Managing patients with shoulder instability

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Abstract

The shoulder is the most mobile joint in the body but, as a consequence is also the most unstable. Stability is aided by boney, ligamentous and muscular structures and as a result may be related to trauma, hyper mobility or muscle patterning. Acute management requires careful history and examination with gentle reduction by a number of means and then may be treated conservatively or surgically. There is a high rate of recurrence with the conservatively treated shoulder in the younger population. Over 150 surgical procedures have been described to treat recurrent shoulder instability and these range from open 'anatomic' repair to tightening procedures though bone/coracoid ligament transfers and arthroscopic procedures. Several factors have been identified to help guide decision-making and these are summarized in the ISIS score which can be used to aid decision-making in these challenging patients.

Keywords dislocation; instability; shoulder; stabilization

Introduction

Shoulder instability can be thought of as a spectrum of conditions and is defined as a symptomatically abnormal movement of the humeral head on the glenoid fossa.[1] Patients with mild instability may experience pain or apprehension whereas more moderate translation of the humeral head causes subluxation and the feeling of the shoulder slipping in and out. The most extreme form of instability is frank dislocation of the glenohumeral joint.

Anterior dislocations occur in a bimodal distribution affecting 1.7% of the population[2] with peaks in the third and ninth decades. For some patients it is an isolated event but for others the damage sustained by the shoulder stabilizers during dislocation can lead to recurrent instability. This is more common in males and athletes[2,3] with the main risk factors being age, return to contact or collision sports, and the presence of a significant defect in the glenoid or humeral head.[3] Multiple series recognize that age is the most important predictor for recurrent instability, with patients under the age of 20 years old having a recurrence rate of up to 90%.[3–5]

The aim of this review article is to provide the reader with an evidence-based approach to managing patients who present with acute and recurrent shoulder instability.

Anatomy

As with all joints there are static and dynamic stabilizers but, due to the unique requirements of the shoulder, it is inherently less stable in order to allow such a wide range of motion. Compared to other joints the shoulder is much more reliant on its dynamic stabilizers, which provide stability through a complex interplay of muscle forces controlled through a feedback mechanism from proprioceptors located within the capsule and surrounding soft tissues.[6]

The static stabilizers include:

1. Glenoid and it's surrounding labrum
2. Differential distribution of articular cartilage and labrum
3. Glenohumeral ligaments (GHLs)
4. Negative intra-articular pressure.

The dynamic stabilizers include:

1. Rotator cuff
2. Long head of biceps tendon.[7]

In order to permit such a wide range of movement the various GHLs cannot be tight throughout the range of motion, but instead contribute to stability depending on the position of the shoulder joint, usually only becoming taut at the extremes of shoulder range (Table 1). They also exhibit a relatively low load to failure of 585 N[8] compared with more than 2000 N seen in the anterior cruciate ligament (ACL), for example.[9]

Pathological lesions arising from instability represent failures of these constraints and the structures that fail depend upon the position the shoulder is in at the time of dislocation, as well as the age of the patient and the energy imparted. With all of the soft tissue lesions there will be plastic deformation occurring before failure, the degree of which will be determined by the biomechanical properties of the tissue in question as well as the degree and rate of load application. It has been postulated that there is more plastic deformation of the soft tissues in younger patients before failure, resulting in a permanent loss of their stabilising properties even if repaired whereas in the elderly there is less plastic deformation before failure, as their tissues are less compliant.[10] This may partially explain the increased rates of shoulder instability in the younger patient and also the increased incidence of rotator cuff failure in the elderly.

It is important to have an understanding of the various pathological lesions associated with anterior dislocation, as these are important when considering both conservative and surgical management.

Bankart lesion: In 80–90% of patients with an anterior dislocation there is an avulsion of the anterior labrum and anterior band of the inferior GHL from the anterior inferior glenoid. This was originally described in 1906 by Perthes as the 'essential lesion', although it was Bankart in 1938 who further studied the effect of an acute anterior dislocation and described the detail of what we now understand.

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Static shoulder restraints

Structure	Arm position	Restraint
Superior glenohumeral ligament (GHL)	Adduction	Inferior translation External rotation
	Flexion, adduction and internal rotation	Posterior translation
Middle GHL	Adduction	External rotation
	Adduction and external rotation	Inferior translation
	45° abduction and external rotation	Anterior and posterior translation
Inferior GHL — Anterior band	90° abduction and external rotation	Anterior and inferior translation
Inferior GHL — posterior band	90° abduction and internal rotation	Posterior and inferior translation
Rotator interval (superior GHL and coracohumeral ligament)	Adduction	Inferior translation
Posterior capsule	Flexion, adduction and internal rotation	Posterior translation

Table 1

Bony Bankart: A compression fracture of the anterior glenoid. While less common than soft tissue Bankart lesions, they are still found in 45% of patients with recurrent dislocations. Burkhart and DeBeer,[11] Sugaya[12] and Itoi et al[13] have all shown that glenoid bone loss of more than 20% results in increased recurrence rates because the safe arc that the glenoid normally provides for the humeral head to articulate with is reduced.[11]

Humeral avulsion of the glenohumeral ligament (HAGL): This is associated with a slightly older patient group and is a risk factor for ongoing instability. It may also be a reason for primary surgical repair if identified.

Glenoid labral articular defect (GLAD): This represents a sheared off portion of articular cartilage along with the glenoid labrum and is similar to the bony Bankart except there is no bone attached to the piece of articular cartilage.

Anterior labral periosteal sleeve avulsion (ALPSA): This is essentially a more severe form of a soft tissue Bankart lesion where the periosteum attached the labrum peels off along the glenoid neck medially and if it is not reduced then it can result in healing of the labrum medially increasing the risk of instability.

Hill–Sachs defect: This is a chondral injury in the posterosuperior humeral head secondary to impaction onto the anterior glenoid rim during dislocation and is found in 80% of recurrent dislocations and 25% of first-time dislocations. The size (both in diameter and depth) as well as location on the humeral head have an influence on stability and the success of any soft tissue surgical procedures (see tracking section later).

Greater tuberosity fracture: This is associated with dislocations in the older patient population and usually remains reduced once the shoulder is relocated. Bony healing restores stability.

Rotator cuff tear: As with greater tuberosity fractures this is usually associated with older patients. Approximately 30% of patients between 40 and 60 years and 80% of patients over 60 years of age will sustain a rotator cuff tear when they dislocate, which can result in rotator cuff dysfunction and secondary pathology as a result.

Classification

In order for the shoulder to dislocate there must be a failure of the static or dynamic constraints and this can occur in one of four ways:[10]

1. A force of sufficient load or rate of application is applied to the shoulder resulting in failure of both static and dynamic stabilizers.
2. There is a structural abnormality, as a result of previous trauma, that reduces the forces required to overcome the natural stabilizers.
3. There is a congenital absence or dysfunction of the constraints.
4. The feedback loop can become damaged resulting in failure of the dynamic stabilizers to function properly despite 'normal' anatomy.

Matsen's[14] simplified classification is useful to think of two broad categories of patients: TUBS - Traumatic, Unilateral, Bankart, (requires) Surgery and AMBRII - Atraumatic, Multidirectional, Bilateral, (requires) Rehabilitation, (or) Inferior capsular shift and Interval closure. However, this classification is limited in that it does not account for those patients with normal anatomy but abnormal dynamic stabilizers due to damage to their feedback loop (number 4, above). This classification also implies that if rehabilitation fails then surgery is appropriate, however we know that surgery for the muscle patterning types (see below) results in very poor outcomes.

The Stanmore triangle (Figure 1) was developed in response to the recognition that the Matsen classification did not include the full spectrum of instability and to help further classify shoulder instability into three Polar groups. It also recognizes that patients can not only have a combination of pathology (e.g. lying on the axis between Type I and Type II) but may also move along the axes with time. The Polar type I group represents the acute traumatic structural group analogous to Matsen's TUBS group. Polar type II represents the atraumatic structural group, analogous the Matsen's AMBRII group. The polar type III group are the muscle patterning, non-structural group and represents those patients with a failure in the feedback mechanism. It is important to recognize this distinct group because, whilst groups I and II may benefit from surgery, the group III patients are unlikely to benefit and may come to harm with surgery, with an increased risk of arthritis and rehabilitation failure.[15]

Clinical assessment

It is important to take a thorough history and examination focusing on the patient's age at first dislocation, mechanism of

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