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Review Article

Clinical assessment of posterior shoulder joint instability

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ABSTRACT

Posterior shoulder instability is less common than anterior and is not as readily recognised. There are numerous clinical tests for posterior instability. They all have benefits and disadvantages, depending on the type of instability and strength of the patient. In this article we describe the most common clinical tests for posterior instability and review the literature supporting each test. In this manner, we hope that this will provide the clinician with a better understanding of each test and it's value.

1. Introduction

The shoulder is capable of the widest range of movement of all joints: for these to be normal and asymptomatic they depend on the interaction of both static and dynamic stabilisers of the shoulder. Static stabilisers include the bony anatomy, the glenoid labrum, the negative intra-articular pressure, the joint capsule, and the glenohumeral ligaments. The dynamic stabilisers are the muscles of the rotator cuff, and those surrounding the joint.[1] Unlike the hip and knee joints, the shoulder glenoid fossa is shallow. Glenohumeral stability from the glenohumeral ligaments of the capsule is effective primarily when the range of motion is at the extremes.[2] To have extensive movement at the glenohumeral joint the ligaments are required to be relatively lax. This requires combined involvement of dynamic and static stabilisers through range of motion.

The shoulder also benefits from the concavity compression mechanism, where the convex head of the humerus is compressed into the concave glenoid fossa to stabilise it against translating forces. The depth of the concavity and the magnitude of the compressive force influence joint stability with the depth of the bony glenoid being significantly less anteroposteriorly (2.5 mm) than superoinferiorly (9 mm), hence the stability against anterior and posterior forces was less than inferiorly and superiorly directed forces.[3] The labrum is a fibrocartilaginous ring around the glenoid increasing the depth of the glenoid upto 50%, contributing to the concavity compression mechanism.[4] The labrum also works alongside the synovial fluid to form a suction effect by adhesion-cohesion forces, providing stability to the articulation.[5] The negative intra-articular pressure also contributes to this effect and centres the humeral head into the glenoid. The attachment points for the glenohumeral ligaments and the long head of biceps arise from the labrum.

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The glenohumeral ligament structure consist of three parts; the superior glenohumeral ligament (SGHL), which resists translation inferiorly with the arm adducted and in neutral rotation; the middle glenohumeral ligament (MGHL), an anterior stabiliser in adduction and the inferior glenohumeral ligament complex. This comprises the anterior band of the inferior glenohumeral ligament (IGHL), which is the primary static stabiliser in a neutral position; and the posterior band of the IGHL (PIGHL), the primary static posterior stabiliser when the arm is flexed and internally rotated. The coracohumeral ligament (CHL) resists posterior and inferior translation when the shoulder is suspended and inferiorly when the arm is adducted.[1] Tension in the ligaments and capsule provide additional proprioceptive feedback to the rotator cuff muscles helping to prevent abnormal joint translation.[6]

The rotator cuff muscles have independent actions that in combination contribute to stability during mid and end range motions of the glenohumeral joint, working in both a concentric and eccentric manner. The rotator cuff muscles also provide compressive force across the joint, helping to centralise the humeral head in the glenoid fossa.

Injury to either the static or dynamic stabilisers of the shoulder may compromise function resulting in instability. In general terms this can be anterior, posterior, multi-directional, traumatic or atraumatic. We like to use the Stanmore classification system, which is based on three polar groups – traumatic structural, atraumatic structural and habitual non-structural (muscle patterning).[7] Basing these three poles as the points of a triangle it is possible to establish a continuum where a patient may fit into one of the three groups, or as is often the case, overlapping and moving between more than one group.

2. Pathogenesis

Posterior instability is less common than anterior instability, and accounts for between 2 and 12% of cases of instability.[8,9] It was typically described as occurring in patients who have experienced posterior dislocation due to seizures, electrocution. In an anatomically normal shoulder it is now considered in three broad etiological categories: acute trauma, repetitive microtrauma and purely atruamatic.[10–12] The most frequent cause being repetitive microtrauma to the posteroinferior shoulder complex often seen in young, active people performing activities such as bench pressing, rugby, rowing and swimming.[13] These activities result in repetitively loading the glenohumeral joint in a flexed internally rotated position, stretching and injuring the PIGHL and posterior labrum. Anatomical abnormalities in glenoid version, hypoplasia and humeral retroversion can also contribute.[8,14,15] We have also found traumatic posterior instability in a high number of contact athletes [REF].

3. Clinical assessment of the posteriorly unstable shoulder

The basis of diagnosing posterior instability is a careful history and physical examination of both the symptomatic and asymptomatic shoulders. Factors to bear in mind during assessment include:

- How the problem affects their activities of daily living
- How the problem affects their work or sporting lives
- What pathology is present or likely to be present
- An appropriate management plan

Often the diagnosis is not clear and several shoulder complaints can arise from different shoulder relate disorders. The primary complaint is often an aching pain with weakness located around the posterior joint line, biceps tendon or superior aspect of the cuff. The physical examination aims to reproduce the symptoms experienced by the patient. Often in cases of posterior instability symptoms are exacerbated with the arm placed in 90° flexion, adduction and internal rotation.[16]

The patient should be assessed for generalised laxity using the Beighton Score. A score of 6/9 or greater indicates hypermobility but not necessarily benign joint hypermobility syndrome.[17] Throughout the clinical assessment it is necessary to bear in mind the difference between laxity and instability. Lax patients can have the same degree of glenohumeral translation as an unstable patient but report no symptoms or discomfort.[18] In fact ligamentous laxity is often seen in athletes where it may provide an advantage in their sport, but this can be associated with an increased incidence of joint instability, for example in rugby union players, laxity in the shoulder joints may confer increased risk for dislocation.[19]

4. Clinical tests for posterior laxity

4.1. *Posterior drawer test*

In 1984 Christian Gerber and Reinhold Ganz discussed the lack of attention in the literature of clinical diagnosis of shoulder instability; instead most accounts were focussed on the surgical procedures themselves.[20] They attributed some of the failures of the surgeries to not adequately detecting anterior and posterior instabilities and so described the anterior and posterior drawer tests. The posterior drawer test requires the patient to be supine with the examiner level with the shoulder, the proximal forearm is held by the examiner who then flexes to the elbow to approximately 120° and moves the shoulder to be abducted from 80° to 120° and flexed forward of 20°–30°. Holding the scapula with the other hand, with the thumb placed lateral to the coracoid process. The humerus is then slightly medially rotated and flexed further to 60° or 80°, the thumb placed lateral to the coracoid subluxes the head of the humerus posteriorly which can be felt by the fingers behind the shoulder. The patient often responds with apprehension when this is performed. There is a lack of published research showing sensitivity and specificity figures for this test (Fig. 1).

4.2. *The load and shift test*

The load and shift test examines glenohumeral translation and should be performed with the patient sitting in an upright neutral position and also supine.[20] With the examiner behind the shoulder a hand over the scapula helps to stabilise it and then the humerus is held and “loaded” into the glenoid fossa