabilitation protocols recommend immobilisation of the arm in a sling during the initial 3 weeks after surgery with passive shoulder abduction and external rotation. As the coracoid is uniting with glenoid, it is important to protect the soft tissues especially the attachments of biceps and coracobrachialis by limiting shoulder extension and excessive external rotation. At 6 weeks post-op the patient can discard sling support and proceed with active assisted motion in all shoulder ranges to active range of motion as tolerated. Shoulder strengthening exercises commence at 6–8 weeks with scapular stabilisers posteriorly and subsequently include anterior musculature including subscapularis, pectoral muscles and biceps through 8–12 weeks. The patient can return to full functional range of movements at weeks 12–16[8].

5. Complications of the Latarjet procedure

In a systematic review of 24 studies, Cowling et al (BJJ 2016)[9] have mentioned about the following complications

Mean graft non-union rate – 3.36% (0–5.7%) This can happen due to older patient, smokers, graft malpositioning, early aggressive rehabilitation. This can be avoided by preparing good bleeding bone bed reciprocal surfaces, using a long graft to increase surface area, and good bicortical compression perpendicular screws for stable fixation.

Screw Breakage rate mean – 9.74%(0–26%) This can be avoided by avoiding graft malpositioning, solid screws with larger root diameter.

Mean infection rate – 1.34%(0–6.67%). Infection could be a cause of failure of bone block and recurrence of instability. This is avoided by meticulous hemostasis, good surgical technique avoiding tissue devitalisation and good soft tissue handling. Infection is managed in most cases with irrigation, debridement and appropriate antimicrobials. Severe infection may necessitate, removal of metalwork, prolonged intravenous antibiotics with microbiology advice

Neurological Injuries (mean) - 0.51% (0–3.33%). This complication is best avoided by avoiding excessive stretching of tissues by self-retainers. Excessive dissection around the medial aspect of coracoid is best avoided. Nerve injuries are managed conservatively with good recovery in majority but appropriate investigation and referral may become necessary if there is failure to improve.

Mean recurrence rate - 5.36% (2.9%–43%).

This is best avoided by careful patient selection, meticulous surgical technique while preparing graft and the graft bed, graft positioning and fixation. Graft should ideally be positioned below the equator of glenoid in sagittal plane and flush to articular surface in the axial position. Management depends on time of presentation (early or late), whether graft if viable, fractured or resorbed.

Other complications which could be encountered are

Bone Block fracture due to poor quality of bone, intra op over tightening, and excessive decortication of bone. It can be minimized by using careful ‘finger ‘tightening of screw, using pre drilling of holes appropriately spaced apart on graft. Intraoperative fracture is managed by smaller screws, use of a washer or buttress plate, or Iliac crest graft.

5.1. Bone block resorption

It has been frequently observed but rarely leads to ongoing apprehension. This can be minimized by avoiding graft devascularisation by limiting soft tissue releases of the graft tip. Usually this is not a problem unless prominent metalwork requires removal due to abutment with humeral head.

5.2. Osteoarthritis

The literature suggests 20–25% of post-operative osteoarthritis. Preoperative osteoarthritis, old age at 1st dislocation, glenoid rim fracture, long postoperative delay, severe Hill Sachs lesion and high demand sports are risk factors. Intra articular screws, prominent screw heads and laterally overhanging coracoid process are also an easily avoidable cause. Lateral coracoid over hang could be addressed by refixation or burring of the prominent edges of the graft. Capsular re attachment and extra articular graft placement could minimize the risk of osteoarthritis.

6. Current evidence and trends

Since its description in1954 by Latarjet, the procedure itself has undergone significant modifications. Originally described as an open procedure, it is increasingly being carried out arthroscopically.

This has been possible due to improvement in arthroscopic skills amongst shoulder surgeons. In a study published recently, the shoulder arthroscopy procedures performed per resident increased by 43% from 2007 to 2013 in the USA[10]. This clearly shows the increasing exposure of residents to arthroscopic techniques.

Lafosse et al[11] first described the arthroscopic version of Latarjet procedure in 2007. Dumont et al[12] reported on 5-year follow-up of arthroscopic Latarjet in 2014. In this study of 64 patients, mean age at surgery was 27.1, mean number of dislocations was 5.3 and mean follow-up was 6.4 years. One patient underwent a total shoulder replacement procedure and of the remaining 63 patients there were no reported dislocations following surgery. One patient reported having subluxations. As regards complications, 3 patients had a haematoma, which resolved. 15.6% patients returned to operating room after arthroscopic Latarjet. 1 patient returned for a displaced coracoid graft that was successfully repositioned, 8 patients had prominent screws that were removed and 1 patient underwent a TSR procedure for glenohumeral arthrosis. The authors concluded that the rate of recurrent instability after arthroscopic Latarjet procedure for treatment of anterior shoulder instability is low.

However we would caution that the procedure remains technically demanding and complications are high at the present times.

The Latarjet procedure has been a procedure of choice by shoulder surgeons mainly in the presence of bone loss or as a revision surgery. The procedure addresses soft tissue as well as bony lesions that affect the anteroinferior glenohumeral joint.

The Latarjet procedure has been compared with Bankart repair for treatment of recurrent instability by VV An et al[13].

In this systematic review and meta-analysis the authors reported on 8 comparative studies with 795 shoulders. 416 patients had open or arthroscopic Bankart repair, and 379 underwent open Latarjet procedure. In the 8 studies reported 6 employed open Bankart repair and 2 employed arthroscopic

Bankart repair. All Latarjet procedures were open procedures. The mean follow-up ranged from 4.9 to 17.5 years. The mean age of patients undergoing Bankart repair was 26.4 years and the mean age of patients undergoing Latarjet was 26.9 years. In the 8 included studies 4 reported recurrence as an outcome. Recurrence was reported in 21.1%and 11.6% of Bankart and Latarjet procedures respectively. This difference was reported to be statistically significant with approximately 2-fold higher risk of recurrence in Bankart cohort.

The Latarjet procedure is a viable alternative to Bankart repair and is reportedly superior in some cases particularly in presence of bone loss, high impact sports and revision stabilization. The Latarjet procedure has less recurrence and revision surgery in comparison to a Bankart's repair with no significant difference in the rate of complications.

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Remplissage as a concept and role in instability

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ABSTRACT

Anterior instability of the shoulder is characterised by bipolar lesions – the Bankart lesion with glenoid bone loss on 'glenoid side' and the Hill- Sach's defect (HSD) on the 'humeral head'. It is well known that the recurrence of the dislocation is likely to be more frequent even after surgical repair in the presence of these lesions. Currently, by and large, two surgical options are available to address the glenoid side lesion: Bankart repair (soft tissue procedure) or Latarjet procedure (Bony procedure). Both the procedures have stood the test of time and have proved to be reliable and reproducible. Based on the amount of glenoid bone loss (roughly, the cut off at 25%) choice of surgery offered for this condition is reasonably straight forward. Bankart repair (Arthroscopic/open) for <25% bone loss and Latarjet for>25% bone loss is widely practiced. Although the Hill-Sachs defect remains very frequent in almost all cases of anterior instability of the shoulder, the role and management offered for Hill-Sach's defect remains debatable and controversial. Various surgical procedures have been described to correct Hill Sach's defect include filling of the defect with infraspinatus (remplissage) or bony procedures like reconstruction with an osteoarticular humeral head allograft or partial resurfacing arthroplasty. This review discusses the role of Remplissage procedure with respect to Bankart repair in cases with medium to large Hill Sachs defects.

Contents

1. Introduction

The management of patients with anterior shoulder instability continues to evolve as the understanding of the role of soft tissue and bony restraints have become more evident in the recent past. Almost all cases of recurrent anterior shoulder instability have bipolar loss namely, glenoid side (Bankart lesion with or without glenoid bone loss) with Hill-Sach's defect. Hill-Sach's defect (HSD) is a posterolateral compression fracture of the humeral head that occurs when the glenoid edge impacts on the humeral head during an anterior dislocation. In recurrent dislocations, the prevalence of glenoid erosion or rim fracture ranges from 8% to 95% whereas HSD are seen in almost 100% of the cases.[1] Burkhart and De Beer drew our attention towards significant bone defects especially on the glenoid side contributing to recurrent instability after repair.[2] Since then, much work has been done on the consequences of glenoid bone deficiency on the anterior instability especially in situations such as failure after the arthroscopic Bankart repair.

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Currently, it is established that a glenoid bone loss more than 20–25% poses a high risk for re-dislocation and should be addressed 'primarily' with a bony procedure such as the Latarjet procedure to counter the failure of Bankart repair.[3] The cases with less than 20–25% glenoid bone loss remain the subject of debate as to whether they should be primarily managed with soft tissue Bankart repair or Latarjet procedure with proponents and opponents on either side. Recently, Gerber et al. published their results on 364 shoulders who underwent Bankart repair (271 shoulders) or Latarjet procedure (93 shoulders) with minimum follow up of six years.[4] In their series, those who underwent Bankart repair reported high incidence of 'feeling of instability' or 'overt instability' by 41.7% and 28.4% respectively. This highlights that mere Bankart repair is not enough to restore shoulder stability in the long term especially if there is a bipolar bone loss.

Nevertheless, the management of HSD in anterior instability has remained controversial for a long time as only larger size lesions were given due attention whereas small to medium size lesions were mostly ignored. HSD was always looked upon as a separate entity. Traditionally, the literature has been more focused on the pathology, diagnosis, and management of anterior bony and soft tissue deficiencies in anterior instability rather than HSD. This review intends to look into the HSD, its biomechanical influence upon the shoulder stability and 'Remplissage' as an emerging option for the management of the same.

2. The influence of Hill Sach's lesion on shoulder biomechanics

In the event of a shoulder dislocation, there is a bipolar injury wherein there is damage to the anteroinferior glenoid labrum as well as HSD on postero lateral aspect of the humeral head. Bigliani et al. classified the size of HSD as small, medium and large when the size of the defect is less than 20%, 20–40% and more than 40% of the humeral head size respectively.[5]

The prevalence of bipolar bone loss is reported to be 64%–70% in first time dislocators and 79%–84% in recurrent dislocators.[6,7] Sekiya et al. demonstrated that HSD as small as 12.5% might affect the biomechanical stability of the shoulder.[8] Furthermore, authors confirmed that these HSD could 'engage' with anterior glenoid defect in 90° abduction and any degree of external rotation of the shoulder. Burkhart et al. concluded that the rates of re-dislocation after primary Bankart repair in "engaging significant bone defects" in a contact athlete could be as high as 89%.[2] Boileau et al. too confirmed that a large HSD could result in failure of Bankart repair.[9] However, the critical size and orientation of HSD that could influence recurrence after primary dislocation or failure after the soft tissue repair remains debatable. Yamamoto et al. introduced a new idea of 'glenoid track' after studying the relationship between the glenoid and Hill-Sach's lesion in cadaveric shoulder model with the arm in the complete external rotation, horizontal extension and varying degrees of abduction in a 3D CT scan. With increasing elevation of the arm, more glenoid contact area moved from the inferomedial to the superolateral portion of the posterior articular surface of the humeral head, thus developing a dynamic contact zone between the glenoid and the humeral head. This contact zone on posterolateral humerus has been labeled as 'glenoid track'. The medial margin of the glenoid track was located 18.4 ± 2.5 mm medial from the footprint of the rotator cuff, which was equivalent to $84\% \pm 14\%$ of the glenoid width. Further, they concluded that if the HSD lies within the track, it is known as 'on-track lesion' and such a HSD would not engage with the anterior glenoid rim. However, if HSD lies medial to the track, it is known as 'off-track lesion,' and there may be the engagement of such an off-track HSD that would render a shoulder unstable.

Di Giacomo et al. suggested that converting an 'off-track' lesion into 'on-track lesion' is mandatory to restore the stability of the shoulder.[10] Regarding the size of the defect; most authors agree that HSD < 20% can be managed conservatively whereas those over 40% certainly need operative intervention. However, the management of HSD of 20–40% defect remains debatable. There are multiple methods to address the HSD like Remplissage, osteochondral bone graft, rotational osteotomy, transhumeral bone impaction or rarely arthroplasty for larger defects. Osteochondral grafting is reserved for the cases with large Hill Sach's defect. Rotational osteotomy is rarely performed as it can result in non-union, delayed union and loss of motion. Remplissage (French; to fill) is an procedure of filling the HSD with soft tissue (infraspinatus + posterior capsule), thereby preventing the engagement of the defect with the glenoid rim. It was initially described by John Connolly, which was later popularised by Wolf et al. using the arthroscopic technique.[11] More recently, the Remplissage has emerged as a popular option for engaging HSD especially those between 20% to 40% of humeral head size.[12]

The aim of remplissage is twofold: (1) Intraarticular lesion converts into extraarticular and thus does not engage with the anterior glenoid margin (2) It acts as a checkrein against anterior translation of the humeral head. The major cited disadvantage of remplissage is that it is a non-anatomical procedure wherein the infraspinatus is tethered onto the HSD region, and may result in loss of movement, especially rotations.[13]

3. Current indications of remplissage

Somewhere between the isolated Bankart's repair and Latarjet procedure for glenoid side lesion, there is void in treatment approach for shoulder instability with moderate to large HSD lesions. Currently, the remplissage is indicated in off-track lesions where glenoid bone loss is less than 25%. Hartzler et al. conducted an interesting cadaveric experiment on eight shoulders and concluded that mere Bankart repair in shoulders with off-track HS lesions would not prevent engagement especially in end range rotation. However, adding remplissage prevents engagement of off-track HSD in all cases in end range rotation.[14]

However, Bankart repair with Hill-Sach's remplissage resulted in supraphysiological stiffness for off-track lesions at mid and end-range rotation. Remplissage is indicated for restoring biomechanical stability in anterior instability with bipolar loss despite a risk of stiffness.

Giles et al. performed an in vitro biomechanical comparison of the remplissage procedure, allograft humeral head reconstruction, and partial resurfacing arthroplasty for moderate to large engaging Hill Sachs lesions.[15] They observed that the remplissage procedure was effective at preventing Hill- Sachs lesion engagement; however, it also significantly restricted some shoulder motions. The remplissage procedure also increased joint stiffness compared with the other reconstructions models. More recently, Longo et al. performed a systemic review of 26 level II–IV studies with 769 shoulders reporting on various interventions for shoulder instability with bone loss. They concluded that combination of remplissage and Bankart procedures was associated with a lower rate of recurrence when compared with Bankart repair alone and that remplissage was the safest technique for the management of patients with shoulder instability with the humeral bone loss.

4. Different techniques of remplissage

Initially, the technique of filling the defect described by the pioneers such as Wolf et al. and Purchase et al. involved placement of the anchor into the valley of the defect and performing posterior capsulodesis and tying down the infraspinatus.[11,16] In 2009, Koo et al., described a modification for filling the Hill Sachs defect.[17] The remplissage sutures are tied in the

subacromial space over the infraspinatus by use of the transtendon double-pulley technique. This technique uses the eyelets of the two suture anchors as pulleys and creates a double-mattress suture.

Recently, Elkinson et al. conducted a biomechanical study on three different techniques of performing the Remplisage technique: T1, anchors in the defect valley; T2 anchors in the humeral head rim; and T3, anchors in the valley with medial suture placement. Outcomes included stability, the internal-external rotation range of motion, and joint stiffness. They concluded that all remplissage techniques enhanced shoulder stability, restricted ROM, and increased joint stiffness.[18] Medial suture placement resulted in the greatest joint stiffness values and consequently, restriction in motion.

5. Outcomes & follow-up

It is difficult to quantify the extent of filling of the Hill Sachs defect after the Remplissage procedure. Moreover, it is not clear to what extent the filling of this defect would be sufficient to prevent the engaging of the humeral head. This is because, the Hill-Sach's lesion is a 3D defect with width, depth, and length, the orientation of which is slightly oblique (Hill-Sachs angle, average 13.8° in the non-engaging lesion and average 25.6° in engaging lesion).[19] In 2014, Rhee et al., devised an interesting scoring system to determine and grade quantitatively the extent of filling into the Hill-Sachs defect by use of magnetic resonance arthrography after an arthroscopic remplissage procedure. The Filling Index Score of Remplissage (FISOR) is calculated by the total scores of the filling index measured in the largest and widest cut in the axial and sagittal planes of the Hill-Sachs defect.[20] However, its usefulness as a measurement tool needs further clinical studies to correlate with the functional outcomes.

Only recently, the longest follow-up of combined arthroscopic Remplissage with Bankart repair has been published by Wolf et al. In their series, only 2 out of 45 patients had re-dislocation following re-injury. Further, there was no significant loss of external rotation or any plane of motion before or after the remplissage procedure that is in contrast to cadaveric studies. These initial clinical results appear to be promising. However, more clinical studies needed to replicate and reinforce the results.

Arthroscopic Hill–Sachs remplissage is gradually gaining popularity as both lesions can be addressed simultaneously. In 2014, Buza et al. conducted a systematic review of patients who underwent a remplissage procedure in association with a Bankart repair for patients who had instability and a humeral head osseous defect.[13] They reported a recurrence rate of 5.4%, which is comparable to published rates for patients without clinically important Hill–Sachs lesions who underwent arthroscopic Bankart repair alone. Buza et al. also concluded that there is a loss in external rotation as compared to normal shoulder. However, it may not be significant. It should be noted that even Bankart repair[21–23] and Latarjet or other bone grafting procedures[24,25] can also result in loss of external rotation. Further, postoperative clinical outcomes were generally good to excellent with no significant complications.

6. Conclusion

Remplissage is an effective procedure for off-track Hill-Sach's defect of any size, and prevents engagement of such a lesion with the anterior glenoid margin resulting in good to excellent outcome after Bankart repair with no significant complications. Loss of rotational movements remains a concern.

Conflict of interest

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Technical note

Arthroscopic shoulder posterior stabilisation – How I do it

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ABSTRACT

An arthroscopic shoulder posterior stabilisation is indicated in symptomatic patients with an isolated posterior labral tear. We present our surgical technique and perioperative management.

1. Background

Whilst the majority of glenohumeral instability is anterior, posterior shoulder instability is present in 2–5% of unstable shoulders.

It can present with instability or posterior pain symptoms. Patients with instability are often able to demonstrate voluntary subluxation/dislocation with forward flexion, pronation and internal rotation; relocation occurs when the arm is brought into abduction and extension (circumduction sign). Posterior pain occurs along the posterior joint line with activities causing a posterior translational load.

Traumatic and atraumatic aetiology is recognised. Traumatic instability follows a distinct history of dislocation or subluxation, often following a significant injury. Atraumatic instability typically follows a history of minor injury or repetitive microtrauma, and is associated with capsular laxity and/or muscle patterning abnormalities.

Treatment includes physiotherapy – particularly in adolescents with atraumatic instability. However, in adults with traumatic instability surgery may be indicated.

AP and axial radiographs, and MRI arthrogram are required to delineate presence of an anatomical cause for instability. In posterior instability posterior labral tears, posterior glenoid fracture or reverse Hill-Sachs lesions may be seen [Fig. 1].

We advocate an arthroscopic shoulder posterior stabilisation in those with an isolated posterior labral tear.

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2. Arthroscopic shoulder posterior stabilisation

Our unit utilises awake anaesthesia where possible. This involves ultrasound-guided interscalene brachial plexus blockade with up to 10–15 ml of Ropivacaine 10 mg/ml, followed by selective blockade of the supraclavicular branches of the superficial cervical plexus using 3–5 ml Ropivacaine 10 mg/ml. Ropivacaine 7.5 mg/ml can also be used but higher volumes may be required. A low interscalene approach provides better cover of the glenoid: the root of C7 is targeted as well as the roots of C5 and C6. Infiltration of the posterior port entry site using up to 5 ml of lidocaine 10 mg/ml is always recommended as the dermatomal supply to this area is variable.

Intraoperatively patients either remain awake or receive conscious sedation using Midazolam or Propofol target controlled infusion. If discomfort is felt by the patient intermittent boluses of 200–300 mcg of Alfentanil can be given. This technique provides excellent analgesia and allows intraoperative patient interaction permitting the surgeon to demonstrate to the patient their anatomical cause for instability and how this has been addressed surgically. However, it is not possible to perform an examination under anaesthesia of the contralateral shoulder if this technique has been utilised.

Patients are positioned in a 70° reclining deckchair position with the arm free. Though a lateral position with traction is useful for opening the glenohumeral joint we find it alters soft tissue tension and therefore prefer to avoid traction.

Surface landmarks are drawn. The arm, axilla and shoulder are prepared with alcoholic Chlorhexidine and a shoulder drape is applied. The arm remains free [Fig. 2]. A surgical assistant is required.

Our technique utilises DePuy Synthes LUPINE loop anchors. A long cannula is frequently required. An extended arthroscope may be required in muscular athletes.

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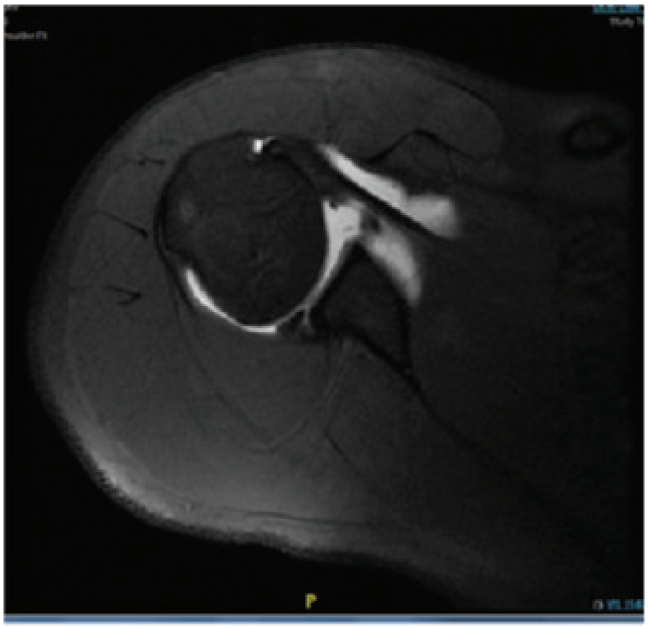

Fig. 1. MRI arthrogram T2 axial demonstrating a posterior labral tear.

Fig. 2. Shoulder arthroscopy surface landmarks demonstrating location of standard posterior and posterolateral portals.

Fig. 3. Arthroscope inserted via posterolateral portal.

Fig. 4. Anterior portal location identified with a needle under direct vision.

The procedure begins with a diagnostic arthroscopy performed via a posterolateral port. This port should be positioned 2 cm inferior to the acromion and far-lateral (beyond acromion edge) [Fig. 3]. It should be able to accommodate a cannula and is mostly used as the working portal. A switching stick can be a very helpful tool to change the arthroscope in the posterolateral portal to a long cannula. A 45° angle of approach toward the posterior inferior glenoid rim is required to allow an adequate angle of approach to the posterior glenoid.

The anatomical cause for posterior instability should be identified including the presence of labral tear. The posterolateral port cannula can be manipulated to provide a better view of the posterior glenoid.

An anterior portal is then made within the rotator interval. In isolated posterior labral repair this should be sited centrally within the interval in line with the glenohumeral joint. If an anterior repair is also required then positioning this portal laterally within the interval is beneficial [Figs. 4 and 5].

The arthroscope is then inserted into the anterior portal and the tear size is assessed and position defined [Fig. 6]. Bony pathology, such as a reverse Hill-Sachs lesion [Fig. 7], should be identified as these may render an arthroscopic stabilisation inadequate.

A liberator is then introduced via the posterolateral portal to elevate the displaced labrum from the posterior glenoid neck [Fig. 8]. Care is taken not to tear the labral tissue.

The 'water off test' is then performed to ensure the labrum has been adequately mobilised. With the arthroscope water inflow turned off the labrum should settle on the posterior glenoid where you would aim your completed repair to reside if it is released

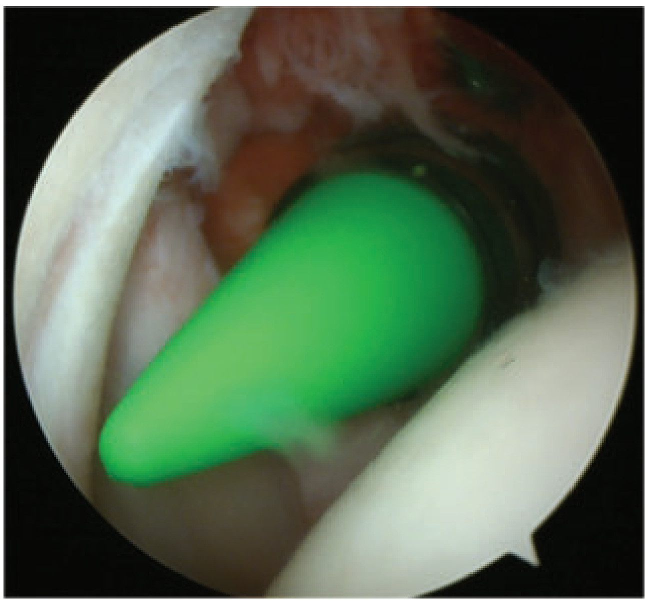

Fig. 5. Anterior portal cannula inserted.

Fig. 6. Posterior labral tear visualised via anterior portal.

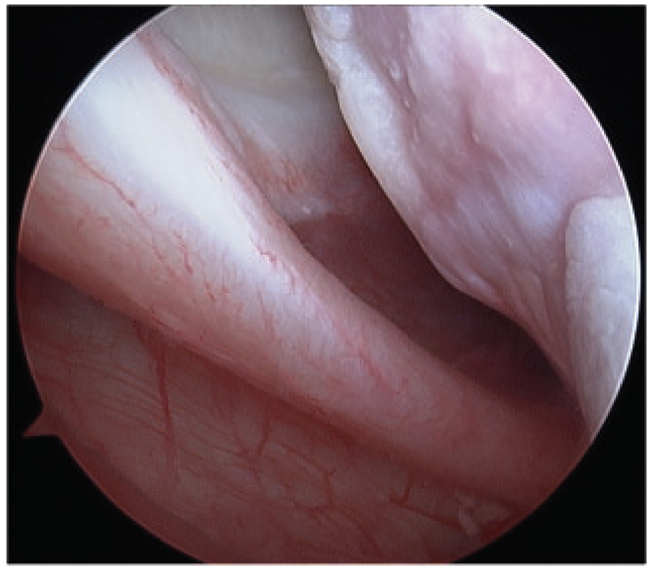

Fig. 7. Reverse Hill-Sachs lesion visualised via anterior portal.

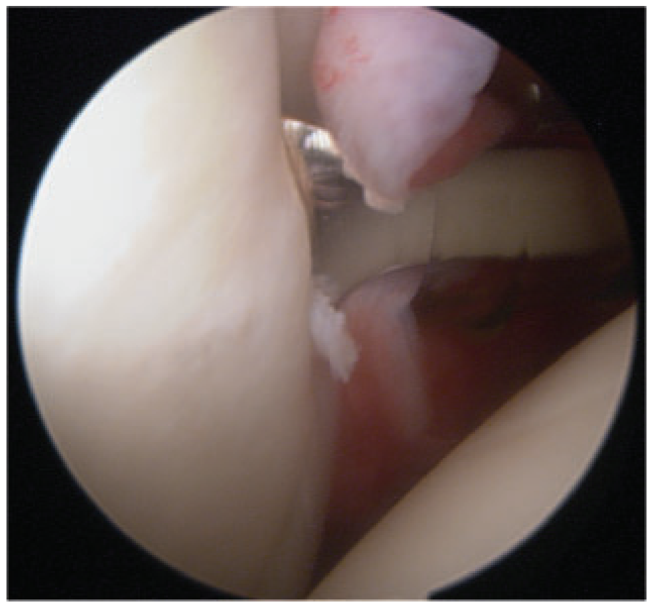

Fig. 8. Liberation of posterior labral tear via posterolateral portal.

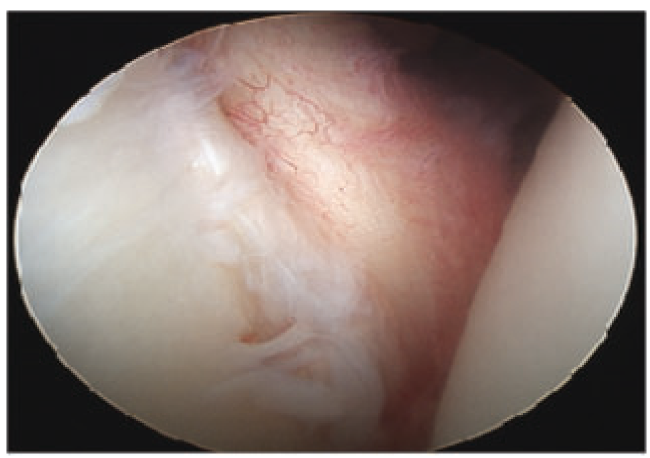

Fig. 9. 'Water off test' following release of posterior labrum.

sufficiently [Fig. 9]. Rasping of the posterior glenoid neck is then performed to obtain a bleeding surface for the labrum to adhere to.

Anchors are inserted into the posterior glenoid just onto the articular surface. Anchor positions are as required following delineation of the front and end of the tear. Frequently only one anchor is required. The anchor drill hole is placed *en-face* on the glenoid to create a bumper effect and augment the concavity within the joint after the repair [Fig. 10].

The anchor suture is passed through the torn labrum and a small amount of capsule and secured with hand tied knots [Fig. 11]. The knots are orientated so that they reside on the far side of the labrum, away from the glenoid articular surface [Fig. 12]. We feel knotted sutures provide better seating of the labrum and more control of the soft tissue tension.

A posterior drive through test is then performed to ensure posterior shoulder laxity has been reduced. The repair should then be visualised via the posterolateral portal to ensure a hood of capsulolabral repair has been achieved [Fig. 13].

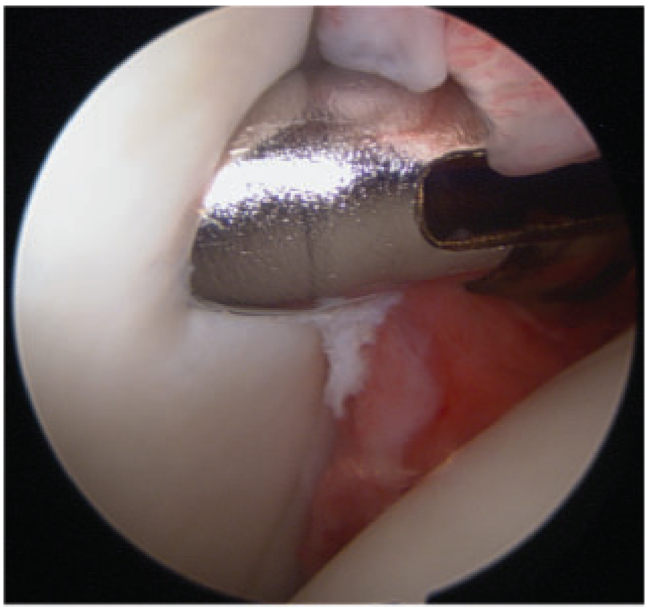

Fig. 10. Glenoid anchor insertion *en-face.*

Fig. 11. Anchor suture passed through torn labrum using suture passer.

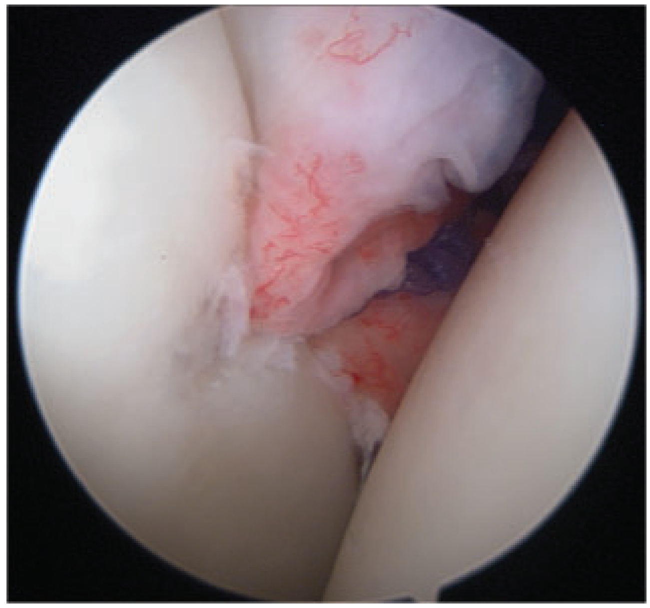

Fig. 12. Anchor suture secured using hand tied knot.

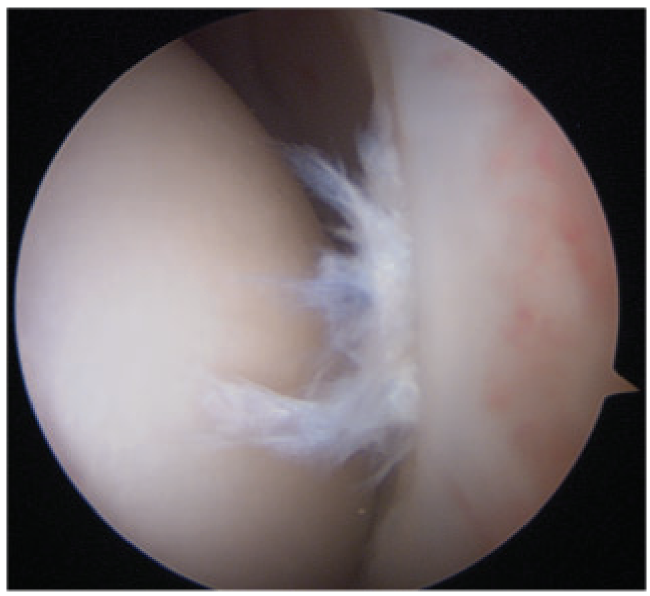

Fig. 13. Posterior labral repair visualised via posterior portal.

Arthroscopic ports are closed with a 3.0 nylon suture and wound dressings applied. A pressure dressing is applied to the shoulder.

The arm is placed into an external rotation sling in neutral. This is applied by our therapists in recovery whilst regional anaesthesia is still active. All procedures are performed as a daycase and patients are discharged with multimodal analgesia including paracetamol, ibuprofen and oral morphine to take when the regional block expires.

Patients remain within an external rotation sling for four weeks. Follow up is scheduled with a specialist shoulder physiotherapist to commence a tailored rehabilitation plan. Regular hand, wrist and elbow exercises can be performed from within the splint once the block has expired to prevent stiffness. At the point of sling removal, active rotation and elevation exercises are commenced for the glenohumeral joint. External rotation is encouraged in elevation.

From six weeks, gentle assisted stretching into internal and external rotation, and elevation, can be added if required. Active, dynamic strengthening of the rotator cuff, again with emphasis on the external rotators, can usually be commenced at this point. Functional and parascapular muscle strengthening may be added as per individual requirements, aiming for a return to normal training by three months. Athletic patients may have a concurrent programme of cardiovascular and contralateral limb exercises, in order to minimise loss of performance.

Consultant review is arranged three months postoperatively with an aim to return to sport, including overhead heavy lifting, at this stage. However, rehabilitation is tailored to the individuals sporting requirements.

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Authors contribution

Daniel L.J. Morris – Manuscript preparation.
David J. Bryson – Manuscript preparation.
Martin A. Scott – Rehabilitation description.
James French – Anaesthetic description.
John Geoghegan – Surgery description and collected arthroscopic images.

Conflict of interest

None.

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Evaluation and management after failed shoulder stabilisation surgery: A review

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ABSTRACT

Shoulder joint being the most mobile joint of the body is very much susceptible to dislocations accounting for half of the dislocations presenting to Emergency departments. Recurrence of instability is quite common owing to the structural defects created as a result of the traumatic event causing the first dislocation. Younger the patient higher is the chance of recurrence. Recurrence after stabilisation surgery is a complication that is indeed a challenge for every shoulder surgeon. Several factors ranging from patient's age and gender to technical errors to missed diagnoses may be responsible for the failure of primary stabilization. A thorough evaluation by detailed history, clinical exam and radiological imaging to explore the causes of failure and then performing the appropriate procedure addressing the causative factor is the key to a successful outcome. Various surgeries have been described in the literature to address the capsulo-labral as well as the bony defects responsible for recurrence. This review article focuses on the etiopathology, evaluation methodology and different surgical treatments available to address the problem of recurrence after primary stabilisation.

Contents

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1. Introduction

The shoulder is the most mobile joint in the body. This wide range of motion also makes the shoulder joint potentially unstable[1]. In the United States, the incidence of shoulder dislocations is 23.9 per 100,000 person-years, with the highest rates in adults in their 20s[2]. Shoulder dislocations account for approximately 50% of all joint dislocations presenting to emergency departments. A traumatic event is usually the precipitating factor for a shoulder dislocation. Recurrent instability following trauma is common due the resultant residual structural defect which is a Bankart lesion most commonly. Recurrent atraumatic instability will not be discussed in this article. Traditionally, open repair has been the gold standard for shoulder stabilization, however, with newer methods and implants, arthroscopic repair is now preferred. Numerous studies over the past decade have shown equivalent outcomes with arthroscopic techniques as compared to open surgery[3]. Despite improvement in outcomes following primary stabilisation surgery, an instability recurrence rate of 3–25% presents the most challenging post-surgical complication[4]. This review article analyses the causes for failure, clinical and radiological evaluation and appropriate management options available when considering revision surgery for failed primary stabilization of the shoulder.

2. Factors contributing to stability

2.1. Soft tissue stabilizers

Glenohumeral joint stability is achieved through a combination of dynamic and static components. The rotator cuff serves as the main dynamic stabilizer, providing compression of the humeral head against the glenoid concavity, centering it during range of motion. Rotator cuff tears can result in uncoupling of these balanced forces across the joint, resulting in instability. Static stability is maintained by the negative pressure within the Glenohumeral joint capsule, the labral complex and the capsuloligamentous structures. The labrum consists of fibrocartilaginous tissue that lines the rim of the glenoid, and serves several functions. First, it increases the surface area of the glenoid and deepens the socket by 50%, thereby providing a "bumper" effect along the bony periphery. Second, and more significantly, it provides a strong anchor point for the capsular ligaments, particularly the anterior band of the inferior glenohumeral ligament[5].

2.2. Bony stability

The shoulder joint is classified as a ball and socket joint, but the glenoid is shallow, it is however, further deepened by the labrum. The glenoid when viewed frontally, is pear shaped with the inferior half wider than the superior half[6]. With recurrent anterior dislocations, two types of osseous defects can result. In the first, attritional loss of the anterior-inferior aspect of the glenoid results from repetitive wear and erosion. Second is a cortical depression in the posterolateral head of the humerus. It results from forceful impaction of the humeral head against the anteroinferior glenoid rim when the shoulder is dislocated anteriorly known as Hill-Sachs lesion.

Burkhart and DeBeer[7,8] concluded that glenoid bone loss of over 22% resulted in a much greater risk of recurrence following arthroscopic treatment of anterior instability and coined the term inverted pear shape for the antero-inferior bone loss. They were the first to draw attention to the concept of engaging and non-engaging Hill-Sachs lesion. They noted that Hill-Sachs lesion with a long axis parallel to the anterior glenoid with the shoulder in the functional position of external rotation and abduction were more likely to result in symptomatic subluxation or dislocation. They defined defects with this obliquity as engaging Hill-Sachs lesions. In contrast, lesions with a long axis nonparallel to the anterior glenoid with the shoulder in the functional position of external rotation and abduction are unlikely to engage the glenoid rim and were termed non-engaging Hill-Sachs lesions. Patients with non-engaging lesions are "candidates for arthroscopic Bankart repair because they do not have a functional articular-arc deficit."

3. Pathology of recurrent instability

Recurrent dislocation following failed repair needs to be evaluated before embarking upon repeat surgery as it is of paramount importance to identify the causative factors which resulted in a repeat dislocation. Ho et al[5] (2016) classified the causes of failure as recurrent trauma, patient factors, misdiagnosis and technique errors. (Table 1)

3.1. Recurrent trauma

Despite good surgical technique, robust fixation and ensuring compliance with rehabilitation to restore functional strength and range of motion of the athlete, return to the provocative sport, inevitably may compromise the primary repair.

Tauber et al[9] studied 41 patients presenting with recurrent anterior instability of shoulder following surgical repair. In their series 59% of cases of recurrent instability were due to recurrent trauma, and majority of these patients had undergone arthroscopic stabilisation as the index procedure. The recurrent instability due to trauma was higher among the patients who were treated arthroscopically as compared to those who had open surgery in their series. Moreover, trauma appears to be the primary mode of failure for open Bankart repair accounting for 100% of recurrences in several studies[10]. Patients who participate in collision, contact, and noncontact sports, including snowboarding, skiing, soccer, cycling, rock climbing, basketball, ice skating, judo, and tennis, are at risk.

Cho et al[11] conducted a study to analyze the clinical outcomes of arthroscopic anterior shoulder stabilisation in athletes and compared the results between collision and noncollision athletes. They concluded that compared with the non-collision group (6.7%), the collision group had a higher failure rate (28.6%).

3.2. Patient factors

Porcellini G et al[12] concluded that age at the time of the first dislocation, male sex, and the time from the first dislocation until surgery were significant risk factors for recurrence. In their series

Table 1
Risk factors for recurrent instability after primary stabilisation.

• Recurrent trauma
• Patient factors
Younger age
Male sex
Increased number of dislocations
Prior procedures
• Missed diagnoses
Anterior glenoid defect
Hill Sachs defect
Capsular laxity
• Technical errors
Medial placement of glenoid anchors
"High" placement of inferior glenoid anchors
Insufficient number of anchors
Improper suture configuration

13.3% were younger than 22 years. Similar findings were reported by Wasserstein et al[13] with 12.6% risk of postoperative dislocation and a 7.7% revision rate after primary stabilisation in patients younger than 20 years compared to rates of 5.5% and 2.8% in older patients aged over 29. Patients with three or more dislocations had double the risk for revision surgery and ten times the risk of re-dislocating.

Tauber et al[9] studied 41 patients presenting with recurrent anterior instability of shoulder, 78% of those patients were male, 59% injured their dominant shoulder and mean age of patients following initially stabilizing surgery was 26 years with mean dislocation of 12 times before the surgery.

3.3. Untreated glenoid bone loss and Hill Sachs defect

Glenoid bone loss can be a major cause of recurrent anterior instability if not addressed during initial surgery. Bigliani et al[14] reported a 12% recurrence rate in patients with glenoid rim fractures that had undergone Bankarts repair. They recommended coracoid transfer if the glenoid rim fracture comprised greater than 25% of the anterior-posterior diameter of the glenoid. Itoi et al[15] performed a cadaver study to examine the effect of glenoid defects on antero-inferior stability with and without repair after simulated Bankart lesions. They concluded that a defect measuring at least 21% of the superior-inferior glenoid length would cause instability that a soft-tissue repair alone might not be able to correct.

Tauber et al[9] studied 41 patients presenting with recurrent anterior instability of the shoulder following surgical repair, and found that 56% of their patients had defect of anterior bony glenoid rim

Burkhart et al[7],[8] reported 67% recurrence rate after arthroscopic soft tissue stabilisation in patients with significant bony defects, and concluded that the inverted-pear glenoid creates an unstable situation. Hence arthroscopic anterior stabilisation is contra-indicated and rather they need the vascularized coracoid graft of the Latarjet procedure. They found the inverted-pear configuration easier to identify than the 25% glenoid loss advised by Bigliani et al as a criterion for coracoid transfer. However, recognition of the inverted-pear glenoid can be rather subjective, but arthroscopically glenoid bare spot can be used as a central reference point to quantify the percentage bone loss of the inferior glenoid.

Sugaya et al[16] applied a best-fit circle to the inferior glenoid on 3D reconstructed CT images. The percentage defect of the glenoid was defined as a ratio of defect width against the diameter of the assumed circle based on the inferior portion of each glenoid. Similarly, Griffith et al[17] CT scanned 218 patients with single or recurrent anterior dislocations, they found Glenoid bone loss is very common, is usually mild (< 10%) in degree, and has a maximum severity of approximately 33%. Bone loss greater than one third of the normal glenoid width was not observed in any patient. This finding is to be expected because more severe glenoid bone loss would involve erosion of the thicker middle third of the glenoid. Compared with arthroscopy, the sensitivity and specificity of CT in detecting glenoid bone loss are 93% and 79% and a high correlation ($r = 0.79$) also exists between CT and arthroscopy regarding severity of bone loss.

The evolving concept of engaging/ non- engaging lesion to on track/off track was introduced by Giacomo et al[18]. They used CT scan images of both shoulders to determine whether the Hill Sachs lesion is on track or off track. They developed a method (both radiographic and arthroscopic) that uses the concept of the glenoid track to determine whether a Hill-Sachs lesion will engage the anterior glenoid rim, irrespective of concomitant anterior glenoid bone loss. If the Hill-Sachs lesion engages, it is called an "off-track" Hill-Sachs lesion; if it does not engage, it is an "on-track" lesion. On the basis of this quantitative method, they developed a treatment paradigm with specific surgical criteria for all patients with anterior instability, both with and without bipolar bone loss. They categorized patients of anterior instability in to four groups irrespective of bipolar bone loss, thus aiding in surgical management of these patients.

3.4. Technical errors

Recurrent instability following stabilisation surgery may be a consequence of technical errors made at the time of index surgery. The technical errors can be classified as 1) Failure to identify and address the pathology. 2) Surgical errors.

Failure to identify the cause can lead to recurrence of symptoms, for example failure to correctly identify and quantify the glenoid and/or humeral head bone loss is associated with higher recurrence rates mainly in patients who participate in contact sports. Similarly, soft tissue pathologies such as posterior labral lesions, generalized capsular laxity and HAGL lesions if not repaired anatomically may lead to recurrent instability.

Surgical errors can relate to position of anchors or number of anchor points. Anchors placed too medial or high anchor position in anatomical repair of soft tissue lesions may lead to recurrence[19.] Boileau et al[20] studied the risk factors for recurrent anterior shoulder instability after Bankart repair. In their series, bony defect, inferior capsular laxity and fewer suture anchors (less than 3) were causes of recurrence. They concluded that minimum four anchor points should be used if there is no significant bony defect or capsular laxity.

Gasbarro et al[21] in their research on reasons for failure after coracoid transfer noted that the most common technical errors leading to revision were placing the graft inferior to the 5 O'clock position on the glenoid face or relying on single-screw fixation. Similarly, Burkhart et al[7] suggested that if the coracoid graft protrudes laterally to joint surface it act as bone block and may lead to late osteoarthritis, conversely, medially placed graft may predispose to recurrent dislocation.

4. Evaluation

4.1. Clinical

The clinical evaluation includes strength, stability, and laxity testing, with comparisons to the contralateral side. Apprehension with the shoulder in 45° of abduction and external rotation, should warn the examiner of bony involvement. The range of motion should be assessed in flexion, abduction, external rotation, internal rotation and external rotation in 45° and 90° of abduction. Excessive external rotation may be due to subscapularis tear and decreased shoulder movement due to excessive tightening of the capsulo-labral complex, impingement from the previous hardware or chondrolysis.

Apart from instability signs, associated lesions such as fractures, rotator cuff tears and neurologic lesions should be suspected as they interfere with the treatment strategy and timing. Rotator cuff involvement after traumatic dislocation of the shoulder has a high incidence in patients over 40 years of age with an incidence in the current literature ranging from 35% to 86%[22–24].

4.2. Imaging

The radiological investigation of patients with recurrent anterior instability is very important to identify the bone loss from the glenoid and humeral head which might have been missed during the initial surgery or might have progressed following recurrent dislocations. More than 80% of surgical failures for shoulder instability are associated with bone loss[7]. Standard radiographs are often the first mode of testing, due to accessibility. This should include a standard AP, true AP, and axillary views.

Bernageau[25] described an effective method for detecting an anterior glenoid rim lesion with the patients in the standing position. Sugaya H et al[26] developed a modified Bernageau method with the patient lying on their axilla in their most relaxed position, which they called the "TV watching position. In this method, clear X-ray images can be obtained more easily with a high probability of ascertaining bony pathology without using fluoroscopic imaging.

Murachovsky and coworkers[27] quantified glenoid bone loss by measuring the distance between the posterior and anterior glenoid rim on Bernageau images and concluded that this quantification provides similar result with that obtained from 3DCT.

Many studies have shown that 3DCT especially the "en face" view is the most accurate method for assessing the glenoid morphology and measuring the glenoid bone loss and Hill Sachs defect. Sugaya H[26] suggested the benefits of 3DCT were three-fold. Firstly, surgeons can recognize glenoid shape and the degree of bone loss intuitively at a glance; Secondly, accurate quantification of bone loss can be possible by using an estimated inferior circle on the en face view of 3DCT; Finally, surgeons can easily assess the size and shape of the bony fragment in shoulders with bony Bankart lesion.

The principle of measuring bone defects on sagittal "en-face" views is based on the fact that the inferior aspect of the glenoid resembles a circle. The circle can be drawn along the posterior, anterior and inferior margins of the glenoid. Baudi et al[28] introduced the Pico method, which is based on CT scanning of both shoulders to provide oblique sagittal images of the healthy and the affected glenoid surfaces. By drawing two identical circumferential areas on the inferior parts of both glenoids, it is possible to measure the missing part of the circle in the affected glenoid and express that area as the size of the defect.

Saliken D et al[29] concluded that radiographs are a useful tool for screening patients of significant for bone loss. However, CT imaging using using the Glenoid Index or Pico Method allows for accurate quantification of glenoid bone loss. The role of CT imaging for Hill Sachs defects is less clear and a criticism of the on track/off track concept is the difficulty to reliably measure the size of the Hill Sachs lesion.

5. Management

Management of recurrence after a failed shoulder stabilisation surgery is indeed a challenge. Identifying the cause for the failure of the index procedure is of paramount importance to avoid another failed procedure. It is well known that chances of poor outcomes increase with every subsequent attempt to stabilize the shoulder[30]. Recurrent rates of instability after a revision surgery range from 0-21.7%[19],[20],[31–39].

The initial management for a patient presenting with recurrence after primary stabilisation is immobilization in a sling for 6 weeks followed by physical therapy for strengthening exercises[34]. Although non-operative treatment is not expected to give desired results in all, some patients may be satisfied with conservative treatment after rehabilitation and activity modification tolerating one or more episodes of dislocation occasionally. However non-operative treatment alone may be best suitable for elderly and low demand patients or where further surgery is contraindicated[59]. It is important to consider factors like collagen disorders responsible for generalized ligamentous laxity or a neurological cause resulting in muscle weakness, which may be responsible for the recurrence and unlikely to be rectified with a revision surgery. Non-operative treatment should be the treatment of choice for such patients.

In patients requiring further surgical intervention a thorough preoperative evaluation to find the underlying cause for recurrence is mandatory before proceeding for the definitive procedure. The chosen revision procedure depends on the underlying cause as well as surgeon's preference and experience with the technique. Labral defect (Bankart lesion), capsular laxity, bony glenoid defect, Hill-Sachs lesion alone or in combination are the commonest issues that the surgeon needs to address in the revision surgery. The choice between an arthroscopic or an open procedure lies with the surgeon depending on his expertise and the lesion to be addressed, Arthroscopy offers an additional advantage as a diagnostic tool helping the surgeon identify any missed pathology with direct visualization of the joint. Even if an open procedure is planned an arthroscopic examination can be done for a 360° assessment of the joint. The technical errors performed in the index procedure may also very well be a cause for recurrence and care should be taken to avoid them in the revision surgery.

5.1. Management of capsulolabral defects

Instability due to a detached capsulolabral complex from the glenoid may be a result of fresh traumatic event or technical failure of the previous stabilisation. It can be addressed by a revision Bankart repair provided there is not >20% bony glenoid loss. Rowe et al[31] demonstrated Bankart lesion (84%) and capsular laxity (83%) to be the commonest causes of recurrence after primary stabilisations. Similar results were reported by Zebinski et al[40] showing Bankart lesion with capsular redundancy to be cause for recurrence in 19 of 23 patients (83%).

Open Bankart repair has been a gold standard for revision stabilisation surgeries in patients presenting with failed Bankart repair[41]. However, the advent of modern implant designs and more refined techniques have closed the gap between the failure rates of open and arthroscopic revisions. Sisto et al[34] treated 30 patients with recurrent instability after failed arthroscopic Bankart procedure with an open Bankart repair. After a mean follow up of 46 months they reported no recurrences and mean modified Rowe scores improved from 25 preoperatively to 84.2 points postoperatively. Cho et al[36] reproduced similar results in 26 patients after failed primary stabilisation with traditional open Bankart repair and 88.5% of the treated patients had good clinical outcome with mean Rowe score of 81 but recurrence in 11.5%.

Several authors have been able to produce successful outcomes with arthroscopic revision Bankart repair as well. Bartl et al[42] treated 56 patients by arthroscopic Bankart repair for a failed primary stabilisation (open or arthroscopic) and after mean follow up of 37 months reported recurrence in 11% of patients while in 86% of patients result were good to excellent with significant improvement in Rowe and Constant scores. In a systematic review including 388 shoulders from different studies Friedman et al[43] reported a recurrence rate of 14.7% after arthroscopic Bankart revision whereas a rate of 5.5% for open Bankart revision. Though the rate of recurrence was found to be lower for open revisions, these differences were not statistically significant.

It is important to assess for any capsular laxity which is not uncommon in patients with multiple episodes of dislocations or multiple failed procedures. Failure to address the capsular laxity will result in failure of any procedure undertaken. An open capsular shift as described by Neer and Foster[44] can be performed. Levine et al[32] performed open antero-inferior capsular shift on 49 patients in addition to revision stabilisation and reported a success rate of 78%. With time arthroscopic capsular plication techniques have also developed to address the issue. Kim et al[19] treated 23 patients after a failed primary surgery with capsular plication and shift in addition to a bankart repair and reported good to excellent results in 83% patients. For inferior laxity, capsule is plicated from inferior to superior to counter the inferior patulous capsule. Closure of rotator cuff interval is also recommended to counter

Table 2
Surgical management of Glenoid bone loss.

Glenoid bone loss	Desired Treatment
<20%	Anterior capsule labral repair (with or without capsular shift) alone
20-33%	Bristow-Latarjet procedure, iliac bone graft
>33%	Fixation of glenoid fragment, structural bone graft

anterior laxity[20]. It is also possible to address the postero-inferior laxity arthroscopically by positioning the arthroscope in antero-superior portals and using posterior portal as the working portal[45].

In our opinion, whenever possible an arthroscopic revision should be preferred as it is a good diagnostic tool as well offering a direct 360° visualization of the joint and may help identify the defects missed in the imaging. With improved implant designs and techniques, the results of numerous studies suggest that arthroscopic revision[19,20,37,39,42]for capsulo-labral defects offer comparable outcomes to open revisions[31,34,36]. Possibility to address anterior as well as posterior defects simultaneously, less soft tissue damage and faster post-operative recovery are some additional advantages of an arthroscopic procedure.

5.2. Management of bone defects

Bone defect in the form of glenoid deficiency or a Hill-Sachs lesion which if neglected in the primary stabilisation are a significant cause for recurrence of instability. These may be present alone or in combination with a recurrent Bankart lesion, and are more commonly encountered in patients who have suffered more violent or more frequent dislocations[46]. Other bone defects that may exist but uncommon are glenoid version abnormalities, humeral torsion abnormalities or a reverse Hill-Sachs lesion which is found in posterior dislocations.

Burkhart and De beer[7] studied 194 shoulders stabilized by arthroscopic Bankart repair and of all the patients who had recurrence 67% had significant bone defect in the form of glenoid deficiency or Hill-Sachs lesion. Similar differences were demonstrated by Chen et al[47].

Glenoid bone loss must be addressed if it is greater than 20% to 25%. The exact figure is still controversial. However, in the presence of Hill-Sachs lesion the threshold to graft glenoid deficiency should be lower[7,47]. A number of different procedures with favorable outcomes have been described in the literature to address the glenoid defect. Coracoid transfer described originally by Bristow and later modified by Latarjet has become the mainstay to address this problem. Burkhart and De beer[8] in another study managed 102 patients with >25% bone loss by open Latarjet procedure and reported recurrence in four (4.9%) patients after a mean follow up of 59 months, hence validating their previous recommendation. Schmidt et al[48] studied results of Latarjet procedure in 49 patients following a failed stabilisation and reported no recurrence after the revision procedure with good to excellent results in 88% patients. In a systematic review Friedmann et al[43] studied 388 shoulders from different studies of revision stabilisations and reported a recurrence rate of 14.3% for patients undergoing revision stabilisation by Bristow-Latarjet procedure for glenoid defects. Though the original technique described was an open procedure performed through the deltopectoral approach, satisfactory results have been obtained using arthroscopic techniques as well. Lafosse et al[49] followed up 100 patients treated with an arthroscopic Latarjet for 26 months and reported excellent outcomes in 91%. However, the data published is for primary stabilsations only and not yet replicated for revision surgeries.

Other procedures available to augment the glenoid surface are by using tricortical iliac bone grafts (Eden-Hybbinette procedure)[50,51] or using a structural allograft. Satisfactory results are well documented for primary stabilisations using tricortical iliac bone grafts. It is particularly useful as a salvage procedure in patients with failed Latarjet stabilisation. Lunn et al[52] studied results of iliac crest bone grafting in 34 patients presenting with failed Latarjet procedure and reported good to excellent outcome in 74% with recurrence in 12% patients.

Amongst different procedures to address the bone defects, using a coracoid transfer is preferred as it eliminates donor site morbidity or other complication related to allografts. However, other augmentation procedures are suitable alternatives in cases of failed Latarjet procedure. The crucial factor when performing a glenoid augmentation is correct placement of the graft as too medial a placement may result in recurrence and too lateral placement may restrict the movement and result in arthrosis in long term[53].

The other bone defect that may contribute to failure of surgery is the Hill-Sachs lesion. These lesions are often undertreated and result in recurrence if significant in size. Hill-Sachs defects when greater than 20% of the articular surface is a significant contributor to recurrence and should be managed surgically[47,54]. Burkhart and De Beer suggested that a Hill-Sachs defects greater than 2 cm in depth, in the setting of failed previous surgery should be addressed in any revision surgery. Treatment options include remplissage, bone grafting or derotation osteotomy.

Bone grafting is an anatomical solution to address larger Hill Sachs defects. Osteochondral allografts can be used to address the defect. The procedure is more likely to succeed in younger population with good bone quality and absence of degenerative changes. Miniachi and Gish[55] treated 18 patients by allograft reconstruction in addition to anterior capsulo-labral repair after a failed stabilisation attempt and a Hill-Sachs lesion of >25%. After a mean follow up of 50 months there was no reported recurrence. Two patients however required screw removal due to partial collapse of the graft resulting in pain.

Remplissage as described by Wolf et al[56] involves capsulotenodesis of the posterior capsule along with infraspinatus into the defect converting the defect into an extraarticular one to prevent it from engaging with the glenoid. Wolf et al studied the results in 59

Table 3
Surgical management of Humeral bone loss (Hill Sachs lesion).

Humeral Bone loss	Desired Treatment
20%	Anterior capsule labral repair (with or without capsular shift) alone
20-30%	Anterior stabilisation + Remplissage or disimpaction and cancellous bone grafting
30-40%	Anterior stabilisation + Cancellous bone grafting or Ostechondral allograft or humeral derotation osteotomy
>45%	Humeral replacement by hemiarthroplasty or total shoulder replacement.

patients treated with arthroscopic Remplissage in addition to an anterior Bankart repair. After a mean follow up of 58 months, they reported recurrence in two patients only which were due to an episode of trauma. Remplissage is beneficial when is used as an adjunct to anterior repair and not as an isolated procedure[57]. Cho et al[58] demonstrated the importance of Remplissage by comparing the clinical results of isolated arthroscopic Bankart repair and those of arthroscopic Bankart repair with posterior capsulodesis. The recurrence rate was 25.7% in the isolated Bankart group and 5.4% in the Bankart repair with Remplissage group. McCabe[59] evaluated the effect of remplissage in primary as well as revision setting for significant Hill-Sachs lesion. They evaluated 31 patients, of whom 11 had remplissage along with anterior stabilisation in their revision surgery and 7 of these went on to have successful outcomes. It is worth mentioning that none of the 20 patients who had remplissage for the bone loss along with primary anterior repair reported any recurrences. This emphasizes the importance of timely identifying the bone loss and addressing it with the primary surgery. The procedure of remplissage however may be associated with a mild loss of external rotation compared to normal side.

For larger defects of the humeral head, Weber described a derotation osteotomy[60]. The principle of this osteotomy is to prevent the engagement of the defect with the glenoid by rotating it away from the articulating arc. The amount of rotation required is determined by degree of external rotation at which the defect engages with the glenoid. The procedure is suitable for younger patients where joint replacement may not be a desirable option. Weber et al[60] studied the results of this osteotomy in 207 patients and reported a recurrence rate of only 7% with good or excellent results in 90% of patients and a mean loss of external rotation of less than 5°.

Chen et al[47] have recommended a treatment algorithm to manage the bony defects whether glenoid or humeral depending on the size of the defect which is determined using axial CT scans or axial films. (Tables 2 & Tables 3)

riedmann et al[43] in their systematic review of 19 studies related to the problem of recurrent instability after primary stabilisation pointed out an interesting finding that studies which failed to report on bone loss had the highest rate of recurrence even after secondary stabilisations. Hence, the importance of bone loss in the stabilisation surgeries cannot be underestimated and should be addressed in the primary stabilisation itself to avoid recurrence.

6. Conclusion

A variety of surgical procedures are available to address the issue of recurrence after primary stabilisation. A careful analysis by detailed history and examination and with the help of various imaging techniques must be done to identify the correct cause for the recurrence before proceeding with further intervention. Non-modifiable factors such as collagen disorder resulting in ligamentous laxity must be taken into account to decide if revision will be of any benefit. The choice of procedure and technique whether open or arthroscopic for the identified cause should also depend on surgeon's ease and expertise with the chosen procedure. Finally, it should be kept in mind and the patient should be well informed as well that the chances of poor outcome are more with every successive revision surgery. Nonetheless if the correct procedure is selected for the correct patient outcome is likely to be successful in the majority of the patients.

Conflict of interest

None

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Review article

The management of sternoclavicular instability☆

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ABSTRACT

Sternoclavicular joint (SCJ) instability is a rare condition that has myriad of presentations ranging from acute dislocation to chronic instability and may have severe consequences for the patient if it is missed. This article outlines the clinical presentations, investigations and the principles behind the available treatment options that can ensure a safe return to a normally functioning shoulder. Instability of the SCJ may happen after a traumatic event or atraumatically with or without joint laxity. The clinician should promptly differentiate the two pathomechanisms as management of them differs significantly. The Stanmore instability triangle is a useful tool when assessing patients with chronic SCJ instability as it enables the clinician to recognise the factors that drive the instability and treat each component separately and in a staged manner.

Treatment is dependent on understanding the various factors including the direction of instability, chronicity and pathomechanisms. This could involve conservative management with resting the arm in a sling followed by targeted physiotherapy or surgical management with closed or open reduction and if required, surgical stabilisation with autograft, suture anchors or plating.

Contents

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1. Introduction

The SCJ is a unique joint that plays a pivotal role in force transmission and smooth scapulothoracic motion during shoulder elevation and is formed by the clavicle which is perched over the top of the sternum and medial end of the first rib. It is stabilised mainly by the strong extrinsic ligaments and a dynamic muscular envelope. It is one of the overlooked joints due to paucity of informed literature on the subject, difficulty in diagnosis, limitations of standard radiography, unfamiliar anatomy and complex biomechanics.[1] The aim of this article is to outline the salient points that an orthopaedic clinician must be aware of when faced with injuries or instability around the SCJ, so that the diagnosis is not missed which thereafter may lead to morbidity.

2. Anatomy

The SCJ is a synovial plane joint formed by the sternal end of the clavicle, the upper lateral part of the manubrium, and in 25% of people, the cartilage of the first rib. Less than half of the joint surface of the medial clavicle articulates with the sternum. The ligaments of this joint include the anterior and posterior sternoclavicular, costoclavicular (between the first rib and the medial clavicle, also known as rhomboid) and interclavicular ligaments (Fig. 1). The intra-articular disc is a flat and nearly circular fibrocartilage, interposed between the articulating surfaces of the clavicle and sternum and attached posteromedially to anterolaterally. It divides the joint into two cavities, each of which is lined by a synovial membrane. This articulation is the only synovial joint between the upper limb and the chest wall. During scapulothoracic motion, the majority of movement is transmitted through the clavicle into the SCJ. This allows motion in nearly every direction, up and down (lateral compartment), forwards and backwards (medial compartment) as well as circumduction. The clavicle elevates 4° for every 10° of arm elevation through the first 90° of forward elevation.[2] The medial clavicle and the intra-articular cartilage glide on the articular surface of the sternum during this motion. Combined movements require rotation, and the clavicle may rotate by as much as 40° along its longitudinal axis.[3]

The SCJ is an inherently unstable joint; however, its main stabilisers include strong extrinsic ligaments and to a lesser extent a dynamic muscular envelope. The important ligaments for stability are thought to be the anterior and posterior sternoclavicular ligaments.[4,5] The anterior is about 50% weaker than the posterior.[6] Rupture of the intra-articular disc could also cause instability5 in young adults. The aponeurotic insertion of the superficial part of the clavicular insertion of the sternocleidomastoid (SCM) is contiguous with the more direct insertion of the clavicular and sternal parts of pectoralis major (PM) below. The subclavius muscle has a tendinous origin from the first rib immediately lateral to the costoclavicular ligament and has a long direct insertion onto the inferior surface of the clavicle. It acts to reduce the rate and range of upward displacement of the clavicle when under lateral compressive loads on the shoulder. Behind the SCJ's lies the thoracic inlet, containing the great vessels of the superior mediastinum, followed by the trachea, oesophagus, vagus and phrenic nerves behind the sternohyoid and sternothyroid muscles (Fig. 2). It should be noted that the medial epiphysis of the clavicle only starts ossifying at 18–20 years of age and doesn't close until 23–25 years of age. Medial clavicle physeal fractures in young adults can therefore be difficult to differentiate from SCJ dislocations.

3. Epidemiology

Traumatic dislocations of the SCJ comprise 1% of all joint dislocations and 3% of those in the upper limb.[7] It is far more common in active, young males who are involved in sporting injuries, road traffic accidents and fall from a height which results in a high-energy mechanism of injury.[8] A direct blow to the medial aspect of the clavicle can cause a posterior dislocation of the SCJ but more commonly these are indirect injuries due to a compressive force applied to the lateral aspect of shoulder girdle.

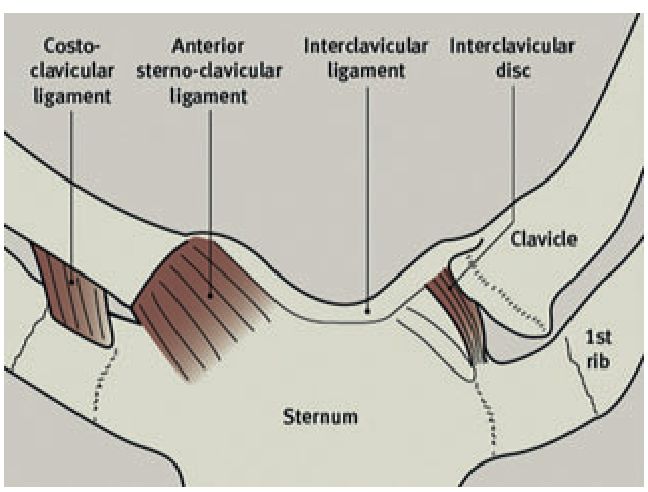

Fig. 1. The anatomy of the sternoclavicular joint.

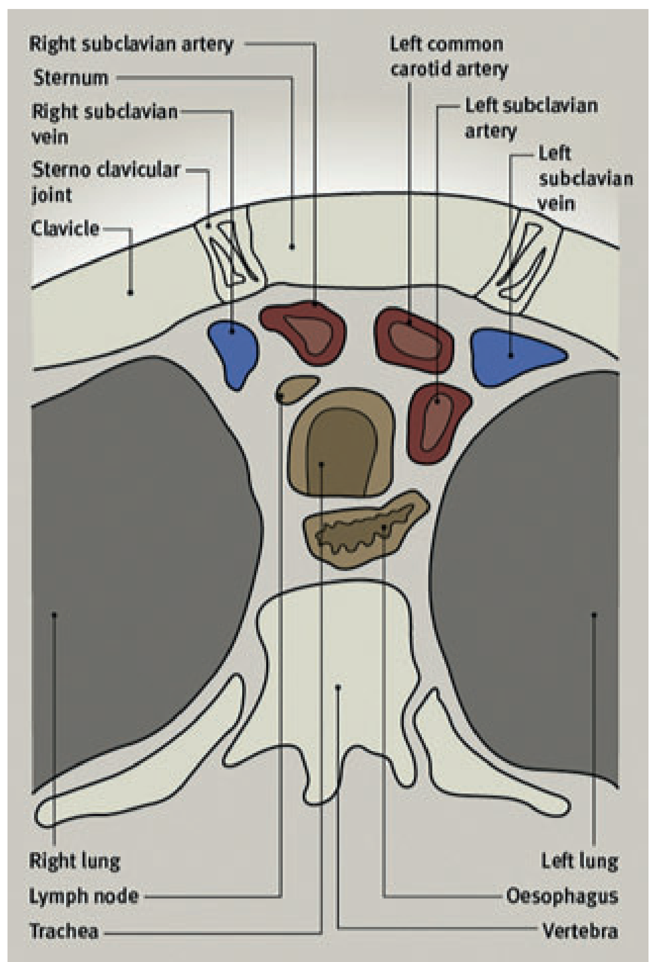

Fig. 2. Cross section of the thorax at the level of the sternoclavicular joint showing the structures immediately posterior to the joint.

4. Classification

SCJ dislocations can be classified according to direction (anterior or posterior), aetiology (traumatic or atraumatic), severity (sprain, subluxation or dislocation) and history (acute or chronic). In patients with no joint laxity, the SCJ dislocation is usually associated with a defined injury (e.g. rugby tackle, road traffic accident or falls from height).[9] It is postulated that a compression to the anterolateral or posterolateral aspect of the shoulder, pushes the humeral head against the glenoid. The energy transmits through the shoulder girdle to the SCJ which then dislocates in the anterior or posterior direction respectively.[10] Anterior dislocations are more common owing to the strength of the posterior sternoclavicular ligament. On the other hand, though posterior dislocations are rarer, they can lead to more serious sequelae due to compression of mediastinal structures.

Atraumatic dislocations of the SCJ could be better understood when used in conjunction with the Stanmore triangle of instability.[3] The three polar groups include type I (traumatic structural) in which there is a clearly identifiable traumatic incident with structural damage to explain the reason for instability, type II (atraumatic structural) which may not be associated with trauma but could be associated with capsular laxity or abnormal shape of clavicle or inflammatory pathology and type III (neuromuscular non-structural) which is associated with muscle patterning without any trauma or structural abnormality and can often be bilateral. It should be noted that there is a continuum between the groups, for example a patient with a type I instability may develop a muscle patterning element (type II) and is now called type I/II.

5. Presentation

5.1. Acute

Patients with anterior dislocation usually present with an immediate prominent swelling or pain over the SCJ and typically the evening or next day, the joint starts to click in and out (becomes dislocatable).[9] Patients with posterior dislocation are unfortunate in that they are more difficult to diagnose and in most danger of developing long-term problems. A palpable, painful defect adjacent to the sternum may be found in a posterior dislocation thus resulting in the ipsilateral shoulder being pulled forward in comparison to the other side. In 25% of cases, there is mediastinal compromise.[8] This may result in venous congestion in the ipsilateral arm, hoarseness, cough or dysphagia. Any of these symptoms should raise the suspicion of compression of mediastinal structures by a posterior dislocation and are indications for urgent reduction.[11]

If the patient is under 25 years of age, a Salter-Harris II medial clavicle fracture should be considered until proven otherwise. In a polytrauma patient, this injury can easily be missed in the secondary survey, so another examination must be completed after the distracting injuries have been treated.

5.2. Chronic

Patients present with either an old injury or history of minor trauma sustained recently. The latter group of patients are often teenagers who have joint laxity and are similar to patients with atraumatic glenohumeral joint dislocation. On assessment, the following should be looked for[9]:

1 magnitude and mechanism of injury
2 presence of generalised joint laxity (consider Beighton score[12])
3 type of dislocation (anterior or posterior, dislocated or dislocatable)
4 evidence of mediastinal compromise (for posterior dislocations)
5 assess ipsilateral shoulder girdle for slumped posture, scapular rhythm and overactivity of PM or SCM muscles

6. Investigations

The Serendipity (Rockwood 40° cephalad tilt) and Heinig (X-ray beam perpendicular to the SCJ but oblique to supine patient) views have been described but plain radiographs have poor sensitivity. They will diagnose a medial clavicle fracture but the mainstay of diagnosing a SCJ dislocation is by way of computer tomography (CT). A CT scan will identify associated mediastinal injuries as well as Salter-Harris II fractures of the medial clavicle. This requires a very careful search for the small fragment of bone attached to the epiphysis[9] (Fig. 3). MRI scan is more useful in cases of chronic SCJ instability to assess intra-articular disc tears, ligament disruption or injury to mediastinal structures.

7. Treatment

7.1. Acute anterior dislocation

If an anterior SCJ dislocation reduces spontaneously, it is recommended that a sling is used for six weeks and then the arm mobilised as it may settle and stabilise itself. The patient should expect a return to normal function and range of movement though ectopic bone formation may occur that may need removal if it becomes symptomatic.[9] If it remains dislocated and is diagnosed within 48 h, a closed reduction under general anaesthesia may be attempted, however, it may remain unstable.[13] Others advocate this within 7–10 days.[14] This is achieved by laying the patient supine, placing a solid pad between the shoulders and placing direct pressure over the medial clavicle.

If closed reduction fails, the surgeon should proceed with surgical reduction. The patient is placed in the beach chair position. A transverse incision should be centred over the joint, identifying the dislocation. The anterior costoclavicular ligament is found avulsed from either the sternal or clavicular side. The senior author prefers to use two anchors inserted into the bone from whichever end the ligaments have been avulsed. These anchors should be placed 45° to the sagittal plane along the joint line (Fig. 4). The

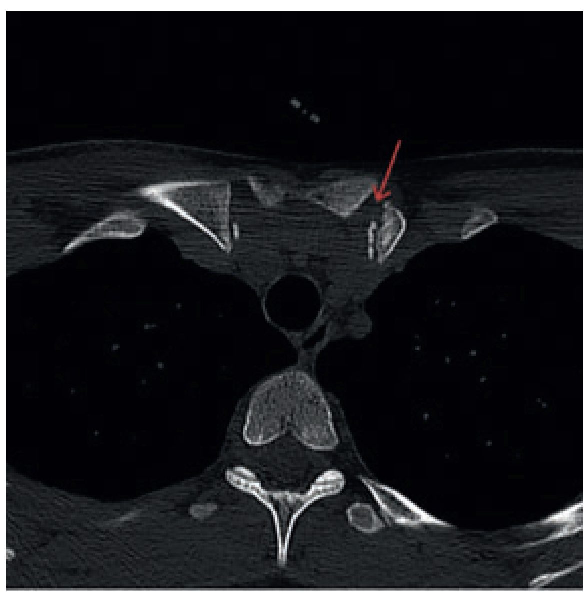

Fig. 3. Unfused epiphysis in a young adult.

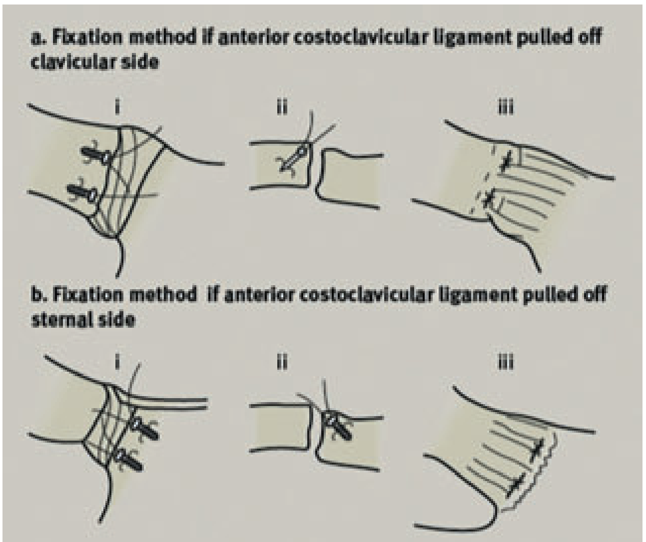

Fig. 4. Reattachment of the anterior sternoclavicular ligament. (a) Showing reattachment at the clavicular end (i) anchor insertion (ii) cross section showing anchor insertion, (iii) showing capsule sutured back in place. (b) Showing reattachment at the sternal end (i) anchor insertion (ii) cross section showing anchor insertion, (iii) showing capsule sutured back in place.

ligament is then reconstructed. Care should be taken when drilling for the anchors and the surgeon should always check that the base of the drill hole is within bone. A layered closure with subcuticular prolene and steristrips provide the neatest scar. The patient is immobilised in a sling for six weeks to maintain scapular protraction and should not be allowed to return to sport until after six months.[9] Patients should be counselled of the risk of recurrence (very small), that the joint will always be prominent[7] and that their function should recover.[14]

There are two schools of thought regarding persistent instability. One is that the disability from this is negligible[10] whereas others report that patients with neglected dislocations have a higher dissatisfaction compared to those treated surgically.[15] Given the modern techniques of reconstruction, an argument can be made for fixing these acutely. This has been substantiated by a recent systematic review[16] and we also believe early fixation is indicated if the joint is unstable after reduction or dislocates recurrently.

7.2. Acute posterior dislocation

It is always advisable to carefully review the CT scan before any procedure to locate the precise position of the clavicle with reference to the mediastinal structures. The usual quoted time for a successful closed reduction to occur is 48 h though there have been reports of up to five days.[10] A sandbag is placed behind the shoulder and while traction is applied to the abducted shoulder, the arm is moved into an extended position. Alternatively, traction to the medial clavicle using a sterile reduction forceps is applied.[10] Should this fail, adducting the arm and applying traction whilst direct pressure is applied to the front of the glenohumeral joint can be used.

The stability of the reduction is then tested under anaesthesia. If it is not stable, it can be assumed that instability will ensue, and the joint can be surgically stabilised. Although some authors may feel unstable anterior dislocations may be left, the same is not true of posterior dislocations because of the risk of mediastinal compression.[11] Given the proximity of the mediastinal vessels in posterior dislocations, it is prudent to have a cardiothoracic surgeon available. The same approach is used as for anterior dislocations with care taken when approaching the SCM tendon. Applying a reduction forceps with gentle traction directly over the medial clavicle should easily reduce the dislocation in the acute setting. On the other hand, if the dislocation has been present for more than a week, the medial clavicle will have developed adhesions. The can be addressed by gently lifting the periosteum and pulling the clavicle out to length.[9] The senior author prefers to stabilise the joint after reduction with a No 2 Vicryl in a figure of eight configuration using 3.2 mm drill holes through the clavicle (from inferior to superior) followed by two holes through the outer cortex of the sternum. Postoperatively, the patient is placed in a sling for six weeks, to allow soft tissue healing. A delayed return to sport is advised (four to six months) and a return to full function and range of motion should be expected.

7.3. Salter-Harris II fractures

As mentioned earlier, Salter-Harris II fractures should always be suspected in young patients and excluded with CT scans. In the experience of the senior author, they are all posterior injuries in ages between 9–24 years old.

Sewell et al[3] reviewed the literature and noted that though it is considered that these injuries could remodel to allow correction of any significant displacement itself, there is paucity of robust data and the current recommendations are to attempt closed reduction for posteriorly displaced fractures and only open when there is mediastinal compromise. He identified that if these patients develop malunion, they often complain of pain or symptoms of thoracic outlet syndrome and fixing them acutely is much easier than dealing with late complications. This matches the senior author's experience. So, our recommendation is, if the patient is seen acutely (within days), a closed reduction may be attempted, though most will need an open reduction. The same approach is used for posterior dislocation with all the necessary precautions taken such as identifying the SCM tendon and gently mobilising the clavicle by freeing the periosteum (if the reduction is delayed) as well as the medial epiphyseal fragment which is narrow and fragile. Once the reduction is achieved, this can be stabilised with a figure of eight No 2 Vicryl suture across the front of the joint (Fig. 5). The patient is then given a sling for six weeks and advised to return to sports after three months. They should expect to return back to normal function and range of motion.

7.4. Chronic sternoclavicular instability

The treatment of chronic SCJ instability depends on the presence or absence of generalised joint laxity and history of trauma. Type I instability patients (traumatic structural, no joint laxity) with a history of trauma are treated with surgery and this will be discussed below. Type 2 instability (atraumatic structural often joint lax) encompasses several pathologies and an accurate diagnosis made is essential for a successful outcome.[3] These include conditions causing capsular laxity e.g. Ehlers-Danlos and Marfans syndromes. The average age at presentation is 18 years but with a wide age range from age 8 upwards.

Patients typically have a story of the joint starting to click/ be painful and a lump appearing over the sternoclavicular joint (constantly or intermittently) when they move the joint, with no clear association with trauma. They are lax jointed. The mainstay of treatment for this group of patients is physiotherapy. Key features to look for are a slumped posture, "a chin poke position of the head on the neck", scapular dysrhythmia and poor central muscle control and is often associated with hyperactive PM and SCM muscles[9] (not unlike atraumatic instability of the glenohumeral joint but the abnormal muscles groups are different). The aim of physiotherapy is to improve core stability and abolish abnormal

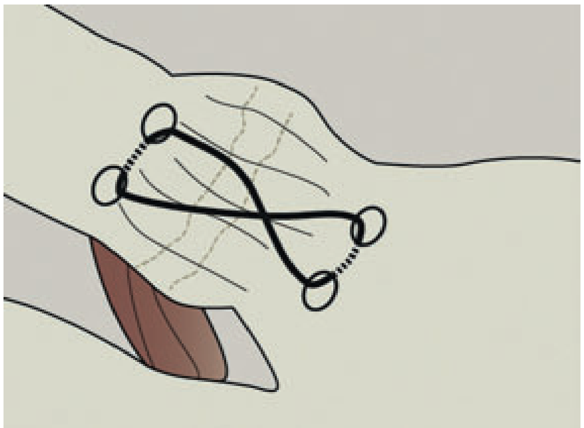

Fig. 5. Figure of eight suture across sternoclavicular joint used to increase stability post reduction.

muscle patterning. Whilst it is imperative to have a compliant patient, it should also be stressed that the physiotherapist must have the correct skill set in dealing with these complex patients. Compliance with a physiotherapy regime for five to six months will usually lead to a stable joint. In difficult cases, the senior author has used Botulinum toxin to temporarily weaken the aberrant muscle groups to assist the physiotherapy.

The most difficult patients to treat are the lax traumatic and non-lax atraumatic injuries.[9] A course of physiotherapy should be offered, to deal with any abnormal muscle patterning followed by reassessing the patient. There are reports in the literature of the use of Botulinum toxin to weaken the aberrant muscle groups prior to surgery with the intention of improving the outcome.[3]

7.5. Surgical reconstruction for the chronically unstable joint

Early methods of stabilising the sternoclavicular joint were based on the erroneous belief that the primary stabilisers were the costoclavicular ligaments. Stabilisation involved wrapping tissue around the medial clavicle and first rib with the inherent risk of intrathoracic complications from subcostal instrumentation.[10]

K wires have also been used but were associated with migration of the wires in the great vessels,[17,18] the pulmonary trunk[19] or mediastinum[20] and should not be used as discussed in two systematic reviews.[16,21]

Current stabilisation methods are based on a better understanding of the main stabilisers (sternoclavicular ligaments).[4,5] The principle is to stabilize with a construct in a figure of eight configuration across the front.[19] The following have been used: tendon autografts (SCM,[22] palmaris longus,[23] semitendinosus,[23–25] gracillis[25]) some with synthetic braids to augment the reconstruction (Orthocord (DePuy Mitek, Raynham, Massachusetts) and Fibrewire[11] (Arthrex, Naples, Florida), anchor sutures[26] or plating across the joint and unloading the joint with an osteotomy followed by plate removal and clavicle plating.[27] (Balser; Peter Brehm, Chirurgie-Mechanik, Wiesendorf, Germany).

The senior author's preference of reconstructing chronic SCJ dislocations is using the least invasive option which is preferentially to use the SCM tendon. Should this be inadequate, palmaris longus is used followed by semitendinosus tendon as a backup (if no palmaris longus). The same skin approach is used as for anterior dislocations. A vertical incision is made in the joint capsule. If the disc is torn, it is debrided to stable tissue; if it is intact, it can be incorporated into the repair. The sternal head of SCM tendon is identified, harvested (leaving the sternal end attached) and tubularised. The tendon graft should be at least 8 cm long; if not then palmaris longus should be used instead, or semitendinosus.

If the SCM tendon is used, a 3.2 mm drill hole is made to the clavicle 3–5 mm lateral to the joint surface in a cephalad direction. The tubularised harvested tendon is then passed through the capsule followed by the hole in the clavicle, then back through the capsule ending back to its sternal origin (Fig. 6). As the joint is most unstable at 120 degrees of abduction, it is reduced and sutured in this position. The tendon is tensioned and sutured in-situ with 2.0 Vicryl. The capsule is double breasted (Fig. 7). The patient is put in a sling for six weeks followed by six weeks of activity limited in front of the body and finally three months of unrestricted movement but no contact sport. Physiotherapy is only indicated if abnormal movement patterns develop.

If palmaris longus or semitendinosus are used, the only difference is that 2 holes are drilled through the anterior sternal cortex and the graft passed through the bone, then passed through the clavicle with the tendon held in a figure of 8 position across the joint. The capsule is double breasted as previously. The tendon once tensioned is sutured to itself with a weave if possible.

8. Further discussion

SCJ dislocations leading to chronic instability is rare and as a result, there is paucity in the literature regarding the optimal method of surgical stabilisation.[16,21,28] Martetschläger et al[28] performed a more recent literature search highlighting the presence of many case reports and small case series dealing with SCJ instability. Of the 321 relevant articles, 23 were English-language case series of 5 or more patients dealing with instability. Furthermore, all of the case series were either level 3 (few) or level 4 (many). Though it is noted that a proper randomised controlled trial is needed, it is impossible to conduct such a study due to the scarcity of these SCJ injuries in clinical practice.

Spencer et al[24] performed a biomechanical study comparing the mechanical strength of three different techniques, including subclavius tendon reconstruction, intramedullary ligament reconstruction and semitendinosus figure of eight reconstruction of the SCJ. They provided evidence of the superiority of the figure of eight

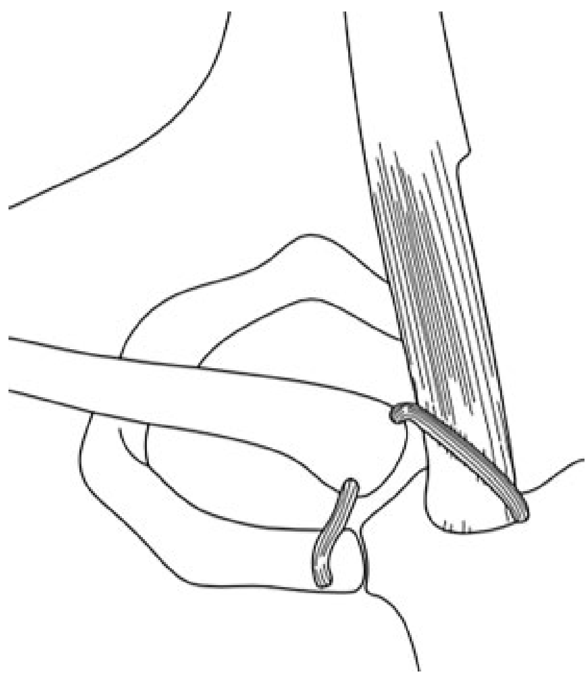

Fig. 6. A diagram demonstrating the harvest of the sternocleidomastoid tendon, kept attached at its sternal end, tubularised and passed through a drill-hole in the medial end of clavicle.

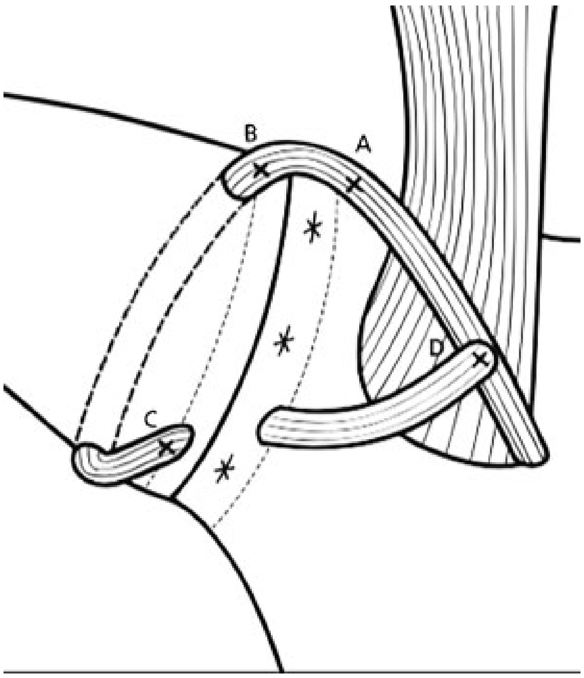

Fig. 7. A diagram demonstrating the method by which the tendon is braided through the anterior capsule to reconstruct the anterior sternoclavicular ligament. Points A–D are the places where the tendon is sutured to the underlying tissue.

reconstruction technique compared to the other techniques. As noted already, there are various ways performing the reconstruction in the published literature, including the use of tendon autografts (SCM,[22] palmaris longus,[23] semitendinosus[23–25] and gracillis[25]), anchor sutures[26] and plates[27] (Balser; Peter Brehm, Chirurgie-Mechanik, Wiesendorf, Germany).

The senior author described the use of SCM tendon graft in six patients with seven chronic SCJ instabilities[22] (6 anterior, 1 anterosuperior) with a mean follow up of 40 months (15–63). The stability of the joint improved in each case: two were normal, four experienced transient subluxations and one had occasional subluxation causing a reduction in activity. Two patients reported transient sensitivity in the scar and one had transient ulnar nerve paraesthesia which resolved.

Singer et al[25] described the use of hamstring tendon autografts (semitendinosus in 4 patients and gracilis in 2 patients) in a figure of eight configurations in six patients with chronic SCJ instability who were initially treated conservatively (3 anterior, 2 posterior, 1 multidirectional) with a mean follow up of 22 months (14–34). The DASH score improved from 54.3 points preoperatively to 28.8 points postoperatively with all patients returning to full activity without limitations including competitive contact sports.

Abiddin et al[26] describes the use of suture anchors on the manubrium and capsular plication in eight patients with acute and chronic SCJ instability (7 anterior, 1 posterior) with a mean follow up of 4.4 years (1–7.6). The mean Constant score was 74.88 with all patients but one returning to their previous employment. There were two failures, one of which occurred after a traumatic event.

Franck et al[27] treated 9 patients with chronic SCJ instability (7 anterior, 2 posterior) with Balser plate stabilisation with a mean follow up of one year. Outcome was assessed with a mean Constant score of 90.2 (84–100) and DASH score of 8.4 (4.1–16.6). Implants were removed from all patients after three months. One patient developed a complication (seroma), requiring surgical drainage. No cases of re-dislocation were noted.

The only published case series reporting patients treated non-operatively was by Sukul et al[29] who treated 10 patients with anterior chronic SCJ instability with a mean follow up of 63 months. They reported a good result in seven patients, fair in two patients and poor in one patient. A systematic review performed by Glass[21] concluded “if a patient does require an open reduction, tenodesis, suture fixation and ORIF are all effective and are the recommended open reduction techniques”. However, it states that the only available articles in the literature were retrospective case series and that a prospective randomized controlled trial will be necessary to more accurately define the most appropriate and successful treatment options.

9. Conclusion

SCJ instability is a rare condition that may have severe consequences for the patient should it be missed. It usually occurs after a traumatic event but may also present in patients with joint laxity. The orthopaedic surgeon should promptly differentiate the two pathomechanisms as management of either differs significantly. The Stanmore instability triangle is a useful tool when assessing patients with chronic SCJ instability as it enables the clinician to recognise what “drives” the instability and treat each component separately and in a staged manner.

Treatment is dependent on various factors including direction, chronicity and pathomechanism. This includes conservative management by resting in a sling (followed by targeted physiotherapy) or surgical management which includes closed or open reduction with or without surgical stabilisation by way of tendon autografts, plating or suture anchors.

Patients with acute or chronic anterior instability may be treated conservatively initially if they tolerate their condition. In cases of acute instability requiring open reduction or inability to maintain a reduction in a posterior dislocation, repair of the joint capsule is sufficient surgical treatment.

Indications for surgical reconstruction in chronic instability include patients with anterior dislocation who have ongoing disability or patients with posterior dislocation who fail to reduce or have ongoing instability. The use of tendon autografts, suture anchors and plates are all acceptable, effective and safe surgical techniques which allow patients return to full activity without limitations. If a tendon autograft is used, the most stable configuration is by way of figure of eight through holes in the sternum and manubrium. K-wire fixation is an absolute contraindication due to possible migration which can lead to catastrophic consequences.

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Conflict of interest

None.

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Review article

Acromioclavicular arthritis: A review

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A B S T R A C T

Acromioclavicular arthritis is one of the most common causes of pain around the shoulder joint in middle-aged and elderly population. It is because of early degeneration of the articular cartilage and disc. The disease usually remains asymptomatic, and it presents as an incidental finding on shoulder X-Ray or Magnetic resonance imaging. It can be due to primary degenerative, inflammatory, traumatic and infective arthritis. The patient may also come with the atypical location of pain like in cervical or over deltoid muscle. A thorough understanding of its anatomy, biomechanics, history and physical examination of the patient is necessary for management of this condition. Treatment options include lifestyle modification, a short course of chemotherapy, muscle strengthening, physiotherapy or local intraarticular injection of steroid. After six months of conservative treatment if the patient still has pain then operative treatment is advisable in the form of open or arthroscopic distal clavicular resection.

Contents

1. Introduction

Acromioclavicular (AC) joint arthritis is the most common cause of pain around shoulder joint in the middle-aged person because of the degeneration of the cartilage and intraarticular disc. Most of these patients are asymptomatic, and may present as an incidental finding in shoulder X-Ray or Magnetic resonance imaging (MRI). Patients present with complaints of pain over the joint while doing overhead and cross body activities. In the study by Jorden et al.[1] symptomatic AC joints were present in 23% of patients undergoing

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Table 1
Table showing the commonly affected joints by OA according to their prevalance.

Most common joint affected by OA in decreasing order	Age standardized prevalence[7] radiographic and syptomatic	
knee	25.4%	15.4%
hip	19.6%	4.2%
hand		
Foot		
shoulder		

shoulder MRI. Osteolysis following micro-trauma is commonly seen with overhead activities like weight lifting[2,3], swimming and some contact sports[4–7]. Patients sometimes complaints of non-specific neck, shoulder, and arm pain which further complicate the diagnosis, that is why a complete understanding of the anatomy of the joint, history and thought out clinical examination is more important in making a diagnosis of this condition (Table 1).

Primary AC joint arthritis is a relatively rare entity in comparison to osteoarthritis (OA) of the knee and hip,[6] but it is more common than primary OA of the shoulder.[8] However, the exact prevalence of AC joint arthritis is still not clear. In one study[9] based on MRI findings symptomatic group shows 100% evidence of OA (28% with severe grade) and in asymptomatic group 82% shows OA changes (4% with severe grade). This symptomatic primary osteoarthritis of AC joint is a relatively uncommon clinical entity.[10] We are presenting a review of AC joint arthritis describing in detail about anatomy, biomechanics, pathophysiology, diagnosis and treatment options.

2. Review

2.1. Anatomy

The AC joint is a diarthrodial type of synovial joint formed by convex distal clavicular end and the concave looking medial acromial facet. The capsule surrounds the joint completely, and it has a synovial lining. The capsule is reinforced superiorly and inferiorly by ligaments around the AC joint. The fibrocartilaginous disk is acting as a cushion for load transferring from shoulder girdle to clavicle just like meniscus in the knee.[10] It is composed of 75% water, 20 % collagen (most of which is type 1 and some amount of type 2, 3 and 4), 5% proteoglycan, elastin and other cells.[9] The AC joint is surrounded by extracapsular or intracapsular ligaments providing additional strength. The quadrilateral shaped AC ligament is an intracapsular ligament, and along with the capsule, it surrounds the joint superiorly, anteriorly, and posteriorly. The thickness of this ligament is about 2.5 mm, and is a major stabilizer of AC joint. It resists 50% anterior and 90% posterior displacement. At its superior end, it is augmented by fibers of deltoid and trapezial fascia. Coracoacromial ligament provides vertical stability to the AC joint[4] and is attached to the tip of coracoid process and the medial articulating surface of the acromian process. It is a primary resist to superior and axial translation, and in the absence of AC ligament, it resists anterior and posterior translation[21]. The coracoclavicular ligament is composed of two-parts namely trapezoid and conoid. A trapezoid is triangular and joints upper surface of coracoid to the inferior clavicle. The conoid attaches posteromedially on conoid tubercle of clavicle and coracoid process in front of the scapular notch forming a vertical triangle band. Conoid and trapezoid are accessory supports to the AC joint [12–15] (Fig. 1).

2.2. Biomechanics of acromioclavicular joint

The thicker superior and thinner inferior AC ligaments in conjunction with the capsule are predominantly responsible for AC joint stability in the anteroposterior as well as in the superior-inferior plane. The conoid provides the greatest contribution to superior translation (62%), and the trapezoid resists most (75%) of the axial compressive loads (as in weightlifting). In all degrees of displacement, the AC capsule and ligaments are the stabilizers of the clavicular translation. The clavicle and scapula simultaneously rotate during this “synchronous scapula clavicular” motion resulting in very little relative rotation (5 to 8°) at the AC joint.[16]

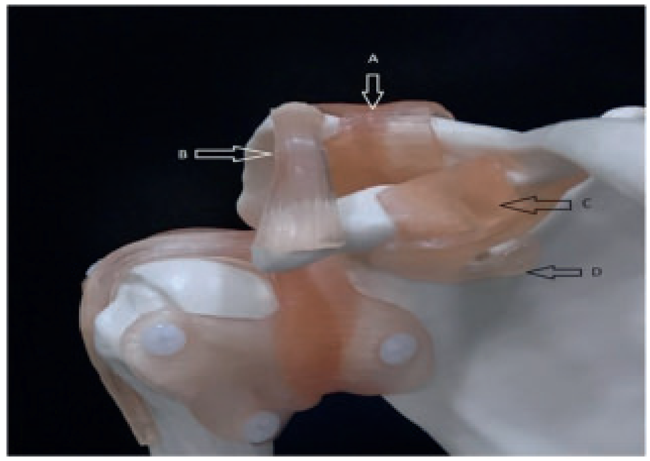

Fig. 1. Acromioclavicular joint anatomy.[A- Acromioclavicular ligament, B-Coracoacromial ligament, C-Trapezoid ligament, D- Conoid ligament].

Scapular motion occurs with the simultaneous motion at AC joint, and it transfers very high load across small articular area via incompletely developed disc.[17,18] A healthy AC joint undergoes 6 mm translation in anterior, posterior and superior direction under the load[19] and 5–8^0 rotation with scapulothoracic motion and 40–45^0 shoulder abduction. The AC joint has dynamic and static stabilizers. Ligament complex act as a static stabilizer. The dynamic stabilizer like deltoid and trapezoid muscle crosses the joint and provides additional stability by distributing forces across AC joint with shoulder movement.

2.3. Pathophysiology

Primary OA of the AC joint appears to be related to the normal age-related damage and subsequent loss of protective cartilage causing painful bone-on-bone contact.[20] DePalma et al. found the degenerative change in specimens by as early as the 2nd decade of life[21] which is further supported by Horvath and Kery.[22] However, true prevalence is still not clear because most of the time arthritic changes goes on silently without producing any symptoms which make diagnosis more difficult.[23] The AC joint has the relatively small surface area, and it is subjected to high loads, and shear stresses leading to early degenerative change[24] especially with repeated overhead abduction. AC joint is also subject to inflammatory and infective changes like septic arthritis which is relatively rare but in this location[25] *Staphylococcus* or *Streptococcus* bacteria is the most common cause[26,27] and a high index of suspicion should be kept in patients with intravenous drug abuse. Inflammatory arthritis like rheumatoid arthritis is also common in this joint. A recent prospective study of 74 patients demonstrated that although the AC joint was affected more often than the glenohumeral articulation by rheumatoid arthritis, both joints were affected in almost half (42%) of the cohort (Table 2).

2.4. Clinical presentation

Although most of the patient remains asymptomatic the pain is the first symptom to develop. Patients come with pain around AC

Table 2
Table showing the unique features of AC joint.

Unique feature of AC joint
Anatomical
1. The relatively small surface area of this joint and it is subjected to high loads and shear stresses with repeated activities and overuse leads to osteolysis.
2. Early degeneration of fibrocartilagenous disc articular cartilage as early as 2nddecade as studied by De palma[67].
3. Superficial location of joint which leads to more prone to get injured.
Clinical
1. Degeneration is a silent process most of the patient remain asymptomatic there for diagnosis often delayed.
2. Common in middle aged individual and athlete involved in overhead and cross body adduction type of activity.
3. Most common pathology of AC joint and one of the common cause of shoulder pain often missed or not diagnosed properly.
Radiological
1. Plane AP x ray of shoulder show overlapping and fails to demo clear joint and often diagnosis missed that why Zenka view is recommended.
2. *T–99 m bone scanning*: Can detect the lesion which are not visible on normal x ray and can detect early lesion and it is highly sensitive and specific.
3. MRI is highly sensitive but have low specificity because most of the time it is incidental finding in shoulder MRI does not correlate with patient present condition.

joint with routine activities involving overhead and cross body movement[28] like while bathing, and combing. Sometimes pain may refer to anterolateral deltoid and trapezius.[29,30] Pain at night is also common during sleeping on the affected shoulder. Swelling is rare, but occasionally patient may develop ugly looking swelling on shoulder especially in case of inflammatory and infective arthritis. Asymmetry of the joint can also be present. Movement restriction is rare in this condition, but active and passive overhead and cross-body adduction are painful. Patients may complain of popping, clicking, grinding, or catching sensation with the movement of their shoulder.[31] Most effective test for diagnosis of AC joint pathology is 'cross-body adduction test' in which examiner stands in front of patient, patient's arm is elevated to 90^0 forward and while holding patient's elbow shoulder is adducted across the body leading to production of pain around AC joint which is suggestive of pathology of AC joint. However, it is not specific for AC joint pathology; false positive may occur in sub-acromial pathology, (most sensitive 77%)[32] (Fig. 2). Hyperabduction pain localised to AC Joint is also diagnostic of AC Joint pathology. In 'O'Brien active compression test', the examiner stands behind the patient and arm of patient is flexed forwardly at 90^0 with elbow in full extension then adducted the arm 10^0 to 15^0 crossbody now internally rotate the arm so that thumb pointed downward now examiner apply uniform downward force against resistance by patient, test is repeated with palm fully supinated test is consider positive if pain elicited by the first maneuver and disappear by second, localization of pain to AC region confirms the diagnosis.(most specific 95%).[32] AC resisted extension test: with the shoulder in 90^0 forward flexion patient asked to actively extend shoulder if this produces pain at AC joint suggestive of AC joint pathology.

2.5. Radiographic evaluation

Plain anteroposterior (AP) X-Ray (Fig. 3) of shoulder or chest often does not demonstrate AC joint anatomy. Therefore, Zanka (Figs. 4 and 5) demonstrated modified AP view by projecting X-Ray in $10–15^0$ cephalic direction and decreasing the kV by 50%. The X-Ray usually shows findings of osteoarthritis-like joint space narrowing, osteophyte formation, subchondral cyst/osteopenia, periarticular sclerosis and distal clavicular osteolysis. Joint space narrowing is not as much significant as OA of knee. For better visualization of bony damage CT scan can also be done. T-99 m bone scanning can detect the lesion which is not seen in normal X-Ray. It is helpful in diagnosing pathology in symptomatic early stage especially in a young individual. It is highly sensitive and specific. MRI[33] (Fig. 6) is highly sensitive in the diagnosis of AC joint pathology and provide information about soft tissue condition[34] but often MRI finding does not correlate well with the patient's condition that's why specificity of MRI is low.

2.6. Joint injection

The injection into the joint may serve both diagnostic and therapeutic purposes [35]. A combination of local anesthetic and corticosteroid is commonly preferred. After palpating joint, the superior approach is recommended. Accurate needle placement into the joint may prove difficult because of variations in joint anatomy, osteophyte formation, and other degenerative changes. Radiographic evaluation in advance of injection can help delineate the local anatomy to aid in successful joint entry. The accuracy of injection can be increased by using USG guidance [36]. If the patient has relief after few minutes, the diagnosis of AC joint pathology is confirmed. Most important criteria for the sensitivity of this technique is patient must be symptomatic before the injection.

Fig. 2. Picture showing the 'Cross adduction test'.

Fig. 3. X-Ray Anteroposterior (AP) view of the shoulder with no clear demonstration of AC joint.

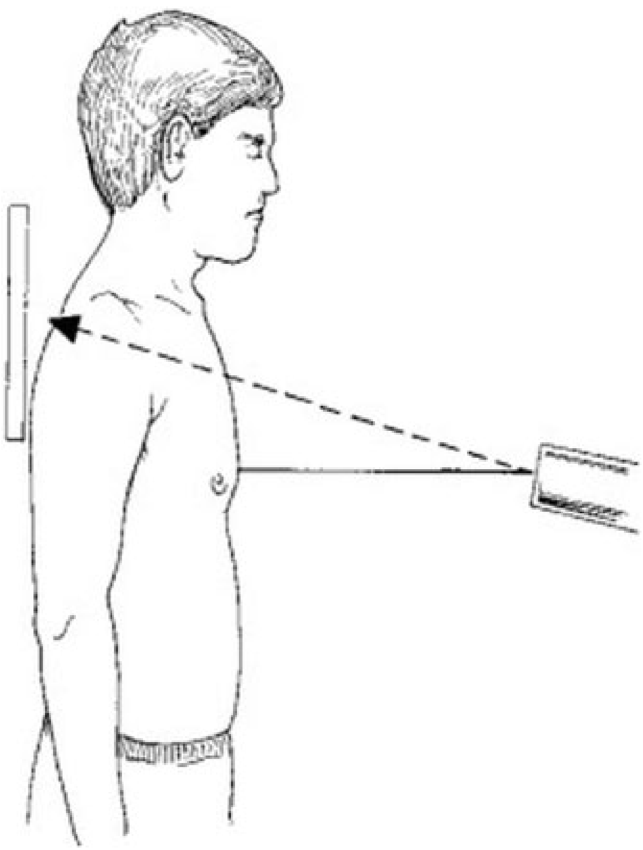

Fig. 4. Zanca view for AC joint is taken with X-Ray beam tilted 10–15° cephalad.

Fig. 5. Zanca view showing clear AC joint anatomy.

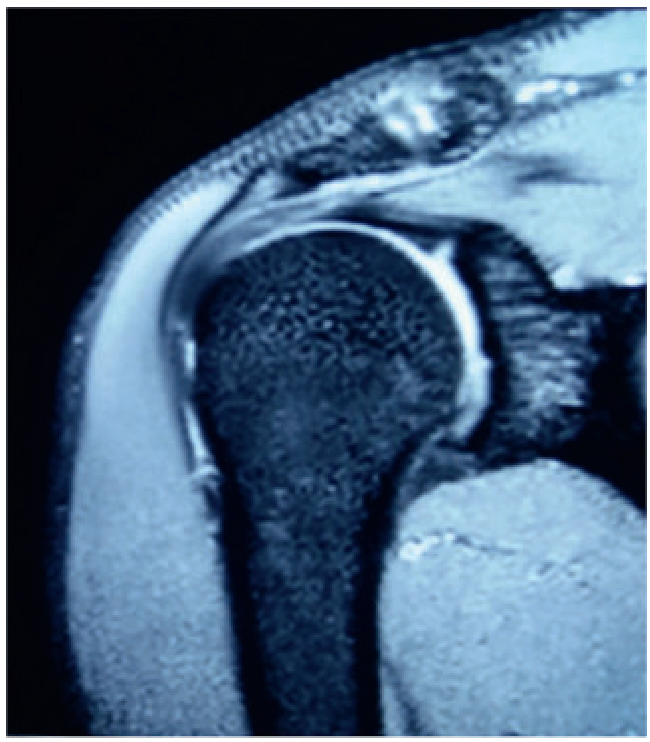

Fig. 6. MRI of the shoulder joint showing AC joint arthritis.

2.7. Differential diagnosis

Can be divided into Intrinsic shoulder problems, such as rotator cuff, sub-acromial sarcoma, lymphoma or multiple myeloma and Extrinsic conditions like referred pain from cervical spine pathology (both mechanical or degenerative diseases of the cervical spine).

2.8. Treatment options

Treatment of AC joint arthritis starts with conservative methods like lifestyle modification, avoiding activities which precipitates pain like overhead and cross body activities. Temporary avoiding these factors are sometimes sufficient to relieve symptoms. This can be combined with a short course of a pharmacological agent like NSAIDs along with moist therapy for temporary relief of pain. At least 6 months of nonoperative treatment is advisable. Strengthening of deltoid and trapezius is also helpful in relieving symptoms by decreasing load across AC joint. However, if the patient still complains of pain after about six months of conservative treatment, then surgical treatment is advisable.

Intraarticular injection of the steroid [39] mixed with the local anesthetic agent is helpful in treating; steroid provides relief for short-term only that is why it should be combined with physio and activity modification to have long-term relief.

2.9. Surgical options

After around six to nine months of conservative treatment if the patient does not have any relief then operative management is advisable which can be either open or arthroscopic.

Open distal clavicle resection: Open distal clavicle resection was performed first time by Mumford and Gurd in 1941. [40,41] Straight incision is given over superior aspect of AC joint in the line of deltoid fiber after splitting deltotrapezial fiber. Superior joint surface is reached by exposing meniscus after this arthritic surface is exposed by elevating clavicle subperiosteally. Distal clavicle is resected but resection should not exceed more than 10 mm. Care is to be taken to avoid any soft tissue damage, and shoulder movement is performed to check any impingement. The advantages of this approach are the relative simplicity of the procedure, less surgical time, adequate bone removal, and the favorable outcome [42]. Disadvantages include the potential for injury and weakness in the reattached deltoid and trapezius and the delay in returning to active function while awaiting muscle healing.

Arthroscopic resection: Arthroscopic resection has been performed since 1986. Advantages include avoidance of detachment, reattachment, and consequent potential weakness of the deltoid and trapezius; shorter duration of postoperative protection; and quicker recovery [43,44]. Two surgical approaches have evolved subacromial (indirect) approach[45], superior (direct) approach. The indirect sub-acromial approach was first reported by Esch et al. in the supine position, he did arthroscopic distal clavicular resection in a patient undergoing subacromial decompression for impingement. It can be performed in lateral and beach chair position[46–48]. In this technique, bursectomy is necessary for proper visualization of AC joint. It is becoming more popular among surgeon because with this approach surgeon not only can do distal clavicle excision but can also perform acromioplasty, subacromial decompression or rotator cuff repair [49,50]. This also reduces the risk of post-op instability of the clavicle by preserving the superior AC ligaments as it prevents superior AC ligament.

In a superior (direct) approach, joint is reached from the direct superior portal, useful for pathology lying in AC joint only. Superior AC ligament injury more common that is why post-op recovery may be delayed. It is advisable for the young individual who want to return to activity early. The result of open or arthroscopic surgery is similar, and the patient has a good amount of pain relief after surgery. However, arthroscopic resection is more popular in today's world because of early postoperative recovery and less soft tissue damage.

Postoperative rehabilitation: If resection is small with no soft tissue damage, 10–15 days of immobilization is given in simple sling. Patient is then advised to encourage pendulum exercise, with active and passive physiotherapy. However, if there is more soft tissue damage or split of deltoid fiber like in open surgery up to 4 weeks of immobilization followed by assisted physiotherapy is advised (Table 3).

Table 3
Clinical feature and management of AC joint arthritis.

Diagnosis and management of AC joint arthritis	
Clinical	
symptoms and sign:	1. mostly asymptomatic but pain is most common symptom associated with overhead and cross body activities. 2. tenderness at joint, popping, clicking, grinding or catching sensation.
physical test:	1. *Cross body* adduction *test* (most sensitive) 2. O'Brien active compression test(most specific) 3. AC resisted extension test.
Imaging:	
x ray	1. AP view of chest with both AC joint visible. 2. Zanka view- shows clear anatomy of AC joint. x ray beam is tilted 10 to 15° cephalad, and decreasing kv by 50%.
MRI	1. Provide batter information about soft tissue involvement 2. It is highly sensitive but less specific
T–99 m bone scanning	1. Can detect early lesion and it is most sensitive and specificity
Diagnostic Joint Injection:	1.Serve both diagnostic and therapeutic purposes 2. Combination of local anesthetic and **corticosteroid** commonly preferred. 3. Patient must be symptomatic is most important criteria.
Treatment	
1. Non operative	• Life style modification • NSAID • Hot fomentation • Intra-articular injection • Physiotherapy
2.Operative	Surgical Options 1. **Open Resection**: Simple and less time consuming but have high postop morbidity. 2. **Arthroscopic Resection** A) **Subacromial (indirect)** approach: Require bursactomy Suitable in dealing subacromial and AC pathology from single approach. B) **Superior (direct) approach**: Direct approach to AC joint no direct access to sub acromial region.

2.10. Complications after surgery

Persistence of pain after surgery is common due to over or under resection of the clavicle. Postoperative instability [51,52] may be present due to injury to the stabilizers of the AC joint i.e. superior AC ligament causing anterior-posterior instability and damage to coracoclavicular ligament causing superior-posterior instability. It is more common with the direct superior approach. Posterior translation after more than 10mm resection is commom[55]. A patient may complaint of persistent pain, if resection is done inadequately. Weakness due to damage to soft tissue like ligaments and deltoid fiber or inadequate repair of deltotrapezial junction leads to deltoid dehiscence during open surgery. Some times patient may develop heterotopic ossification [53,54].

3. Conclusion

Acromioclavicular arthritis is a common cause of pain in the shoulder of a middle-aged person engaged in overhead and cross body abduction type of activities. Most of the time it remains asymptomatic and comes as an incidental finding in shoulder imaging. Diagnosis completely depends on good clinical history and examination supported by appropriate imaging. Initial treatment is conservative followed by arthroscopic or open surgery. Arthroscopic resection has early postoperative recovery and less morbidity.

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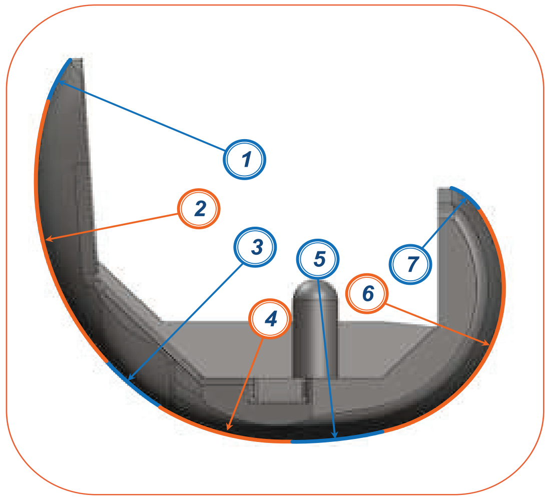

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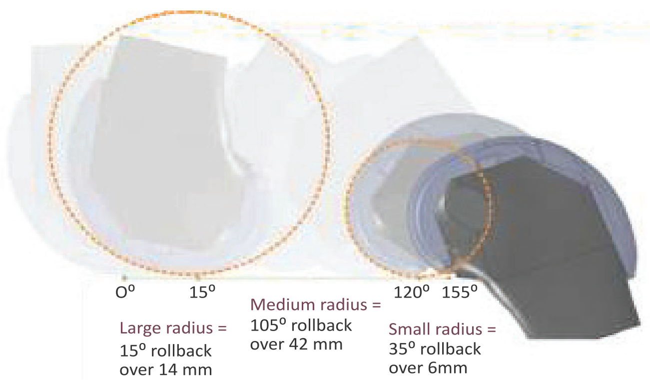

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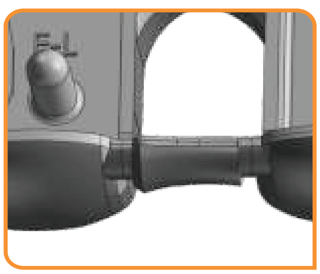

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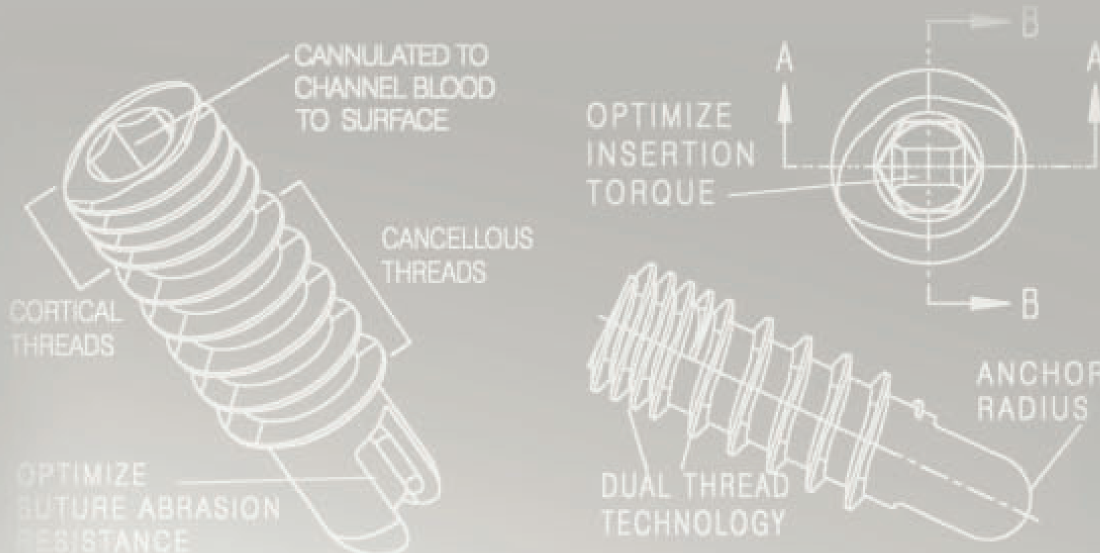

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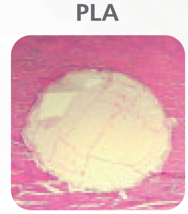

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