



ELSEVIER

available at www.sciencedirect.com



journal homepage: www.elsevier.com/locate/cuor

Current
ORTHOPAEDICS

SHOULDER

The clinical assessment and classification of shoulder instability

A.M. Hill ^a, A.M.J. Bull ^{a,*}, J. Richardson ^d, A.H. McGregor ^d, C.D. Smith ^a,
C.J. Barrett ^{b,c}, P. Reilly ^b, A.L. Wallace ^c

^a Department of Bioengineering, Imperial College London, South Kensington Campus, London SW7 2AZ, UK

^b Department of Musculoskeletal Surgery, St. Mary's Hospital, Paddington, London, UK

^c The Shoulder Unit, Hospital of St. John and St. Elizabeth, London, UK

^d Musculoskeletal Surgery, Department of Surgery, Anaesthesia & Intensive Care, Charing Cross Hospital, Fulham Palace Road, London, UK

KEYWORDS

Shoulder instability;
Capsulolabrum;
Glenohumeral joint;
Functional anatomy;
Clinical assessment;
Testing algorithm

Summary

Assessment of the glenohumeral joint relies upon a detailed, time-dependent history of the presenting complaint and a thorough examination of its subtle signs. As such, the complex is most appropriately approached as a unit, rather than as a discrete number of examinable components. However, understanding the interactions between these components means that a high level of skill is needed to adequately assess the joint, and learning these skills is compounded by the large number of eponymous tests described for examining separate elements.

The stabilising mechanisms of the shoulder may crudely be classified as passive (non-contractile) or active (contractile) in function; this artificial distinction neglects the role of contractile tissue in maintaining stability whilst not contracting, but serves to facilitate understanding, and indeed categorise modes and methods of physical examination. Indeed, modes of failure are specific to these groupings. Determining the degree of instability caused by the passive stabilising mechanisms is commonly fraught with both intra- and inter-rater discrepancy, and as such, requires a great deal of experience to implement and interpret. However, an evidence-based approach to a clinical examination sequence can improve its predictive value.

A critical review of the literature on examination of passive stabilising mechanisms is presented, followed by a distillation of current concepts resulting in the presentation of an evidence-based approach to examination that is practical, and can be implemented successfully by all involved in the rehabilitation process.

Crown Copyright © 2008 Published by Elsevier Ltd. All rights reserved.

* Corresponding author.

E-mail address: a.bull@imperial.ac.uk (A.M.J. Bull).

Introduction

The skeletal system is composed, in its most fundamental form, of bones or segments connected by joints. The bones provide distance between joints in order to maximise muscle efficiency whilst joints act as the interface of relative motion between these bones.¹ Joints can be classified according to the amount of movement available. For example, whilst the glenohumeral joint has six 'degrees of freedom', the acromioclavicular and sternoclavicular joints are relatively constrained in one or more planes.

Shoulder motion

The movement of the upper limb can be described in many ways. Clinically, the most appropriate are humerothoracic motion, or movement of the upper arm relative to the thorax, and scapulohumerothoracic rhythm, which recognises the role played by the scapula. Typically, the term glenohumeral motion has been confused with these descriptions; movement of the individual joints of the shoulder complex can be considered independently, and as such, a distinction should be made between joint motion descriptions, and those of the entire shoulder complex.

Humerothoracic motion

Traditionally, arm elevation is considered clinically in three planes (Fig. 1); the frontal (abduction), sagittal (flexion) and scapular (scaption, or 30° anterior to the frontal) planes.² Humerothoracic motion describes movements of the shoulder as abduction, adduction, flexion, extension and axial rotation. Elevation is defined as movement of the humerus away from the side of the thorax in any plane; forward flexion is shoulder elevation in the sagittal plane, scaption as elevation in the scapular plane, whilst abduction is elevation in the frontal plane. Abduction in the plane of the scapula, or scaption, has been discussed as the most functional form of elevation,^{3,4} as it is considered that this

plane does not deform the inferior glenohumeral joint (GHJ) capsule, and the deltoid and supraspinatus muscles are optimally aligned for elevation of the arm.

Scapulohumerothoracic (SHT) rhythm

The SHT rhythm, since initially described by Codman,⁵ has evoked much interest. It has been described as both an open-chain^{6,7} and closed-chain mechanism⁸; that is, a mechanical linkage constrained at one end, and a mechanical linkage constrained at both ends, respectively. Each mechanism has 3 *true* independent articulations—glenohumeral, acromioclavicular and sternoclavicular—and one *false* articulation (the scapulothoracic gliding plane). Each joint, whilst capable of independent motion, contributes to the normal function of the upper extremity, participating in a simultaneous, rather than successive, manner.⁹ Inman and others⁹ described a planar scapulohumeral rhythm as the ratio of humeral rotation to the movement of the scapular spine (Fig. 2). The published magnitude of this ratio ranges from 2.3¹⁰ to 3.2⁹, although Saha¹¹ calculated a ratio of 3.7 when measuring scapula rotation from the medial ridge (Table 1).

Glenohumeral joint kinematics

Movement about the GHJ, described as a 'beach ball balancing on a seal's nose'¹² can be uniquely described to consist of both 3 rotations and 3 translations. Rotations about the joint are in the abduction and flexion planes as well as about the long humeral axis, termed axial rotation. Although rotations at the articulation are widely accepted, the degree of translation is often neglected within the non-pathological population. This is due to the limited magnitude of translations in a stable joint. Translations of up to 3 mm in the superior direction have been reported in the first 60° of abduction in the scapular plane,^{13–17} and up to 1 mm after this point.^{13,14,16,18,19} Supine abduction has been reported to incur inferior translation,¹⁸ whereas anterior translations of up to 5 mm have been documented during passive glenohumeral

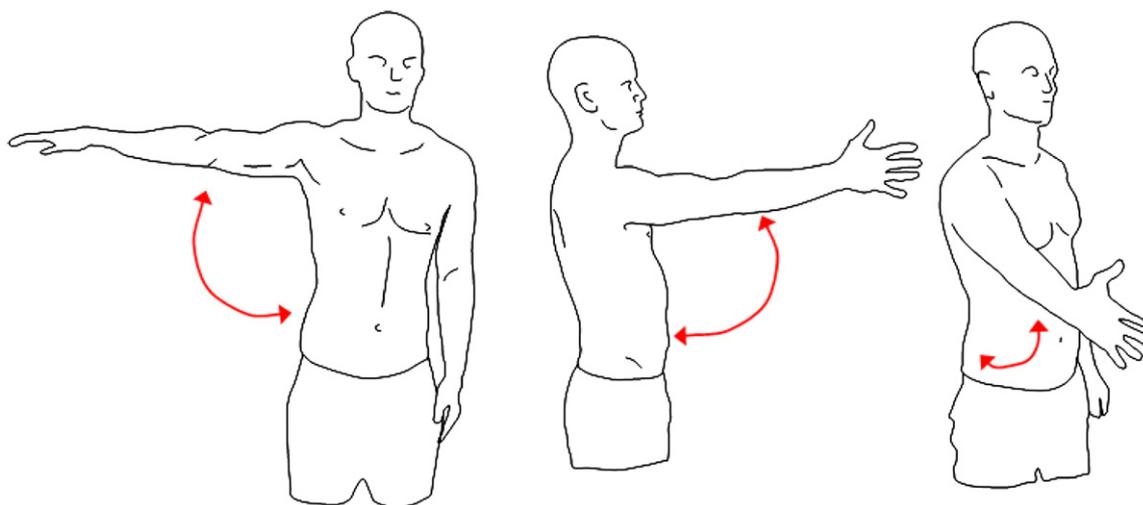


Figure 1 Diagrammatic representation of elevation in 3 planes. From left to right: elevation in the frontal (abduction) plane, sagittal (flexion) plane and scapular (scaption) plane.

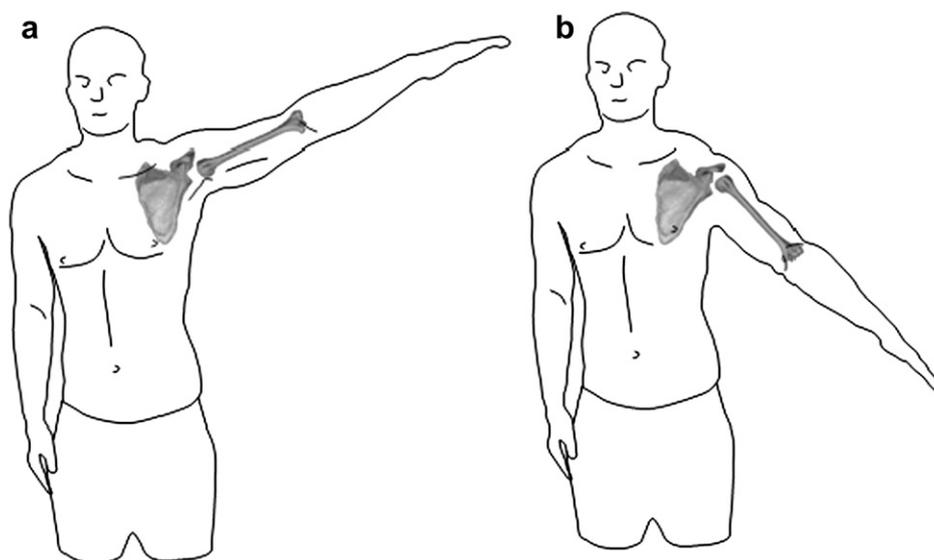


Figure 2 Diagrammatic representation of the scapulohumeral rhythm: (a) illustrates 120° of humerothoracic abduction, but approximately 90° of scapulohumeral abduction, whilst (b) demonstrates a similar mismatch in humerothoracic and scapulohumeral angles at lower degrees of abduction.

flexion.^{20,21} However, similar movements at a GHJ with a lesser degree of stability, either traumatic or atraumatic in aetiology, are frequently significantly larger in magnitude; translations up to 19 mm anteriorly, 18 mm posteriorly and 20 mm inferiorly have been recorded in the most extreme atraumatic multidirectional instabilities.^{22–24}

Scapulothoracic joint motion

Although the pattern of scapular motion is constrained by geometry, muscular activity and the articulating rigid clavicle, a constrained spatial motion has been described including 3 rotations and 2 translations.^{25,26} Due to the functional anatomy of the scapula, scapula distraction–compression along the axis perpendicular to the plane of the scapula – is negligible unless the whole segment acts outside of its gliding plane. Scapula motion can be altered by surrounding soft tissue constraints; for example, abnormal muscle coordination may result in scapula ‘dyskinesia’

(abnormal scapular motion). In this condition, it may be advantageous to record spatial, or three-dimensional motion.

The scapula has been investigated in a number of different fashions; early studies used two-dimensional methods,^{9,13} whilst more recent work has appreciated the spatial nature of motion.^{8,27–31} Van der Helm and Pronk⁸ initially described 3 rotations of the scapula: upward rotation about an anteroposterior axis, external rotation about a superoinferior axis and posterior tilt about a mediolateral axis. During scapular plane elevation in healthy individuals, the scapula upward rotation increases and tilt occurs from anterior to posterior consistently,^{32–34} whereas external–internal rotation occurs less. Upward rotation is described as the predominant motion during both scapulohumeral abduction in the scapula plane and forward flexion: McClure and colleagues²⁵ directly measured 50° of scapular rotation, 30° of posterior tilting, and 24° of external rotation during 120° of abduction (Fig. 3).

Table 1 Tabulation of estimates of the scapulohumeral rhythm from available literature

Study	Movement plane	Ratio (GH:ST)
Inman et al. ⁹	Frontal	2:1 (between 30° and 170°)
Saha ¹¹	Scapula	Variable (between 0° and 30°)
Freedman and Munro (1966)	Scapula	2.3:1
Poppen and Walker ¹³	Scapula	1.5:1 (between 0° and 135°)
Bagg and Forest ¹⁰	Scapula	1.3:1 (After 30°) 4.3:1 (between 0° and 30°)
Michiels and Gravenstein (1995)	Scapula	4.3:1 (between 20.8° and 81.8°) 1.7:1 (between 81.8° and 139.1°) 4.49:1 (above 139.1°)
Sugamoto et al. (2002)	Scapula	2:1
	Scapula	2.4:1 (at 60° of low-speed humerothoracic abduction) 2.3:1 (at 150° of low-speed humerothoracic abduction) 2.9:1 (at 60° of high-speed humerothoracic abduction) 1.70:1 (at 150° of high-speed humerothoracic abduction)

Some authors describe a mean value for the entire range of motion, whilst others recognise the effects of angular dependency and velocity. (GH:ST is the glenohumeral to scapulothoracic ratio.)

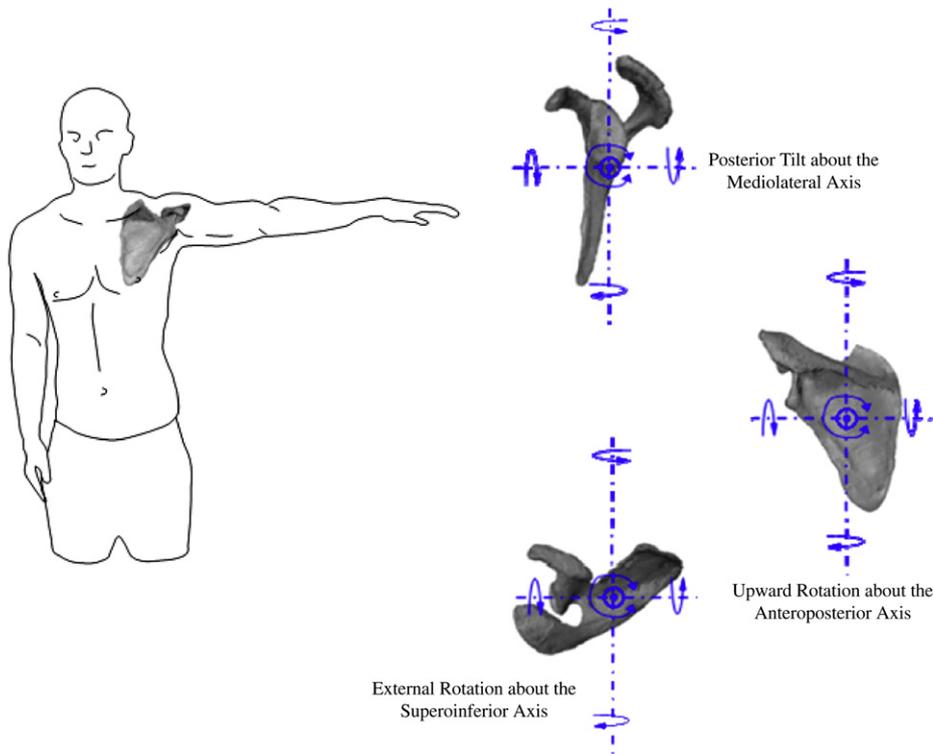


Figure 3 Rotations of the scapula. From top to bottom, posterior tilt occurs about a mediolateral axis, upward rotation about an anteroposterior axis and external rotation about a superoinferior axis.

Translations of the scapula have been less well considered, possibly due to its intimate relationship with the rigid-bodied clavicle, tethered by the acromioclavicular joint,³⁰ which has 3 degrees of freedom, and the relatively well constrained gliding plane of the thorax. Scapula positions have previously been considered as sternoclavicular rotations in orthogonal planes: elevation–depression of the clavicle representing superoinferior translation of the scapula, and clavicular protraction–retraction intimating anteroposterior translation of the scapula²⁶ (Fig. 4).

Acromioclavicular and sternoclavicular motion

Both acromioclavicular and sternoclavicular joint motion have had limited consideration, probably due to the limited motion seen relative to both the glenohumeral and scapulothoracic joints of the shoulder complex.

The acromioclavicular joint is enveloped by capsule, thickened superiorly, anteriorly and posteriorly, restricting rotation during elevation to about 20°. The paired sternoclavicular ligaments are primary restraints to rotation during depression of the clavicle,^{35,36} whilst lateral and medial displacement of the clavicle is resisted by anterior and posterior costoclavicular ligaments.³⁷ The range of motion of the sternoclavicular joint is approximately 30–35° in upward elevation, approximately 35° in the anteroposterior direction and between 44° and 50° in axial rotation.⁹ Thus, commonly, the motions of the clavicle are described as elevation–depression, protraction–retraction and axial rotation (Fig. 4). Translations can occur, especially post-trauma, although to a lesser degree than those of the GHJ.

Clinical instabilities

Stability of the shoulder complex relies upon both active and passive stabilisation mechanisms. Clinically, glenohumeral instability can be defined as a ‘condition in which

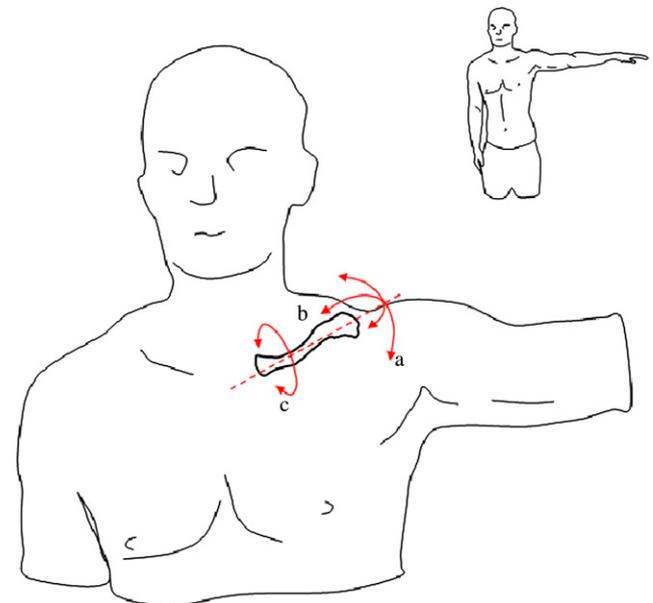


Figure 4 Diagrammatic representation of clavicle elevation–depression (a), clavicle protraction–retraction (b) and clavicle axial rotation (c). The range of motion of the sternoclavicular joint is approximately 30–35° in upward elevation, 35° in the anteroposterior direction and 44–50° in axial rotation.

unwanted translation of the humeral head on the glenoid compromises the comfort and function of the shoulder'.³⁸ Each year, 30–40% of adults experience shoulder discomfort causing 1–5% of them to access primary medical care.^{39,40} Shoulder instability is a common, debilitating condition; the shoulder is the most commonly dislocated joint in the body.⁴¹ The mean age range of primary presentation is 15–40 years old, with bimodal incidence peaks in the 2nd and 6th decades.⁴² These coincide with the main peaks in lifetime relative wealth.⁴³ Therefore, this condition is of significant socio-economic consequence. Although the majority of 'unstable' events occur anteriorly, posterior instability has all too often been unrecognised due to the subtlety of diagnosis; one recognised aetiology in this condition that is less subtle in its presentation is the sudden, strong internal rotation forces generated by the latissimus dorsi, pectoralis major, and subscapularis muscles during abnormal electrical stimulation as occurs in seizures or electrocution, overpowering the relatively weaker external rotator muscles.⁴⁴

Maintenance of stability

GHJ stability is maintained through an intricate interplay between a number of static, or non-contractile, and dynamic, or contractile, structures and physiological mechanisms. At this juncture, it must be acknowledged that a potentially contractile anatomical structure can also provide stability when momentarily non-contractile; the terminology used refers to those structures that have the potential to dynamise, and those that do not, are termed static. Indeed, these terms also provide distinction between the modes of eventual stability failure. The joint relies upon both the anatomy and function of local soft tissues to prevent excessive translations. Dynamic stability

of the joint may be coordinated by higher cortical control of local musculotendinous units, providing direct stability by generating a joint reaction force and maintenance of optimal scapulohumeral balance⁴⁵ through proprioceptive feedback. The end effectors of such control are the rotator cuff musculotendinous unit and associated periscapular and shoulder girdle musculature. Passive stability is mainly conferred by the glenoid labrum and capsuloligamentous components, although the elastic properties of the shoulder musculature may confer a significant degree of passive stability, in particular at the extremes of the range of motion. Negative intra-articular pressure^{46–48} generated by an interaction between capsule and synovial fluid is a further physiological mechanism of stability (Fig. 5), the relative significance of which is debatable.

Classification of instability

Due to temporal drift of concepts in joint instability, a number of different classification systems have been proposed over time. Invariably, these classifications encompass both contractile and non-contractile components of stability. Rockwood⁴⁹ initially described a simple system focusing on traumatic aetiology with or without previous dislocation (Types I and II) or atraumatic voluntary (Type IIIa with psychiatric problems and Type IIIb without psychiatric problems) and involuntary (Type IV) subluxation. However, the system lacks the subtlety to determine modes of trauma which may have a bearing on management and outcome as well as distinguishing between mixed or time-dependent pathologies. Further simplifying the spectrum of instability, Thomas and Matsen⁵⁰ neglected volition and proposed a management algorithm based upon the acronyms of traumatic unidirectional Bankart lesion treated with surgery (TUBS), and atraumatic multidirectional

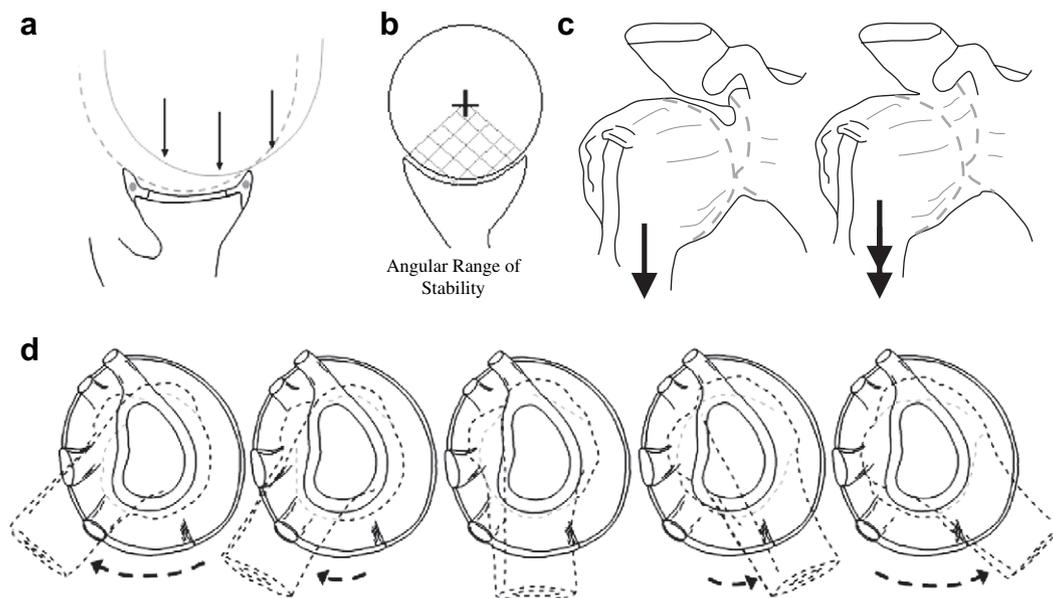


Figure 5 Mechanisms of glenohumeral stability: (a) demonstrates the effect of the concavity depth on stability, (b) the angular range of stability resulting from glenohumeral balance, (c) demonstrates the effect of limited volume and negative intra-articular pressure, whilst (d) the role of articular compression due to soft tissue tensing during rotation.

bilateral treated with rehabilitation and possibly inferior capsular shift (AMBRI). This classification may misplace 2 patient groups; those with a muscle patterning component, and those non-hyperlax individuals with acquired overuse, commonly as a result of sporting activity.

Schneeberger and Gerber⁵¹ developed a classification system in which the degree of joint laxity, trauma and instability were defined, but this system again marginalises volition. More recently, Gerber and Nyffeler⁵² refined the definition of instability by describing 3 classes of instability; static, defined by the absence of classic symptoms yet characterised by humeral head displacement, dynamic, in which a subjective loss of normal glenohumeral stability and momentary, but restorable loss of articular congruency is described, and voluntary, reserved for those who may dislocate at will. However, a conclusive model was never presented.

The development of the Stanmore classification has been influenced by the limitations of all pre-existing systems. Patient presentations are grouped into 3 polar classes: Type I (true TUBS), Type II (true AMBRI), or Type III (muscle patterning or habitual non-structural disorders). In recognition of the continuity between groups, a triangular model was proposed in which a degree of polarity may be tailored to each presentation⁵³ (Fig. 6). As such, the Stanmore classification provides a system to group all presentations of shoulder instability irrespective of time dependency. However, its commonest limitation is the difficulty in defining axial groups. Although polar groups are more straightforward, inherent within axial group description is a significant degree of both inter- and intra-rater error.

Clinical examination

Specific evaluation of both anatomical and functional components of the disrupted shoulder has been described through the use of a number of eponymous tests. Subsequent review of the sensitivity (the proportion of patients with an anatomical or functional deficit who test positive with a test aimed at eliciting such a deficit), and specificity (the proportion of patients without an anatomical or

functional deficit who test negative with the same test), have often conflicted with the validity described by the presenting authors. Similarly, positive predictive value (PPV, the probability that a patient has an anatomical or functional deficit with a test positive), and negative predictive value (NPV, the probability that a patient has no anatomical or functional defect with a negative test result), is commonly incongruent between test description and later peer review. However, clinical assessment of the shoulder relies significantly upon a generic history of the injury, and musculoskeletal examination.

Before conducting a physical examination, both a general and specific shoulder history should be taken, including an evaluation of the presenting complaint, be it pain, a sensation of instability, stiffness, deformity, locking, catching or swelling. An understanding of the patient's functional loss (i.e. an inability to compete at a chosen level, possibly due to a lack of confidence in the stability of their shoulder, or a 'dead arm' sensation) as well as previous treatment, surgery and injury is also of vital importance in both diagnosing and managing any shoulder instability. In addition, a family history of atraumatic instability may be important in these patients, as is an assessment of general flexibility compared to their peers.

Physical examination of the unstable shoulder requires an understanding of its limitations; instability may be obvious or subtle and difficult to demonstrate. Two important caveats exist to elucidating positive instability tests; firstly, a normal shoulder has a degree of physiological laxity,²⁰ and secondly, it is difficult to determine whether a translation under manual guidance is subluxation to an abnormal position or relocation to a normal position.⁵⁴ However, it must be recognised that this is of theoretical relevance clinically, as both can represent a pathological condition. As such, not only is bilateral assessment of vital importance, but also testing for generalised hyperlaxity, as evidenced by elbow, finger and thumb hyperextension, together with knee recurvatum and increased ankle dorsiflexion.⁵⁴ Although there is a known relationship between shoulder instability and gross connective tissue disorders such as Marfans and Ehlers-Danlos Syndrome,⁴¹ there is far less understanding about the relationship between generalised joint laxity and shoulder laxity.

Of fundamental importance in evaluating passive restraints to glenohumeral motion is relaxation of the active muscular restraints. A tendency to guard the injured joint and prevent examination may be overcome by conducting laxity examination on the contralateral side before the involved limb. Following this, provocation testing can often be conducted with a little more ease.

Laxity tests

By definition, laxity tests should not precipitate symptoms, but demonstrate the degree of translation at the joint. The amount of translation on laxity testing is determined by the length of the capsuloligamentous complex at the beginning of the assessment, and thus the originating position of the humerus; for example, a greater degree of anterior laxity may be elucidated if the arm is in internal rotation compared to an externally rotated position. Three tests

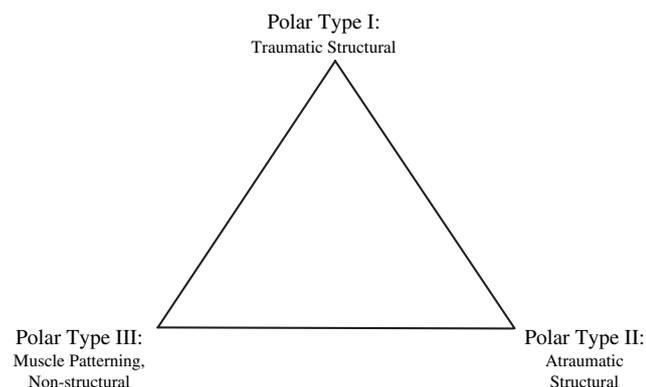


Figure 6 Stanmore triangle (developed by Bayley et al., 1986–2004; adapted from Lewis et al.⁵³). The triangular polarities represent extreme types of instability. In practice, instabilities fall within the triangle.

of laxity are commonly performed: the load-and-shift test, the drawer test and the Sulcus sign. These tests are not specific for particular pathologies, but are intended to provide evidence of excessive laxity.

Load-and-shift

The load-and-shift test was first described by Hawkins and colleagues,⁵⁵ although modified in recent years. The principle of this test is to determine the amount of translation of the head of the humerus on the glenoid. Whether the patient is positioned supine or seated, the examiner uses their first hand to grasp the humeral head of the affected shoulder, and position their second hand over the shoulder girdle in order to stabilise the scapula. Simultaneously, the posterior joint line is palpated with the thumb, whilst the anterior joint line is palpated with the index and middle fingers, positioned over the coracoid and the humeral head, respectively. The first hand then loads the joint to ensure concentric reduction before applying an anterior or posterior shearing force, the magnitude of which may be appreciated as the index finger approaches the middle finger (Fig. 7). Similar tests to the method described have been presented in the literature.⁵⁶

The reliability of this test was found to be optimal when tested in 0° abduction for the posterior and inferior directions⁵⁷ (intraclass correlation coefficient (ICC), for inter-rater reliability and test–retest reliability was 0.68 and 0.79, respectively). By abducting to 90°, the anterior direction reliability is also good (ICC: 0.72).

Drawer tests

Akin to the load-and-shift principle, drawer tests, first described in the shoulder by Gerber and Ganz,⁵⁸ are commonly used to assess anteroposterior laxity. These tests share a common grading system with the anterior drawer

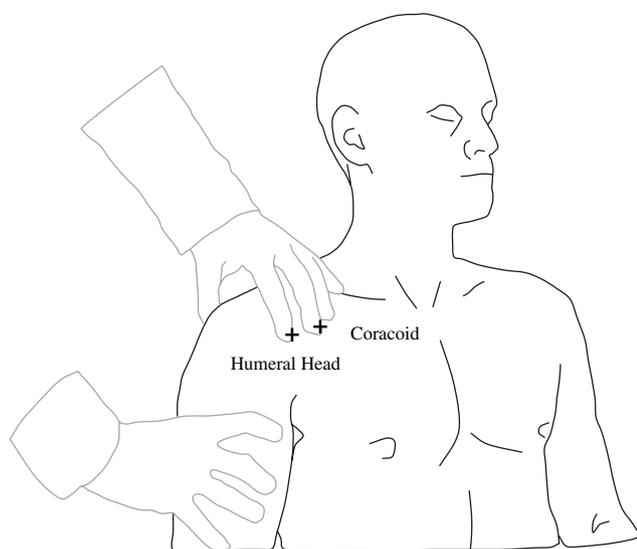


Figure 7 Load-and-shift test. The first hand of the examiner is used to manipulate the humeral head, whilst the second hand is used to stabilise the scapula.

test of the knee.⁵⁹ However, unlike the load-and-shift test, anterior and posterior drawers must be conducted with the patient supine, as the reproducibility is fundamentally reliant upon relaxation of the musculature.⁶⁰

The anterior drawer test is performed with the extended arm of the affected side cradled at 80–120° of abduction, 0–20° of forward flexion and 0–30° of external rotation by the examiner's hand in their axilla whilst manipulating the neck of the humerus. The examiner's other hand grips the scapula from coracoid to scapula spine in order to control anterior and posterior translations (Fig. 8). The posterior drawer is performed with the examiner standing to the test side of the supine patient, grasping the forearm and flexing the elbow to about 120°, whilst positioning the shoulder in 80–120° of abduction, and 20–30° of forward flexion. The other hand grasps the girdle with the thumb positioning on the lateral edge of the coracoid. In order to exacerbate a posterior translation, the humerus must be internally rotated slightly and flexed 60–80°; during this manoeuvre, the thumb subluxates the head of the humerus posteriorly.⁵⁸ Although the reliability of this test is unknown, Levy et al.⁶¹ noted that the interobserver reliability of the sulcus sign and laxity tests using Altchek's grading system⁵⁹ was 47% with a kappa value of less than 0.5.

Sulcus sign

The first reference to the sulcus sign was by Neer and Foster⁶² in assessing multidirectional instability (MDI; Fig. 9). If a depression is observed between the lateral edge of the acromion and the humeral head on gentle downward traction of the humerus, the sign is positive. Quantification of the sign through grading was added by Siliman and Hawkins.⁶³ Although Neer and Foster⁶² suggested that a positive sulcus may indicate inferior capsular redundancy, more recent selective sectioning experiments have demonstrated a considerable anterosuperior component to inferior laxity,^{21,64–67} a result of either superior glenohumeral ligament, coracohumeral ligament or rotator interval release.

All of these tests suffer from a difficulty in distinguishing relative bone motion deformations from overlying soft tissue deformations (Table 2).

Grading laxity tests

Determining a degree of joint laxity is fallible to both inter- and intra-examiner error. This disagreement may be in part due to both methodological and interpretation inconsistencies. Not only might a lack of objective measurement and human error play a part, but elements such as the amount of force used to assess the laxity, and interpretation of 'stiff' or 'soft' endpoints, or the point at which to interpret sufficient stability provided to joint laxity, may play a significant role. Elements of error may be reduced with experience, as well as adopting grading systems to present findings and add weight to management algorithms.

Grossly, both continuous and discrete subjective systems have been used to grade the degree of laxity present at the GHJ. Harryman and colleagues²¹ suggested 2 methods;

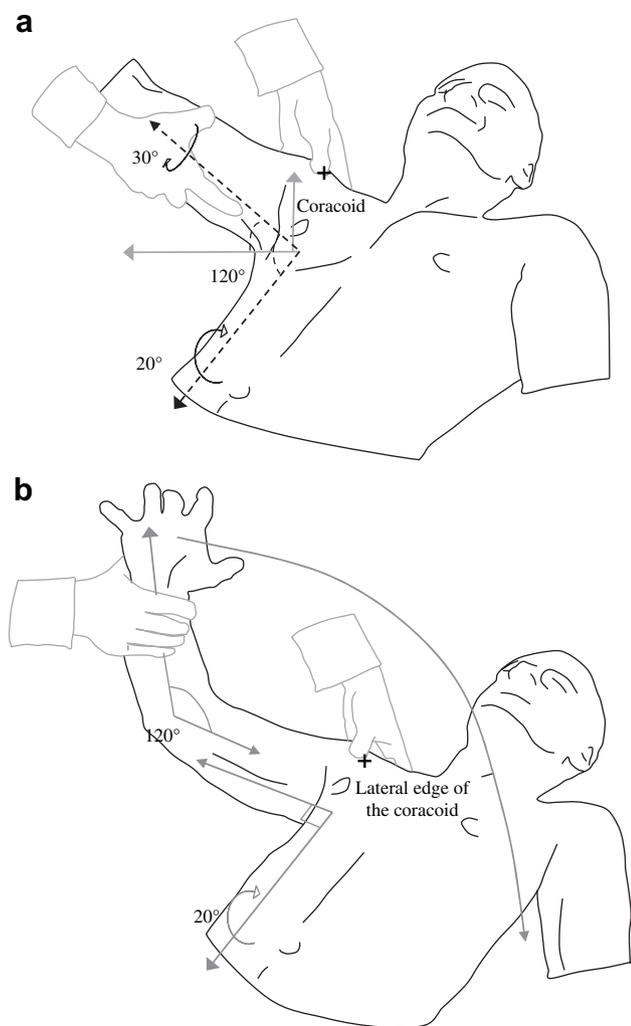


Figure 8 Anterior and posterior drawer test: (a) demonstrates the anterior drawer test, whilst (b) demonstrates the posterior drawer test; the cross body manoeuvre exacerbates posterior subluxation about the examines thumb.

measurement of the translation in millimetres, although this requires a tool to be accurate, or estimation of the percentage of humeral head translated across the glenoid, graded as I (25% head diameter), grade II (50% head diameter) or grade III (greater than 50% head diameter). The latter method, although theoretically plausible, is practically difficult as it relies upon an initial estimate of both humeral head and glenoid size. A further method was described by Hawkins and Bokor⁶⁸ in order to evaluate the positional relationship of the humeral head to the glenoid; type 0 is demonstrable by normal translation, type I by translation to the glenoid rim (i.e. equivalent to Harryman's grade II), type II over the glenoid rim (equivalent to Harryman's grade III), whilst type III describes a locked position. However, difficulty arises over differentiating between type 0 and I. Furthermore, type III rarely occurs, and therefore, the grading may not be very specific. Finally, a grading from 1+ to 3+ for joint laxity was proposed by Altchek et al.⁵⁹; a grade of 1+ indicates excessive translation compared to the contralateral side, but no subluxation, 2+ indicates subluxation of the humeral head over the glenoid rim, although

spontaneously reducing, and 3+ indicates frank dislocation with no spontaneous reduction. Finally, it is important to contextualise the findings of laxity grading scales by determining the generalised joint laxity within the patient.

Criteria for assessing generalised joint laxity were first described by Carter and Wilkinson,⁶⁹ modified by Beighton and Horan^{70,71}; the Beighton and Horan index is the most commonly employed instrument to date.⁷² However, excessive laxity can all too often be asymptomatic, and as such, management of those who suffer symptoms requires assessment of the functional impairment resulting from these.

Provocation tests

Provocation tests examine the ability of the shoulder to resist challenges to stability in positions where ligaments are normally under tension and, as such, exacerbate the symptoms of clinical instability; that is, pain, apprehension, loss of function and physical signs such as joint clicking or popping. A number of provocation examinations have been described, but commonly, the core manoeuvre performed is that of apprehension, relocation and release (Table 3). Although the primary intention of these clinical tests is to assess stability provided by the capsuloligamentous structures by stressing this mechanism, other key stability mechanisms are also loaded, and as such the tests are unable to distinguish between a static and dynamic aetiology to the symptom or sign representative of a positive test; that is, whether the pathology is of the capsulolabral tissue, rotator cuff, or mixed in nature.

Apprehension/augmentation test

The first step in what has now become a sequential manoeuvre is the apprehension test, initially alluded to by Rowe and Zarins⁷³ as a seated test in which an anteriorly directed force is applied in abduction external rotation (Fig. 10). Other authors have described similar tests such as the fulcrum test,⁷⁴ in which abduction external rotation is conducted supine with the examiner's hand between the head of the humerus and examination couch, acting as a fulcrum, the crank test^{75,76} in which a manual torque is applied at the wrist to a seated patient in an abducted externally rotated position, or Feagin's test.⁷⁷ However, the exam is better conducted without anterior force, as this is not often necessary to elucidate apprehension or pain, which may be relieved by the ensuing relocation test in the sequential manoeuvre. In recognition of this concept, Silliman and Hawkins⁷⁸ referred to this manoeuvre as the apprehension test if performed without the need to apply force to elicit a positive sign, and the augmentation test if force was used. The test can be conducted in either a supine or seated position. The examiner's first hand manoeuvres the patient's wrist on the affected side, and the second hand holds the affected shoulder girdle. With the arm in the adduction internal rotation position, the arm is brought into abduction external rotation, whilst using the second hand to palpate an anterior subluxation.⁷⁹ Pain or a feeling of apprehension without dislocation, have both been described as positive results.⁸⁰



Figure 9 Sulcus sign. The examiner's hand stabilises the scapula girdle whilst the other hand applies downward axial traction to the humerus; a sulcus between the lateral acromion and humeral head can be seen in global laxity.

Relocation test

Described by Jobe et al.,⁸¹ the relocation test involves applying a posteriorly directed force to relocate the humeral head on the glenoid; a positive test is determined by the diminution of apprehension during the applied force, recognised as 'Fowler's Sign'.⁶³ This test was validated by Speer et al.,⁸² though not at the extreme of external rotation as Jobe et al.⁸¹ first described, as in order to standardise the position, they tested all patients in 90° abduction and 90° of external rotation.⁸⁰ Their results when apprehension was used as the diagnostic criterion demonstrated a sensitivity of 68, a specificity of 100% for abnormal anterior translation, a PPV of 100%, a NPV of 78% and an accuracy of 85%, although this was reduced when apprehension was substituted with pain.

Release test

Silliman and Hawkins⁶³ described the release, or surprise test after a positive Fowlers sign in relocation as a diagnostic tool in its own right. The test is positive if a re-initiation of symptoms is seen with sudden removal of a relocating force. Gross and Distefano⁸³ validated the test in a series of 82 patients (sensitivity 92%, specificity 89%, PPV 87% and NPV 93%). During the apprehension–relocation–release routine, mean positive and negative predictive values of 93.6% and 71.9% were seen in subjects who had a feeling of apprehension in all 3 provocation tests. The release, or surprise test was the single most accurate test for abnormal anterior laxity (sensitivity of 63.89% and specificity of

Table 2 Laxity tests

Laxity	Test	Author	Patient position	Humeral head load	Humeral head rotation
<i>Common</i>					
A-P	Load and shift	Hawkins et al. ⁵⁵ ; <i>Protzman</i> ⁵⁶	Seated supine	A-P	Neutral
A-P	Drawer	Gerber and Ganz ⁵⁸	Supine	A or P	80–120° Abd 0–20° Flex 0–30° ER
MDI	Sulcus sign	Neer and Foster ⁶² ; <i>Silliman and Hawkins</i> ⁶³	Standing seated	I	Neutral
<i>Others</i>					
P	Posterior subluxation	Clarnette and Miniaci ⁵⁴	Supine	P	Add, IR, 70–90° Flex Then, Abd and ER to relocate
A	Anterior jerk	Leret et al. (1994)	Seated	S and then Jt line A	Abd, IR
P	Posterior jerk	Matsen et al. ⁹²	Seated	S	Abd, IR and then Flex. Ext to Relocate
P	Flexion–rotation pivot	Norwood and Terry ⁹³ ; <i>Miniaci et al.</i> ⁹⁴	Supine	P	90° Abd, 0° AxRot. Then, Flex and Add

Authors in italics are workers describing similar tests with the same objective, or authors adding to the development of the assessment. Caveat: these tests are not specific for particular pathologies, but are intended to provide evidence of laxity. Laxity is commonly determined as directional due to the association of a singular pathological lesion. However, in dissociating laxity from specific pathologies, laxity might move appropriately considered as global in character, rather than unidimensional, as an increased translation invariably results in or from modulation of the entire envelope of constraint. However, as is convention, the primary axis or direction of laxity is documented in this table. A, anterior; P, posterior; I, inferior; S, superior; MDI, multidirectional instability; Abd, abduction; Add, adduction; AxRot, axial rotation; ER, external rotation; IR, internal rotation; Flex, flexion; Ext, extension; Jt, joint; ICC, intraclass correlation coefficient (the extent to which a measure is repeatable representing non-systematic error).

Table 3 Provocation tests

Positive symptomatic provocation	Test	Author	Patient position	Humeral head load	Humeral head rotation	Sensitivity and specificity	PPV:NPV
							Mean for manoeuvre 94%:72%
<i>Core provocation manoeuvre</i>							
Jt pain, or apprehension w/out dislocation	Apprehension/augmentation	Rowe and Zarins ⁷³ ; <i>Boublik and Silliman</i> ⁷⁴ ; <i>Liu et al.</i> ^{75,76} ; <i>Rockwood</i> ⁷⁷	Seated <i>supine</i>	Augmentation, A, apprehension, none ⁷⁸	90° Abd, IR to ER		
Fowler's sign (diminuation of apprehension), Silliman and Hawkins ⁷⁸	Relocation	Jobe et al. ⁸¹	Supine	P to relocate apprehension manoeuvre	90° Abd, ER of apprehension manoeuvre	68%:100% At 90° Abd, 90° ER ⁸²	100%:78% At 90° Abd, 90° ER ⁸²
Re-initiation of Apprehension following +ve Fowler's sign	Release	Silliman and Hawkins ⁶³	Supine	None	90° Abd, ER of apprehension manoeuvre	92%:89% ⁸³ ; 64%:99% ⁸⁰	87%:93% ⁸³
<i>Others</i>							
Jt pain and apprehension	Posterior apprehension	O'Driscoll ⁹¹	Seated		90° Flex, IR		
Subacromial sulcus, or apprehension	Inferior apprehension (ABIS)	Feagin, Itoi et al. ⁹⁶	Standing	I	Abd (rested on examiners shoulder)	Unknown ⁹⁶	
Increasing apprehension or pain	Biceps load	Kim et al. ¹⁰⁷	Supine	Resisted elbow Flex	90° Abd, ER 120° Abd, ER	91%:97% for SLAP at 90° Abd, ER ¹⁰⁷ (n=75); 90%:97% for SLAP at 120° Abd, ER ¹⁰⁸ (n=38)	83%:98% at 90° Abd, ER ¹⁰⁷ (n=75); 92%:95% for SLAP at 120° Abd, ER ¹⁰⁸ (n=38)

Authors in italics are workers describing similar tests with the same objective, or authors adding to the development of the assessment. Caveat: these tests are not specific for particular pathologies, but are intended to provide evidence of laxity. Laxity is commonly determined as directional due to the association of a singular pathological lesion. However, in dissociating laxity from specific pathologies, laxity might move appropriately considered as global in character, rather than unidimensional, as an increased translation invariably results in or from modulation of the entire envelope of constraint. However, as is convention, the primary axis or direction of laxity is documented in this table. A, anterior; P, posterior; I, inferior; Abd, abduction; Add, adduction; AxRot, axial rotation; ER, external rotation; IR, internal rotation; Flex, flexion; Ext, extension; Jt, joint; ICC, intraclass correlation coefficient (the extent to which a measure is repeatable representing non-systematic error); PPV, positive predictive value; NPV, negative predictive value.

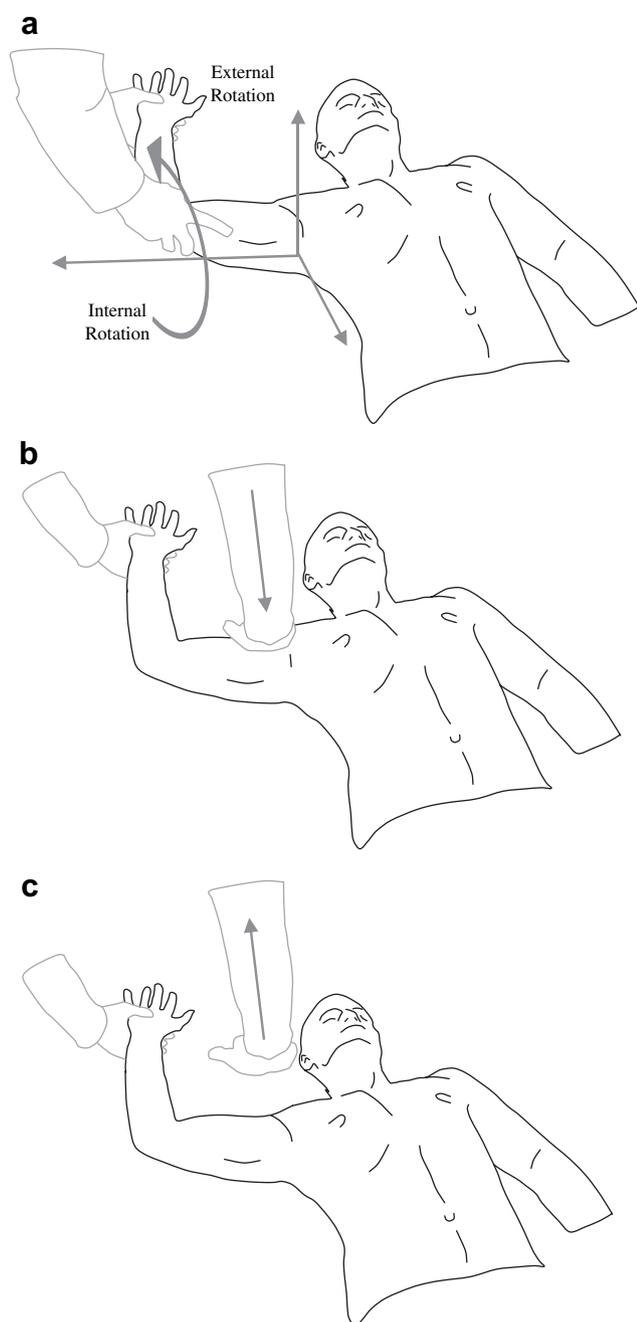


Figure 10 Apprehension–relocation–release manoeuvre: (a) exacerbates anterior translation of the humeral head, (b) relocates the humeral head diminishing any apprehension, and (c) produces a rebound apprehension.

98.91%), whilst an improvement in the feeling of apprehension or pain with relocation added little to the value of the tests. Despite this, the use of the 3-exam routine is highly specific and predictive of traumatic anterior glenohumeral instability.⁸⁰

Summary of provocation tests

The reliability of the common provocation manoeuvre is relatively undetermined. Throughout the provocation

manoeuvre, although more commonly the relocation and release routine, a number of authors have described pain as a positive diagnostic result^{84–86} in addition to apprehension. Further discrepancy arises in the interpretation of apprehension or augmentation test findings. One school of thought suggests diagnosis of instability as a result of overuse injury if pain is caused during this test.^{87–90}

Clinical tests for posterior instability

Posterior instability, although less common in isolation than anterior instability, often forms part of an MDI profile. A number of clinical tests have been described. These include a specific test for posterior laxity and a number of posterior provocation manoeuvres.

Posterior subluxation

Clarnette and Miniaci⁵⁴ developed the posterior subluxation test in which the patient is supine with the test shoulder over the edge of the couch. The test arm is adducted and internally rotated at 70–90° of flexion. The examiner takes the elbow and applies a posteriorly directed force causing the humeral head to fill the subacromial hollow in posterior instability. From this position, the humerus is abducted and externally rotated slowly to the point of relocation, palpable with the examining hand.

Posterior apprehension test

In diagnosing subacromial impingement, O'Driscoll⁹¹ observed that flexing the humerus to 90° and internally rotating elicited the symptoms of instability in patients with posterior instability.

Jerk test

In order to assess the posterior capsular integrity, the patient is seated with the arm in abduction internal rotation. The examiner grasps the elbow and axially loads the humerus in a proximal position. Whilst axial loading of the humerus is maintained, the arm is moved horizontally across the body. A positive test is indicated by a sudden jerk as the humeral head slides off the back of the glenoid. When the arm is returned to the original position of 90° abduction, a second jerk may be observed, that of the humeral head returning to the glenoid.⁹²

Flexion rotation pivot test

Norwood and Terry⁹³ described a further test for the presence of posterior instability. The patient's arm is positioned in 90° of abduction and neutral rotation with the elbow flexed 90° in a supine position, held with the examiner's first hand, whilst the other is placed on the anterior axillary fold. The examiner then forward flexes and adducts the patient's arm whilst exerting a posteriorly applied force on the humeral head, enabling a subtle subluxation and relocation to be palpated (Fig. 11).

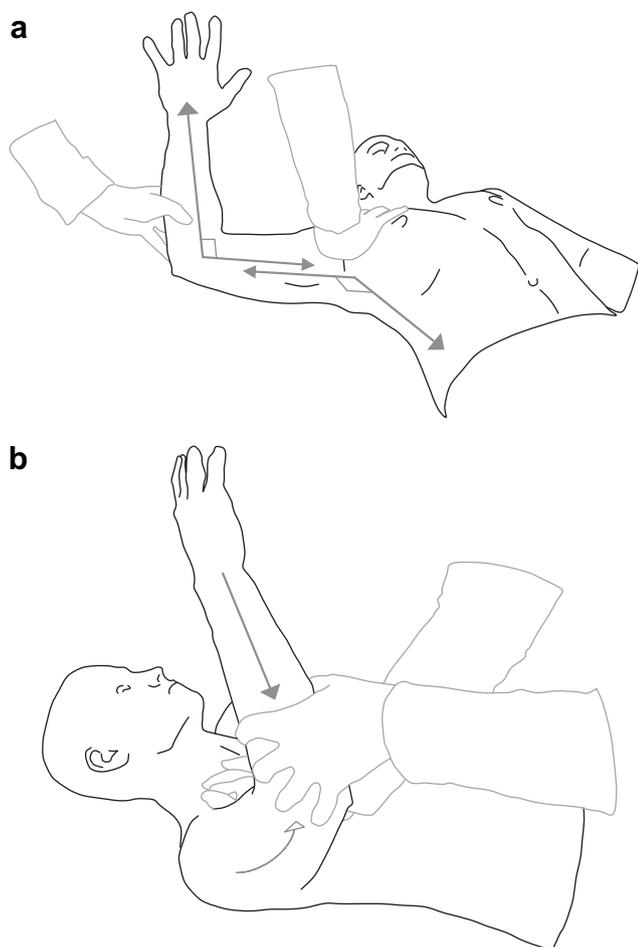


Figure 11 Flexion–rotation pivot test: (a) the elbow is flexed to 90° and the glenohumeral joint is abducted to 90°; (b) demonstrates coupled forward flexion and adduction from this position, whilst manually applying posteriorly directed force on the humeral head.

Miniaci et al.⁹⁴ described a similar test in which the palpating hand is placed on the anterior axillary fold stabilising acromion and clavicle to precipitate symptoms of posterior instability.

Other unidirectional tests

Inferior laxity often occurs as a result of both failure of the axillary pouch and the remaining capsule, and thus is a sign of MDI.

Hyperabduction test

Described by Gagey and Gagey,⁹⁵ the hyperabduction test is performed by stabilising the scapula whilst passively abducting the humerus; a constant value is achieved between bilateral paired shoulders in 95% of non-pathological shoulders examined ($n=100$), whilst lengthening and laxity of the inferior glenohumeral ligament is associated with more than 105° of abduction compared with 90° seen within the contralateral stable joint in 85%

of patients. Although this test has been supported by the presenting authors through cadaveric measurement, no review of the validity of this test has been presented to date.

Inferior apprehension, or abduction inferior stability (ABIS) test

Initially described by Feagin, and further refined by Ito and colleagues,⁹⁶ this examination is performed with the patient's affected upper limb held in abduction, and the patient's forearm resting on the examiner's shoulder. The examiner exerts bimanual downward force over the neck of the humerus. If the shoulder is unstable, the head will be translated inferiorly, and a subacromial groove will appear. A positive clinical sign may also be apprehension. There is no validation of this manoeuvre to date.

Clinical tests for labral lesions

In order to determine the extent of laxity resulting from labral failure, a number of clinical tests have been described (Table 4).

Active compression test

O'Brien⁹⁷ used the active compression test to determine superior labral tears with a sensitivity of 100%, specificity of 97%, PPV of 89% and NPV of 100%. The examiner performs the test by standing behind the patient applying a downward force on their 90° flexed arm in 10° of adduction and full internal rotation with the elbow in full extension. If pain is elicited 'inside' the shoulder, and eliminated if externally rotated, a positive result is recorded. Stetson and Templin⁹⁸ found a sensitivity of 54% and a specificity of 31% using this test, with a PPV of 34% and NPV of 50%, similar to the findings of McFarland et al.⁹⁹

Crank test

Performed with the patient seated and the arm elevated to about 160° in the scapula plane, the examiner applies a joint load along the axis of the humerus whilst axially rotating the humerus.⁷⁵ A positive result is elicited by either pain during the manoeuvre with or without a click, or reproduction of the characteristic symptoms⁷⁶ (sensitivity 91%, specificity 93%, PPV 94% and NPV 90%). Contrary to these results, Stetson and Templin⁹⁸ demonstrated a PPV of 41%, specificity of 56%, sensitivity of 46% and an NPV of 61%, concluding that the crank test was not sensitive enough for detecting glenoid labral tears. The crank test may be more sensitive in a select group of younger patients prone to labral lesions. A further study conducted by Mimori et al.¹⁰⁰ used both MR arthrography and arthroscopy to verify the pain ratings from the crank test in 32 patients with throwing injuries of the shoulder. Detachment of the labrum was detected by the crank test with a sensitivity of 83%, a specificity of 100% and an accuracy of 87%.

Table 4 Labral lesion tests

Labral lesion	Test	Author	Patient position	Humeral head load	Humeral head rotation	Sensitivity and specificity	PPV:NPV
General labral lesion	Active compression	O'Brien et al. ⁹⁷	Seated	I	90° Flex, 10° Add, full IR	100%:97% ⁹⁷ ; 54%:31% ⁹⁸ ; 47% sensitivity for SLAP ⁹⁹ (n=426)	89%:100% ⁹⁷ ; 34%:50% ⁹⁸
General labral lesion	Crank	Liu et al. ^{75,76}	Seated	Ax Comp	160° Scap, then AxRot	91%:93% ^{75,76} ; 46%:56% ⁹⁸ ; 83%:100% ¹⁰⁰ .	94%:90% ^{75,76} ; 41%:61% ⁹⁸ .
SLAP SLAP	Biceps tension Compression—rotation	Snyder et al. ¹⁰¹ Snyder et al. ¹⁰¹	Standing Supine	Resisted Flex Ax Comp	Flex 90° Abd, then circumduct and rotate		
General labral lesion AS general labral lesion	Clunk Anterior slide	Kibler ¹⁰⁵	Standing seated Seated Standing	Ax Comp AS directed axial	Ext to Flex Hands on Hips, thumbs directed P	78%:92% ¹⁰⁵ ; 84% specificity ⁹⁹ (n=426)	
Biceps tendonitis Biceps tendonitis	Speed's Yergason's	Yergason ¹⁰⁶	Standing Standing	Resisted Flex Resisted supination	Flex Neutral, 30° ER, Elbow 90° Flex		

Caveat: these tests are not specific for particular pathologies, but are intended to provide evidence of laxity. Laxity is commonly determined as directional due to the association of a singular pathological lesion. However, in dissociating laxity from specific pathologies, laxity might more appropriately be considered as global in character, rather than unidimensional, as an increased translation invariably results in or from modulation of the entire envelope of constraint. However, as is convention, the primary axis or direction of laxity is documented in this table. A, anterior; P, posterior; I, inferior; S, superior; MDI, multidirectional instability; Ax Comp, axial compression; Abd, abduction; Add, adduction; AxRot, axial rotation; ER, external rotation; IR, internal rotation; Flex, flexion; Ext, extension; Scap, scaption; Jt, joint; ICC, intraclass correlation coefficient (the extent to which a measure is repeatable representing non-systematic error).

Biceps tension and compression—rotation tests

The Biceps tension test and compression—rotation test were first described by Snyder et al.¹⁰¹ as a means of eliciting positive signs of a SLAP lesion; that is, a superior labral detachment extending from anterior to posterior, and thus incorporating the biceps anchor. The biceps tension test examines resisted shoulder flexion with the elbow extended and the forearm supinated, akin to the active compression test for labral lesions. Field and Savoie¹⁰² noted that this test was positive in 20 consecutive patients with a diagnosis of a SLAP.

The compression—rotation test is performed with the patient supine, the shoulder abducted 90° and the elbow flexed 90°. A compression force is applied to the humerus axially which is then circumducted and rotated in an attempt to trap the torn labrum. A catch and snap, akin to McMurray's test¹⁰³ of lesions of the posterior meniscal horn of the knee may be felt at the joint line. However, no validation of this test has been conducted.

Speed's and the clunk test

Similar tests to the biceps tension and compression—rotation tests are the clunk test and Speed's test: Speed's test is performed by flexing the shoulder against resistance whilst maintaining the elbow in extension and the forearm in supination. Pain is a positive indicator of biceps tendonitis.

During the Clunk test, the arm is rotated and loaded from a position of extension to one of forward flexion. A clunk like sensation is felt if a free labral fragment is caught in the joint, again similar to McMurray's test¹⁰³ of the meniscus in the knee. However, it has been reported that a click is a common occurrence in patients with labral tears in the absence of joint instability.¹⁰⁴

Anterior slide test

The anterior slide test, first described by Kibler¹⁰⁵ requires the patient to be either seated or standing, with their hands on their hips and their thumbs pointing posteriorly. With one of the examiner's hands across the shoulder girdle from posterior to the anterior aspect of the acromion, and the other hand applying an anterosuperior force at the elbow with the patient resisting this motion, pain elicited at the front of the GHJ under the examiners index finger, and/or a pop or click in the same area is considered positive. A positive result is also recorded if it recreates the symptoms that occur during overhead activity¹⁰⁵ (sensitivity of 78% and a specificity of 92% measured by the describing author).

Other examinations of passive stability

Other clinical exams determine the integrity of the biceps tendon. Although not distinct from the bicipital anchor, and thus the superior labrum, the exams were conceived to establish bicipital wear or incompetence, and thus are presented here.

Yergason's test

Yergason's test is performed with the elbow flexed to 90° and the forearm pronated.¹⁰⁶ At this point, the examiner holds the wrist to resist active supination by the patient. Pain in the bicipital groove is indicative of tendonitis or wear of the biceps tendon.

Biceps load test

The biceps load test was first described by Kim et al.¹⁰⁷ In assessing recurrent anterior instability, an apprehension test in the supine position with the forearm supinated has been advocated. Once an apprehensive position is reached, the patient should be asked to flex the elbow against the force of the examiner; if apprehension is not reduced, or indeed, if it gets worse, the test is thought to be positive for a superior lesion from anterior to posterior (SLAP; Fig. 12). In 75 patients, Kim et al.¹⁰⁷ reported a sensitivity of 91% and a specificity of 97%. The PPV was 83% and NPV 98%. However, if only one traumatic episode has been documented, the test should be conducted in 120° of abduction. If pain, or more pain is noted during the resisted elbow flexion, the test is positive. Kim et al.¹⁰⁸ evaluated 127 shoulders following a primary traumatic event arthroscopically, of which 38 were positive tests (the sensitivity was 90%, specificity 97%, PPV 92% and NPV 95%).

Reliability of clinical tests

McFarland et al.⁹⁹ variably performed the active compression, anterior slide and compression rotation tests on 426 patients prior to arthroscopy. They demonstrated that the incidence of positive results were not statistically different between those with and without SLAP lesions, whilst no patient with a SLAP was positive for all 3 tests, as might be expected if infallible. The active compression test was the most sensitive to SLAP lesions (47%), yet had lowest overall accuracy (54%), whilst the most specific test and the most accurate was the anterior slide test (specificity of 84% and accuracy of 77%). Furthermore, McFarland and colleagues⁹⁹ noted that the accuracy of these tests may be confounded by co-existing injury or pathology, as both the presence of a click, or the location of pain are not reliable diagnostic indicators.

Tzannes et al.⁵⁷ demonstrated in a cohort of 13 patients with a history suggestive of instability, that correlation between 4 examiners is reliable in the load-and-shift, sulcus and provocation tests when care is taken with respect to arm position, and apprehension is used as a positive test, rather than pain. Levy et al.⁶¹ demonstrated similar results for intraobserver and interobserver reproducibility (46% and 47%, respectively) for the anterior and posterior drawer tests in 43 asymptomatic collegiate athletes. The degree of laxity was graded according to Altchek et al.,⁵⁹ and as such, a subjective degree of laxity was determined rather than the reproduction of patient symptoms, possibly confounding the reproducibility of these results.

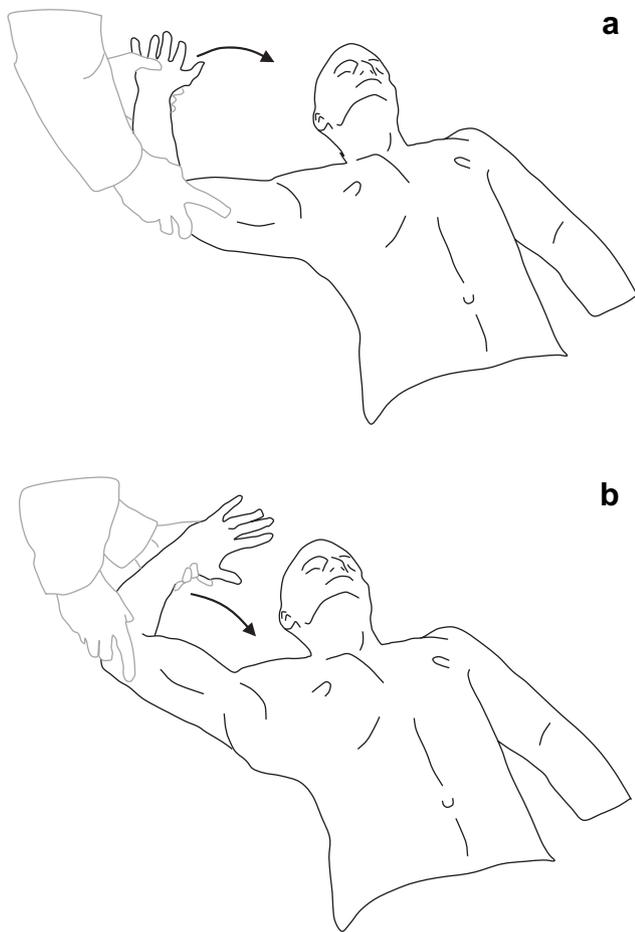


Figure 12 Biceps load test and biceps load test II. In (a), manual resistance is applied against elbow flexion at 90° of abduction, whilst (b) depicts a similar active movement at 120° of abduction.

Summary of shoulder assessment and testing algorithm

Assessment of the integrity of the passive stabilising system of the GHJ has been described in a number of ways. This inconsistency is compounded by the difficulty in eliciting subtle signs in order to determine a positive test result. As such, an evidence-based approach to examination that is practical, and can be implemented successfully by all involved in the rehabilitation process, might aid in the assessment of instability; this might appropriately take the form of an algorithm.

Intuitively, an algorithm that can appropriately be applied clinically might reflect the simplest group of tests to achieve the diagnosis, rather than all of the tests that one may apply. Typically, a generic shoulder assessment might form the foundation of such an assessment, in which range of motion is assessed in a standing position, in an attempt to elicit apprehension (and the degree of elevation at which it occurs), pain, abnormal muscle coordination and scapula dyskinesia. Following this, a gross assessment of comparative rotator cuff strength is important, as is use of the Beighton Score. Next, an assessment of the current position of the humerus relative to the scapula is important; if the

joint is dislocated, is the humeral head reducible passively, or does it require active manipulation. Guidance at this step is critically dependent upon a detailed history taken prior to examination, as if the joint is located, a history descriptive of instability would assume the need to determine the envelope of laxity. Assessment of anteroposterior stability is best performed with the load-and-shift test, followed by the core provocation manoeuvre (apprehension, relocation and release sequence manoeuvre); load-and-shift might appropriately be performed at 0° of abduction for suspected posterior instability, and 90° of abduction for anterior instability. Provocation in a tested direction is positive on eliciting pain or apprehension. Following this, assessment of the superior laxity and the long head of biceps anchor is most appropriately performed primarily by O'Brien's active compression test,⁹⁷ followed by Speed's and Yergason's test.¹⁰⁶ This might be supplemented by the biceps load test if the clinical picture remains equivocal.¹⁰⁷ Finally, inferior instability is evident upon demonstration of a positive Sulcus sign,^{62,63} and the hyperabduction test.⁹⁵

Conclusion

The shoulder complex affords a large range of motion to the upper limb in order to position the hand to perform its function. This highly mobile state is maintained by a number of physiological stability mechanisms. Due to loading conditions upon the shoulder and alterations in the structural quality of the stability mechanisms, disruption to stability may occur. This is examined and then classified to derive a chosen course of management, aimed at optimising acute, recovery and functional phases of restoration. A number of tests have been described in order to diagnose excessive laxity or instability, albeit, many of the physical examinations are reliant upon similar mechanisms to elicit positive clinical signs. However, many of the tests have little supporting anatomical study, nor has an extensive independent analysis been conducted. From the statistics available, it may be generalised that no one test is diagnostic for any one traumatic or pathologic entity, thus questioning the weight given to such tests in the clinical diagnosis and subsequent management of traumatic shoulder instabilities. However, it is important to recognise the synergistic value of a thorough history to clinical examination of the joint of interest in order to arrive at a differential diagnosis; this may then be clarified by imaging examination of the joint, within which the gold standard is MR arthrography.

Acknowledgements

The authors would like to thank the assistance of the Engineering and Physical Sciences Research Council Doctorate Training Studentship and the Arthritis Research Campaign.

References

1. Kinzel GL, Gutkowsky LJ. Joint models, degrees of freedom, and anatomical motion measurement. *J Biomech Eng* 1983; 105(1):55–62.

2. Pascoal AG, van der Helm FF, Pezarat Correia P, Carita I. Effects of different arm external loads on the scapulo-humeral rhythm. *Clin Biomech* 2000;**15**(Suppl 1):S21–4.
3. Johnston TB. The movements of the shoulder joint. A plea for the use of the 'Plane of the Scapula' as the plane of reference for movements occurring at the humero-scapular joint. *J Bone Joint Surg* 1937;**25**:252–60.
4. Saha AK. Mechanism of shoulder movements and a plea for the recognition of "Zero Position" of glenohumeral joint. *Indian J Surg* 1950;**12**:153–65.
5. Codman EA. *The shoulder: rupture of the supraspinatus tendon and other lesions in or about the subacromial Bursa*. Boston: Privately Printed; 1934.
6. Dempster WT. Mechanisms of shoulder movement. *Arch Phys Med Rehab* 1965;**46**(1-A):49–69.
7. Dvir Z, Berme N. The shoulder complex in elevation of the arm: a mechanism approach. *J Biomech* 1978;**11**(5):219–25.
8. Van der Helm FCT, Pronk GM. Three-dimensional recording and descriptions of motions of the shoulder mechanism. *J Biomech Eng* 1995;**117**:27–38.
9. Inman VT, Saunders JB, DeC M, Abbott LC. Observations on the function of the shoulder joint. *J Bone Joint Surg* 1944;**26**:1–30.
10. Bagg SD, Forrest WJ. A biomechanical analysis of scapular rotation during arm abduction in the scapular plane. *Am J Phys Med Rehab* 1988;**67**(6):238–45.
11. Saha AK. *Theory of the shoulder mechanism: descriptive and applied*. Springfield, IL: Charles C. Thomas; 1961.
12. Kibler WB. Closed Kinetic chain rehabilitation for sports injuries. *Phys Med Rehab Clin N Am* 2000;**11**(2):369–84.
13. Poppen NK, Walker PS. Normal and abnormal motion of the shoulder. *J Bone Joint Surg Am* 1976;**58-A**:195–201.
14. Chen SK, Simonian PT, Wickiewicz TL, Otis JC, Warren RF. Radiographic evaluation of glenohumeral kinematics: a muscle fatigue model. *J Shoulder Elbow Surg* 1999;**8**:49–52.
15. Ludewig PM, Cook TM. Translations of the humerus in persons with shoulder impingement symptoms. *J Orthop Sports Phys Ther* 2002;**32**(6):248–59.
16. Kelkar R, Flatow EL, Bigliani LU, Soslowsky LJ, Atewshian GA, Pawluk RJ, et al. A stereophotogrammetric method to determine the kinematics of the glenohumeral joint. *Adv Bioeng ASME* 1992;**19**:143–6.
17. Thompson WO, Debski RE, Boardman III ND, Taskiran E, Warner JJ, Fu FH, et al. A biomechanical analysis of rotator cuff deficiency in a cadaveric model. *Am J Sports Med* 1996;**24**:286–92.
18. Graichen H, Stammberger T, Bonel H, et al. Magnetic resonance based motion analysis of the shoulder during elevation. *Clin Orthop Relat Res* 2000;**370**:154–63.
19. McMahon PJ, Debski RE, Thompson WO, Warner JJ, Fu FH, Woo SL. Shoulder muscle forces and tendon excursions during glenohumeral abduction in the scapular plane. *J Shoulder Elbow Surg* 1995;**4**(3):199–208.
20. Harryman II DT, Sidles JA, Clark JM, McQuade KJ, Gibb TD, Matsen III FA. Translation of the humeral head on the glenoid with passive glenohumeral motion. *J Bone Joint Surg* 1990;**72-A**:1334–43.
21. Harryman DT, Sidles JA, Harris SL, Matsen III FA. Laxity of the normal glenohumeral joint: a quantitative *in-vivo* assessment. *J Shoulder Elbow Surg* 1992;**1**:113–8.
22. Tillander B, Norlin R. Intraoperative measurement of shoulder translation. *J Shoulder Elbow Surg* 2001;**10**(4):358–64.
23. Jorgensen U, Bak K. Shoulder instability. Assessment of anterior-posterior translation with a knee laxity tester. *Acta Orthop Scand* 1995;**66**(5):398–400.
24. Hawkins RJ, Neer 2nd CS, Pianta RM, Mendoza FX. Locked posterior dislocation of the shoulder. *J Bone Joint Surg Am* 1987;**69**(1):9–18.
25. McClure PW, Michener LA, Sennett BJ, Karduna AR. Direct 3-dimensional measurement of scapular kinematics during dynamic movements *in vivo*. *J Shoulder Elbow Surg* 2001;**10**:269–77.
26. Michener LA, McClure PW, Karuna AR. Anatomical and biomechanical mechanisms of subacromial impingement syndrome. *Clin Biomech* 2003;**18**:369–79.
27. Ludewig PM, Cook TM. Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. *Phys Ther* 2000;**80**:273–6.
28. Meskers CG, Vermeulen HM, de Groot JH, Van der Helm FCT, Rozing PM. 3D shoulder position measurement using a six-degree-of-freedom electromagnetic tracking device. *Clin Biomech* 1998;**13**:280–92.
29. Price CI, Franklin P, Rodgers H, Curless RH, Johnson GR. Active and passive scapulohumeral movement in healthy persons: a comparison. *Arch Phys Med Rehab* 2000;**81**:28–31.
30. Karduna AR, McClure PW, Michener LA, Sennett B. Dynamic measurements of three-dimensional scapular kinematics: a validation study. *J Biomech Eng* 2001;**123**:184–90.
31. de Groot JH, van Woensel W, Van der Helm FCT. Effect of different arm loads on the position of the scapula in abduction postures. *Clin Biomech* 1999;**14**:309–14.
32. de Groot JH. The variability of shoulder motions recorded by means of palpation. *Clin Biomech* 1997;**12**(7/8):461–72.
33. Ludewig PM, Cook TM, Nawoczenski DA. Three-dimensional scapular orientation and muscle activity at selected positions of humeral elevation. *J Orthop Sports Phys Ther* 1996;**24**(2):57–65.
34. Borstad JD, Ludewig PM. Comparison of scapula kinematics between elevation and lowering of the arm in the scapular plane. *Clin Biomech* 2002;**17**:650–9.
35. Bearn JG. Direct observations on the function of the capsule of the sternoclavicular joint in clavicular support. *J Anat* 1967;**101**:159–70.
36. Cave AJE. The nature and morphology of the costoclavicular ligament. *J Anat* 1961;**95**:170–9.
37. Acus RW, Bell RH, Fisher DL. Proximal clavicle excision: an analysis of results. *J Shoulder Elbow Surg* 1995;**4**:182–7.
38. Matsen FA, Harryman DT, Sidles JA. Mechanics of glenohumeral instability. *Clin Sports Med* 1991;**10**(4):783–8.
39. Croft P, Pope D, Silman A. Primary care rheumatology society shoulder study group. The clinical course of shoulder pain. *Br Med J* 1996;**313**:601–2.
40. Van der Windt DA, Koes BW, de Jong BA. Shoulder disorders in general practice. *Ann Rheum Dis* 1995;**54**:959–64.
41. McFarland EG, Torpey BM, Curl LA. Evaluation of shoulder laxity. *Sports Med* 1996;**22**(4):264–72.
42. Rowe CR. Prognosis in dislocations of the shoulder. *J Bone Joint Surg* 1956;**38A**:957–77.
43. Scambler G. *Sociology as applied to medicine*. 4th ed. W.B. Saunders; 1997.
44. Schwartz E, Warren RF, O'Brien SJ, Fronek J. Posterior shoulder instability. *Orthop Clin North Am* 1987;**18**(3):409–19.
45. Lippitt SB, Matsen III FA. Mechanisms of glenohumeral joint stability. *Clin Orthop* 1993;**291**:20–8.
46. Gibb TD, Sidles JA, Harryman 2nd DT, McQuade KJ, Matsen 3rd FA. The effect of capsular venting on glenohumeral laxity. *Clin Orthop Relat Res* 1991;**(268)**:120–7.
47. Kumar VP, Balasubramaniam P. The role of atmospheric pressure in stabilising the shoulder. An experimental study. *J Bone Joint Surg Br* 1985;**67**(5):719–21.
48. Warner JJP, Deng X, Warren RF, Torzilli PA, O'Brien SJ. Superior-inferior translation in the intact and vented glenohumeral joint. *J Shoulder Elbow Surg* 1993;**2**:99–105.
49. Rockwood CA. Subluxation of the shoulder: the classification, diagnosis and treatment. *Orthop Trans* 1979;**4**:306.

50. Thomas SC, Matsen FA. An approach to the repair of avulsion of the glenohumeral ligaments in the management of traumatic anterior glenohumeral stability. *J Bone Joint Surg* 1989;71-A:506–13.
51. Schneeberger AG, Gerber C. Classification and therapy of the unstable shoulder. *Ther Umsch* 1998;553:187–91.
52. Gerber C, Nyffeler RW. Classification of glenohumeral joint instability. *Clin Orthop* 2002;400:65–76.
53. Lewis A, Kitamura T, Bayley JIL. The classification of shoulder instability: new light through old windows!. *Curr Orthop* 2004;18:97–108.
54. Clarnette RG, Miniaci A. Clinical exam of the shoulder. *Med Sci Sports Exerc* 1998;30(4 (Suppl 1)):S1–6.
55. Hawkins RJ, Schutte JP, Huckell GH. The assessment of glenohumeral translation using manual and fluoroscopic techniques. *Orthop Trans* 1988;12:727–8.
56. Protzman RR. Anterior instability of the shoulder. *J Bone Joint Surg Am* 1980;62(6):909–18.
57. Tzannes A, Paxinos A, Callanan M, Murrell GAC. An assessment of the interexaminer reliability of tests for shoulder instability. *J Shoulder Elbow Surg* 2004;13:18–23.
58. Gerber C, Ganz R. Clinical assessment of instability of the shoulder. With special reference to anterior and posterior drawer tests. *J Bone Joint Surg Br* 1984;66(4):551–6.
59. Altchek DA, Warren RF, Ortiz G, et al. T-Plasty: a technique for treating multidirectional instability in an athlete. *J Shoulder Elbow Surg* 1991;73-A:105–12.
60. Tennent TD, Beach WR, Meyers JF. A review of the special tests associated with shoulder examination. *Am J Sports Med* 2003;31(2):301–7.
61. Levy AS, Linter S, Kenter K, Speer KP. Intra- and interobserver reproducibility of the shoulder laxity examination. *Am J Sports Med* 1999;27(4):460–3.
62. Neer CS, Foster CR. Inferior capsular shift for involuntary inferior and multidirectional instability of the shoulder. *J Shoulder Elbow Surg* 1980;62-A:897–908.
63. Silliman JF, Hawkins RJ. Classification and physical diagnosis of instability of the shoulder. *Clin Orthop Relat Res* 1993;(291):7–19.
64. Bowen MK, Deng XH, Warren PA, Torzilli PA, Altchek DW, O'Brien SJ. Role of the inferior glenohumeral ligament complex in limiting inferior translation of the glenohumeral joint. *Trans Orthop Res Soc Washington DC* 1992;497.
65. Moorman CT, Deng XH, Warren, et al. *The coracoacromial ligament: is it the appendix of the shoulder? Speciality Day*. Orlando, FL: American SES, AOS; 1995.
66. Lee TQ, Black AD, Tibone JE, McMahon PJ. Release of the coracoacromial ligament can lead to glenohumeral laxity: a biomechanical study. *J Shoulder Elbow Surg* 2001;10(1):68–72.
67. Ovesen J, Nielsen S. Experimental distal subluxation in the glenohumeral joint. *Arch Orthop Trauma Surg* 1985;104(2):78–81.
68. Hawkins RJ, Bokor DJ. Clinical evaluation of shoulder problems. In: Rockwood CA, Matsen FA, editors. *The shoulder*. Philadelphia: WB Saunders; 1990. p. 149–77.
69. Carter CO, Wilkinson JA. Persistent joint laxity and congenital dislocation of the hip. *J Bone Joint Surg Br* 1964;46:40–5.
70. Beighton P, Horan F. Orthopaedic aspects of the Ehlers–Danlos syndrome. *J Bone Joint Surg Br* 1969;51:444–53.
71. Beighton P, Solomon L, Soskolne CL. Articular mobility in an African population. *Ann Rheum Dis* 1973;32:413–8.
72. Boyle KL, Witt P, Riegger-Krugh C. Intrarater and interrater reliability of the Beighton and Horan joint mobility index. *J Athlet Train* 2003;38(4):281–5.
73. Rowe CR, Zarins B. Recurrent transient subluxation of the shoulder. *J Bone Joint Surg Am* 1981;63(6):863–72.
74. Boublik M, Silliman J. History and physical examination. In: Hawkins R, Misamore G, editors. *Shoulder injuries in the athlete*. New York: Churchill Livingstone; 1996.
75. Liu SH, Henry MH, Nuccion SL. A prospective evaluation of a new physical examination in predicting glenoid labral tears. *Am J Sports Med* 1996;24:721–5.
76. Liu SH, Henry MH, Nuccion S, Shapiro MS, Dorey F. Diagnosis of glenoid labral tears. A comparison between magnetic resonance imaging and clinical examinations. *Am J Sports Med* 1996;24:149–54.
77. Rockwood CA. Subluxations and dislocations about the shoulder. In: Green DP, Rockwood CA, editors. *Fractures in adults*. Philadelphia: JP Lippincott; 1984.
78. Silliman JF, Hawkins RJ. Current concepts and recent advances in the athlete's shoulder. *Clin Sports Med* 1991;10(4):693–705.
79. Leffert RD, Gumley G. The relationship between dead arm syndrome and thoracic outlet syndrome. *Clin Orthop* 1987;225:20–31.
80. Lo IK, Nonweiler B, Woolfrey M, Litchfield R, Kirkley A. An evaluation of the apprehension relocation, and surprise tests for anterior shoulder instability. *Am J Sports Med* 2004;32(2):301–7.
81. Jobe FW, Kvitne RS, Giangarra CE. Shoulder pain in the overhand or throwing athlete. The relationship of anterior instability and rotator cuff impingement. *Orthop Rev* 1989;18(9):963–75.
82. Speer KP, Hannafin JA, Altchek DW, Warren RF. An evaluation of the shoulder relocation test. *Am J Sports Med* 1994;22(2):177–83.
83. Gross ML, Distefano MC. Anterior release test. A new test for occult shoulder instability. *Clin Orthop Relat Res* 1997;(339):105–8.
84. Jobe CM. Superior glenoid impingement. Current concepts. *Clin Orthop Relat Res* 1996;(330):98–107.
85. Boublik M, Hawkins RJ. Clinical examination of the shoulder complex. *J Orthop Sports Phys Ther* 1993;18(1):379–85.
86. Wilk KE, Arrigo CA, Andrews JR. Current concepts: the stabilising structures of the glenohumeral joint. *J Sports Physiother* 1997;25(6):364–79.
87. Carallo RJ, Speer KP. Shoulder instability and impingement in throwing athletes. *Med Sci Sports Exerc* 1998;30(4 Suppl):S18–25.
88. Kvitne RS, Jobe FW. The diagnosis and treatment of anterior instability in the throwing athlete. *Clin Orthop Relat Res* 1993;(291):107–23.
89. Miniaci A, Fowler PJ. Impingement in the athlete. *Clin Sports Med* 1993;12(1):91–110.
90. Glousman RE. Instability versus impingement syndrome in the throwing athlete. *Orthop Clin North Am* 1993;24(1):89–99.
91. O'Driscoll SW. A reliable and simple test for posterior instability of the shoulder. *J Bone Joint Surg* 1991;73B(Suppl 1):50.
92. Matsen III FA, Thomas SC, Rockwood Jr CA. Glenohumeral instability. In: Rockwood Jr CA, Matsen III FA, editors. *The shoulder* 1990;vol. 1. p. 550.
93. Norwood LA, Terry GC. Shoulder posterior subluxation. *Am J Sports Med* 1984;12:25–30.
94. Miniaci A, Dowdy PA, Fowler PJ. Clinical assessment of shoulder injuries. In: Chan KM, editor. *Sports injuries of the hand and upper limb*. New York: Churchill Livingstone; 1995.
95. Gagey OJ, Gagey N. The hyperabduction test. *Journal of Bone and Joint Surgery* 2001;83(1-Br):69–74.
96. Itoi E, et al. Scapular inclination and inferior stability of the shoulder. *J Shoulder Elbow Surg* 1992;1:131.
97. O'Brien SJ, Pagnani MJ, Fealy S, McGlynn SR, Wilson JB. The active compression test: a new and effective test for diagnosing labral tears and acromioclavicular joint abnormality. *Am J Sports Med* 1998;26:610–3.

98. Stetson WB, Templin K. The crank test, the O'Brien test, and routine magnetic resonance imaging scans in the diagnosis of labral tears. *Am J Sports Med* 2002;**30**(6):806–9.
99. McFarland EG, Kim TK, Savino RM. Clinical assessment of three common tests for superior labral anterior–posterior lesions. *Am J Sports Med* 2002;**30**(6):810–5.
100. Mimori K, Muneta T, Nakagawa T, Shinomiya K. A new pain provocation test for superior labral tears of the shoulder. *Am J Sports Med* 1999;**27**(2):137–42.
101. Snyder SJ, Karzel RP, Del Pizzo W, et al. SLAP lesions of the shoulder. *Arthroscopy* 1990;**6**:274–9.
102. Field LD, Savoie III FH. Arthroscopic suture repair of superior labral detachment lesions of the shoulder. *Am J Sports Med* 1993;**21**(6):783–90.
103. McMurray TP. The semilunar cartilages. *Br J Surg* 1941;**29**:407.
104. Glasgow S, Bruce RA, Yacobucci GN, Torg JS. Arthroscopic resection of glenoid labral tears in the athlete. *Arthroscopy* 1992;**8**:48–54.
105. Kibler WB. Specificity and sensitivity of the anterior slide test in throwing athletes with superior glenoid labral tears. *Arthroscopy* 1995;**11**:296–300.
106. Yergason RM. Supination sign. *J Bone Joint Surg Boston* 1931;**13**:160.
107. Kim SH, Ha KI, Ahn JH, et al. Biceps load test: a clinical test for superior labrum anterior and posterior lesions in shoulders with recurrent anterior dislocations. *Am J Sports Med* 1999;**27**:300–3.
108. Kim SH, Ha KI, Ahn JH, et al. Biceps load test II: a clinical test for SLAP lesions of the shoulder. *Arthroscopy* 2001;**17**:160–4.