Shoulder instability is defined as the abnormal and symptomatic movement of the humeral head across the face of the glenoid fossa. This is caused by failure of the stabilising mechanisms of the shoulder. The extent of this abnormal translation will determine the severity of the presenting symptoms. Minimal translation causes apprehension or pain. Moderate translation causes subluxation; patients report this as a feeling of instability or the shoulder slipping in and out. Finally, extreme translation will lead to full dislocation.

**Traumatic shoulder dislocation**

Acute shoulder dislocation is common with a reported incidence of 11 to 44 per 100,000 person years. To put this in context, the incidence of wrist fractures is 62 per 100,000 person years. There is a bimodal age distribution with peaks in the 3rd and 9th decade. Athletes and males are at particular risk.

For many patients an acute traumatic shoulder dislocation is an isolated event. For others, damage to the stabilising mechanisms of the shoulder impairs their function resulting in recurrent instability. It is reported that about 26% of patients will go on to further dislocation within four years. This increases in younger patients and if the patient is under 20 years of age their risk of further dislocation goes up to between 64 and 87%.

Injuries associated with acute traumatic shoulder dislocation include neurological injury (48% incidence on EMG but only 13% clinically evident), vascular injuries, rotator cuff tears in patients older than 40 and greater tuberosity fractures.

**Management of traumatic shoulder dislocation**

There are three key aspects of the management of the traumatically dislocated shoulder:

1. Safe and expeditious reduction of the acute dislocation (this will not be covered in this article)
2. Initial treatment of the recently reduced shoulder with the management of pain, restoration of function and prevention of further episodes of instability
3. Management of recurrent episodes of instability, if they occur. This requires an understanding of the cause of recurrent instability and the strategies to address them.

**Initial treatment of the recently reduced shoulder**

The patient should be placed in a simple broad arm sling to manage pain. This should be removed and the shoulder mobilised as pain allows. Prolonged use of a sling with the shoulder immobilised in either internal or external rotation does not reduce the risk of recurrence. The role of acute surgery is controversial. Arthroscopic washout does not reduce the risk of further dislocation. A recent Cochrane review supports acute stabilisation in the young contact.
Management of recurrent traumatic instability

Surgical strategies to manage recurrent instability are based on an understanding of the biomechanics and anatomy of the normal shoulder and how these fail.

Biomechanics

There is an inverse relationship between range of movement and intrinsic stability in every joint. The intrinsic or static components of stability are determined by bony anatomy and the ligaments and capsular condensations. The shoulder has a wide range of movement to allow precise positioning of the hand in space. This is achieved at the expense of intrinsic stability. There is minimal bony constraint of the shoulder. The glenoid concavity is deepened by the glenoid labrum and the non-uniform distribution of the articular cartilage. This increases the static constraint of the shoulder. The glenohumeral ligaments constrain the movement of the humeral head but only become tight at the extremes of range of shoulder range. The ligaments also have a relatively poor mechanical load to failure with a mean ultimate load of 585N, this compares to 2160N for the anterior cruciate ligament.

Therefore, the shoulder relies heavily on its dynamic stabilisers: a complex interaction of muscle forces simultaneously effect rotational movement of the humeral head whilst minimising translational movements (Table 1). These automatic muscle patterns require sensory inputs to locate the humeral head relative to the glenoid, so they “know” how hard to pull. The capsule and labrum have abundant proprioceptive mechanoreceptors to feedback the humeral head position and allow the dynamic stabilisers to function appropriately (Figure 1).

The capsuloligamentous-labral complex, therefore, functions both as a static stabiliser as well as the efferent limb of the proprioceptive feedback loop (Figure 2).

Mechanisms of stabiliser failure

The stabilisation mechanism fails in predictable ways:

1. A catastrophic load in which the speed of application and magnitude overcome both static and dynamic stabilisers and leads to traumatic dislocation
2. Recurrent instability occurs after a traumatic dislocation if it causes a structural abnormality of both the static and dynamic stabilisers
3. The stabilisers can be congenitally deficient
4. The efferent/afferent dynamic stabilisation loop can become disordered in the absence of structural abnormality.

This categorisation explains the origin of the most widely used and accepted clinical classification of recurrent shoulder instability: the Bayley or Stanmore triangle (Figure 3).

This system describes three polar types:

- Polar type I: Traumatic structural (analogous to TUBs in the Thomas and Matsen classification)
- Polar type II: Atraumatic structural analogous to AMBRI in the Thomas and Matsen classification
- Polar type III: Muscle patterning non-structural.
In general, ligaments fail at their origin or insertion, not in their mid-substance. Structural failure of the capsuloligamentous-labral complex occurs most commonly at the interface between the labrum and the glenoid, but occasionally failure occurs at the humeral insertion of the capsular ligaments.

Avulsion of the capsuloligamentous complex from the glenoid:
Bankart tear: The typical anterior labral pathology seen in traumatic anterior shoulder dislocation. The labrum is pulled from the glenoid and the glenoid neck periosteum ruptures, thus detaching the labrum from the glenoid (Figure 4).

ALPSA lesion: Anterior Labroligamentous Periosteal Sleeve Avulsion. This is also an anterior labral pathology, but differs from a Bankart lesion as the glenoid periosteum remains intact. In this situation the labrum falls medially onto the glenoid neck (Figure 5).

Perthes lesion: This lesion is very similar to an ALPSA, the glenoid periosteum is again intact; the labrum is avulsed from the glenoid but not displaced. This is hard to visualise on MR imaging, but can still lead to instability (Figure 6).

Avulsion of the capsuloligamentous complex from the humeral head:
HAGL: Humeral Avulsion of Glenohumeral Ligament. This lesion is seen when the capsule and its ligamentous structures are torn from the humerus during the dislocation.

Bone defects:
Bony Bankart: This lesion is an anterior glenoid fracture. The anterior labrum is still attached to the detached bone fragment.

Hill Sachs lesion: The glenoid impacts into the posterior humeral head when it dislocates stripping the cartilage and creating a defect in the postero-superior humeral head. This lesion may 'engage' on the anterior glenoid rim during movement leading to instability.

Pathological changes in the capsuloligamentous complex:
It is our assertion that prior to avulsion of the capsuloligamentous complex from either the humeral head or the glenoid, the elastic limit of the capsuloligamentous complex is exceeded and plastic deformation occurs. The amount of plastic change will depend on the compliance of the tissues: the older the patient, the less compliant the tissues are and the less permanent is the change in length of the capsuloligamentous complex. We postulate that this results in less disruption and hence the reduced chance of re-dislocation in older patients.

Patients with instability who have a type I or II component may benefit from surgery to correct the structural pathology. Patients with pure polar type III instability will not benefit from surgery and may indeed be harmed as they will have an increased risk of arthritis and rehabilitation failure.

In this article we will concentrate on polar type I (recurrent traumatic) instability where trauma has caused a structural abnormality damaging both the static and dynamic stabilisers.
Investigation of recurrent traumatic instability

MRI and CT arthrography have both been used to evaluate the post-dislocation anatomical damage. They both have their merits, but MRI has significant advantages when evaluating the soft tissue structures, especially labral tears (sensitivity 44–100%, specificity 66–95%). MR with arthrography has a sensitivity of 86% and 91% and a specificity of 86–98% for labral pathology. In view of this, MRI arthrography is the investigation of choice. It is generally accepted that CT is the superior modality for assessing bone pathology.

Surgical management of recurrent traumatic instability

Open procedures: The aim of most open soft tissue repairs is to re-tension the anterior capsule, particularly the anterior band of the inferior glenohumeral ligament. This can be done by double breasting the anterior capsule with a T shaped capsular incision (Neer and Foster or Altchek). Historically, both the capsule and the subscapularis were tightened (Putti-Platt or Magnuson-Stack). Although the Putti-Platt repair had high levels of success in treating instability, it resulted in increased joint reaction forces and was associated with a high incidence of joint degeneration.

Arthroscopic procedures: In an arthroscopic repair the anatomical lesion is identified and repaired. The most common variation is the Bankart repair. The labrum is elevated from the anterior glenoid neck; the neck is prepared to bleeding bone and the labrum repaired using two or three bone anchors to the face of the glenoid. In addition to the labral repair, the capsule is re-tensioned. This can be achieved in a variety of ways, including reefing of the capsule or attaching the repair to the glenoid face rather than the glenoid edge and shifting the capsule superiorly. This type of repair can also be utilised for ALPSA and Perthes lesions.

A HAGL can also be treated arthroscopically. In this situation, the torn inferior capsule is repaired with bone anchors to the humerus.

Rehabilitation after surgery

The role of rehabilitation after stabilisation surgery moves through three phases:

Phase 1: Protection of healing repair
Phase 2: Regain normal ROM
Phase 3: Regain power, endurance and shoulder control to allow return to desired function.

Conclusions

Traumatic anterior shoulder instability is an evolving area, but there are helpful guidelines to facilitate decision making in the clinical environment.

It is essential to assess the patient carefully and to establish which structures are likely to have been injured. The patient can be mobilised early. The decision on further imaging should be based on the likelihood of the need for surgical intervention. Once a patient has developed recurrent instability it is essential to make on the type of surgery to perform in an effort to avoid recurrence.

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