TUTORIAL

Diagnosis and Management of the Painful Shoulder. Part 2: Examination, Interpretation, and Management

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■ **Abstract:** Diagnosis, interpretation and subsequent management of shoulder pathology can be challenging to clinicians. Because of its proximal location in the schlerotome and the extensive convergence of afferent signals from this region to the dorsal horn of the spinal cord, pain reference patterns can be broadly distributed to the deltoid, trapezius, and or the posterior scapular regions. This pain behavior can make diagnosis difficult in the shoulder region, as the location of symptoms may or may not correspond to the proximity of the pain generator. Therefore, a thorough history and reliable physical examination should rest at the center of the diagnostic process. Effective management of the painful shoulder is closely linked to a tissue-specific clinical examination. Painful shoulder conditions can present with or without limitations in passive and or active motion. Limits in passive motion can be classified as either capsular or noncapsular patterns. Conversely, patients can present with shoulder pain that demonstrates no limitation of motion. Bursitis, tendopathy and rotator cuff tears can produce shoulder pain that is challenging to diagnose, especially when they are the consequence of impingement and or instability. Numerous nonsurgical measures can be implemented in treating the painful shoulder, reserving surgical interventions for those patients who are resistant to conservative care. ■

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INTRODUCTION

Diagnosis, interpretation and subsequent management of shoulder pathology can be challenging to clinicians. The term "shoulder" refers to a complex of joints and soft tissue structures in the upper quadrant of the torso. Because of its proximal location in the sclerotome and the extensive convergence of afferent signals from this region to the dorsal horn of the spinal cord, pain reference patterns can be broadly distributed to the deltoid, trapezius, posterior scapular regions, and or the arm all the way to the wrist. This pain behavior can make diagnosis difficult in the shoulder region, as the location of symptoms may or may not correspond to the proximity of the pain generator.[1] Compounding this diagnostic dilemma, imaging results can be misleading.[2,3] Therefore, a thorough history and reliable physical examination should rest at the center of the diagnostic process.[4]

Afflictions in the elevation chain can be categorized as primary or secondary in nature. For example, primary arthropathies are painful disturbances in the joints that develop as result of trauma or disease, such as synovitis, arthrosis, or chondropathy.[5] Conversely, secondary afflictions emerge in tissues adjacent to underlying nonpainful joint, capsuloligamentous or

muscular structures. For example, shoulder impingement and subsequent subacromiodeltoid bursitis often results from posterior capsular limitation at the glenohumeral joint.[6]

CLINICAL EXAMINATION

History

A patient's age and sex can provide the clinician with a glimpse of the patient's disorder, because different disorders are more common within specific sexes and age groups.[5] For example, traumatic synovitis of the glenohumeral joint (GHJ) is more common in individuals older than 45 years of age. One would expect to witness GHJ idiopathic adhesive capsulitis (IAC or frozen shoulder) more frequently in females between the ages of 40 and 60 years.[7] Primary arthrosis of the GHJ is expected to occur more often in the shoulders of individuals greater than 60 years, while the secondary arthrosis associated with previous intra-articular fracture, instability, or rheumatological disorder can be seen in individuals less than 45 years.

Epiphyseal closure of the SCJ can occur later than other joints (as late as 25 years). This creates a dilemma for clinicians, who might interpret a traumatic epiphyseal plate separation as a joint separation in post adolescent individuals. Moreover, sternoclavicular joint (SCJ) hyperostosis is more frequently observed in females between the ages of 30 and 50 years. While this condition is commonly benign, age appears to be related to its incidence.

Increased age appears to increase the incidence of subacromiodeltoid bursitis, as well as the risk for rotator cuff tears associated with instability and subsequent arthopathies. Traumatic, internal rotator cuff tears are more frequently witnessed in younger athletes, while external degenerative tears are observed more commonly among individuals older than 40 years. Furthermore, trauma appears to compromise the labrum more frequently in persons less than 45 years, versus tears to the capsular substance with individuals older than 45 years. While general glenohumeral laxity is more frequently observed in females less than 45 years, traumatic anterior glenohumeral instability is more commonly witnessed in males in the same age group. Finally, subscapularis tendinitis is more common in females between the ages of 40 and 60 years.

Interpreting the relevance of a patient's shoulder pain location can be troublesome to the clinician, due to the shoulder's complex neuroanatomy. This neuroanatomical arrangement lends to large, vague areas of pain and diffuse pain reference distributions.[1] Distal segments within an extremity are more densely innervated and subsequently give rise to more sensory discrimination. Likewise, superficial structures are more densely innervated and give rise to more sensory discrimination than deep segments. In concert with convergence of afferent pathways in the spinal cord's dorsal horn,[8,9] deep proximal joints will refer pain more diffusely than superficial distal joints. This referred pain can be triggered by trauma, inflammation, and muscle contractions.[10] Moreover, this referred pain is a deep aching[11] that occurs distal to the pain generator and is frequently accompanied by referred tenderness[12–14] and possible increased sensitivity of surrounding tissue.[15] Finally, due to the intricacy of this symptom referral pattern and the layers of overlying structures, early shoulder palpation during the clinical examination is likely to have poor diagnostic relevance.

Although complicated, the location of a patient's pain can provide a clue to the clinician regarding the joint system responsible for the patient's pain. Pain from structures associated with the glenohumeral joint is experienced distal to the acromiohumeral interval (AHI) in the C5 distribution. Additionally, acute glenohumeral lesions commonly produce C5 pain in the region of the deltoid versus chronic lesions that produce C5 pain as far distal as the base of the thumb. Conversely, the sternoclavicular and acromioclavicular joints produce pain that is in the C4 dermatomal distribution, proximal to the AHI. Moreover, pain arising from the acromioclavicular joint is localized to the joint's proximity, due to its distal location in the C4 distribution. On the contrary, the sternoclavicular pain is more frequently vague and diffused over the ipsilateral cervical, retroclavicular and pectoral regions, due to the joint's proximal location in the C4 distribution.

Consequently, when a patient presents to the clinic with shoulder pain that is distributed distal to the AHI, one can be confident that the patient is *not* suffering from an affliction of the acromioclavicular or sternoclavicular joints. However, while these joints produce pain that is located proximal to the AHI, one cannot assume that shoulder pain in the ipsilateral cervical or trapezial regions is *absolutely* caused by a sternoclavicular affliction. While pain in this distribution can stem from a host of cervical afflictions, a chronic glenohumeral condition could give rise to spasms and discomfort in this region, due to overuse and accompanying compensatory movements, as well as central

sensitization in the dorsal horn of the spinal cord. Because of these complex possibilities, a tissue-specific clinical examination is merited.

Diagnostic Imaging

Imaging techniques, such as magnetic resonance imaging (MRI) and computed tomography (CT), can be somewhat helpful in the diagnosis of shoulder afflictions. However, the results can frequently be misleading. For example, Miniachi et al (1995), demonstrated that 100% of supraspinatus tendons in asymptomatic shoulders demonstrated a grade-I rotator cuff lesion, described as focal linear or diffuse intermediate signal through the tendon.[16] Needell et al discovered that 75% of asymptomatic shoulders exhibited signs characteristic of ACJ osteoarthrosis, and 33% demonstrated other bony and soft tissue disturbances indicative of shoulder pathology.[3] Hodge et al demonstrated that the use of magnetic resonance imaging for systematically grading shoulder instability consistently underestimated the degree of instability in symptomatic shoulders.[17] While this grading was improved when performed with the shoulder under stress, the investigators suggested further validation was necessary prior to widespread clinical use. Therefore, while imaging studies can serve as a helpful adjunctive diagnostic tool, they should be interpreted in context with a thorough history and a reliable functional examination.

Clinical Examination

Visual inspection of the shoulder and upper quadrant region can be very informative and should not be overlooked. General skin integrity, color and texture are observed. Head, shoulder, scapular position should be noted. Next, atrophy of muscles around the shoulder girdle region might suggest nerve lesions, muscle ruptures, disuse atrophy, or possibly serious pathology in the case of atrophy of the trapezius and sternocleidomastoid muscles, such as a tumor. Deformity in the mid clavicular region might represent a callus mass from a clavicular fracture. A prominent distal clavicle with or without local swelling might suggest ACJ subluxation, while a bone mass over the SCJ might indicate an idiopathic hyperostosis. Fullness in the supraclavicular region might indicate an elevated first rib,[18] while a flattened deltoid appearance is common with traumatic luxation of the glenohumeral joint. Postural disturbances provide clinicians with good information about the patient's problem. Rounded shoulders are often linked to impingement. Finally, holding the arm in the sling position is an assumed posture seen with instability, tendopathy, or acute glenohumeral bursitis, while hanging the arm at the side in effort to increase the subacromial space is often seen in chronic glenohumeral bursitis cases.[5]

Pellechia demonstrated that a clinical examination process according to Cyriax demonstrates a significant degree of construct validity and is reliable between testers for differential diagnosis of shoulder afflictions.[4] A Cyriax-based examination model (see Appendix A) utilizes provocation testing to determine the structure or tissue involved and subsequently suggests tissue-specific treatment strategies. Any shoulder evaluation should begin with a screen of the cervical region. Here, active cervical range of motion is requested while noting quantity and quality of movement, as well as symptom provocation. If cervical movement elicits the patient's pain, a more in-depth examination of the cervical and thoracic outlet regions is warranted.

Shoulder girdle testing begins with the examination of shoulder girdle protraction, retraction, depression, and elevation. Here, the clinician should evaluate for movement quantity and quality, as well as symptom provocation. Because of their important contribution to should girdle function,[19] these tests serve as the first appraisal of ACJ, SCJ and scapulothoracic joint (STJ) function. Frequently, however, small limitations in motion at the ACJ and SCJ will not become evident until end-range arm elevation is evaluated. This subtlety is related to the required clavicular backward spin on the sternum with upper extremity elevation.[20] Therefore, when a clinician suspects ACJ or SCJ involvement, caution must be exercised in the presence of normal isolated shoulder girdle movements. Consequently, a more through examination SCJ and ACJ function should be performed with the arm in an elevated position.

Static and dynamic tests of the STJ help determine whether there is a scapulothoracic junction instability.[21] The first test involves observation of scapula posture with the upper extremities in a resting position, looking for static scapular winging, tipping, or downward rotation. For the second test, the clinician observes for similar scapular postures while the patient places the dorsum of his or her hands on the hips. For the third test, the clinician observes for similar scapular postures while passively positioning the patient's arm at 90° of abduction and again positioned at 90° flexion and internal rotation.

The fourth test evaluates dynamic scapular control during upper extremity elevation and return to resting

position. For the test, the patient is asked to elevate the upper extremity as high as possible, while the clinician observes for scapulothoracic movement disturbances. The clinician notes the elevation strategy selected by the patient (flexion or abduction) and then asks the patient to repeat the procedure using the other strategy. Here, scapular winging, tipping, or downward rotation will most commonly be observed during the eccentric return from elevation at the transition between the elevation and setting phases, in the proximity of 60°.[20,22] These poor scapular postures may suggest weakness or fatigue of parascapular muscles, including rhomboids, trapezius, and/or serratus anterior.[23] Such muscular dysfunction may influence the onset and or severity of shoulder impingement, instability and or elevation limitations[24–26] and, consequently, rehabilitation of these conditions merits re-activation of the para-scapular muscle groups.

Scapulothoracic behavior is not the only feature noted by the clinician during full arm elevation. Pain provocation is equally important to the clinician, especially if a painful arc is observed (discussed later). The clinician should note the location in the range, extent, and severity of the painful arc. In addition, bilateral active arm elevation is compared to unilateral active arm elevation. Patients should demonstrate a greater range of unilateral elevation, due to obligatory rotation of the cervicothoracic spine. If unilateral elevation is not greater than bilateral elevation, the limits in cervicothoracic mobility should be suspected.

Passive arm elevation should be tested in three different directions, so to evaluate all provocation possibilities (See Figure 1). In each case, the arm is passively lifted into elevation, while the quantity of motion and quality of end feel are evaluated. In addition, provocation of symptoms is considered, as differences in provocation patterns between the test directions may represent different afflictions. For the first direction, the contra-lateral shoulder is stabilized while the ipsilateral upper extremity is lifted into full elevation, followed by posterior medial overpressure. If most painful of the three tests, this direction indicates the contribution of the ipsilateral ACJ, SCJ, and cervicothoracic spine to the painful affliction, meriting further tests of those structures. Second, the ipsilateral scapula and acromion are fixated after passively lifting the patient's upper extremity to 150° elevation, and end-range is assessed with posterior medial overpressure, testing for internal impingement of the rotator cuff. Lastly, the ipsilateral scapula and acromion are fixated after lifting the upper extremity to 150° elevation, followed by posterior overpressure at the endrange. If most painful, this test indicates subacromiodeltoid bursal involvement.

Quantity and quality of the motion, as well as symptom provocation, are evaluated during the passive testing of GHJ abduction, internal rotation, and external rotation. Passive GHJ abduction is tested while palpating the inferior angle of the scapula, noting the extent of abduction at the first detectible scapular motion. Internal and external rotation are tested with the humerus at the patient's side, as Cyriax suggested that testing these movements from that resting position

Figure 1. Passive Arm Elevation Testing, where: (a) the contralateral shoulder is stabilized while the ipsilateral upper extremity is lifted into full elevation, finished with posterior medial overpressure at end range; (b) the ipsilateral scapula and acromion are fixated after passively lifting the patient's upper extremity to 150° elevation, finished with posterior medial overpressure at end range; (c) the ipsilateral scapula and acromion are fixated after passively lifting the patient's upper extremity to 150° elevation, finished with posterior overpressure at end range.

Figure 1. *Continued.*

is more specific for determining the pattern of GHJ limitation.[1] External rotation is tested starting in resting position (arms at side) with the elbows flexed to 90 degrees while the contralateral shoulder is stabilized. Passive horizontal adduction can confirm the diagnosis of ACJ pathology when pain is produced at the top of the shoulder (C4 dermatome) at the end range of passive elevation, internal rotation and external rotation. However, passive adduction is not as sensitive as other tests to be described later. In all cases, end-feel is evaluated and overpressure is added if the test was not initially provocative.

Resistive testing procedures should initially include adduction, abduction, external rotation and internal rotation. These tests should be performed from the resting position (with the patient's arm at the side) and should be isometric in nature in order to isolate specific muscle, musculotendinous junction, and tendon components of the rotator cuff. Test results can be witnessed in Table 1, where each member of the rotator cuff demonstrates unique provocation patterns during resistive tests. Unfortunately, subacromiodeltoid bursitis can produce similar provocation during these tests, making the test less specific to rotator cuff tendopathy. However, differential diagnosis between rotator cuff tendopathy versus bursitis can be achieved through the use of the *Pull Test*, which will be discussed later.

INTERPRETATION AND MANAGEMENT

Diagnosis and Management of Capsular Pattern Limitations

After performing the clinical examination, the clinician can proceed with an interpretation of the findings. First, the clinician must observe whether the patient demonstrates limited shoulder motion. If a limitation exists, then the clinician must observe whether the limitation is demonstrated during passive and active motion versus

Table 1. Provocation Test Patterns for Tendons of the Rotator Cuff

	Resisted Abd	Resisted Add	Resisted IR	Resisted ER
Supraspinatus	(++)	(–)	(–)	(+)
Infraspinatus	(–)	(–)	(–)	(++)
Subscapularis	(–)	(–)	(++)	(–)
Teres Minor	(–)	(++)	(–)	(++)
Latissimus Dorsi Pec Minor Teres Major	(–)	(++)	(++)	(–)

active motion only. If the limits are during active motion only (whereby passive motion is within normal limits), then the clinician should suspect a central nervous system affliction, peripheral nerve injury or rotator cuff tear. Conversely, if the limitation is observed during active and passive motion, then the clinician must discern whether the limitation is in the form of a capsular pattern or noncapsular pattern.[5]

The validity and reliability of this differential diagnostic model has been examined within other synovial joint systems. Investigators have demonstrated reliability when using the system at the knee,[27] hip, and lumbar spine.[28] Moreover, Pellechia et al found that this model can serve as a highly reliable schema for assessing patients with shoulder limitation and pain.[4] Therefore, identifying a joint's limitation pattern can guide the clinician towards a more complete diagnostic concept of the patient's problem.

A capsular pattern is a predictable, repeatable and reliable pattern of passive motion limitation that is unique to each joint, is initiated by either intra-articular swelling[29] or protective muscle guarding,[30] and represents either synovitis (arthritis) or arthrosis (degenerative changes) within the joint.[30] The capsular pattern (CP) for the glenohumeral joint is as follows: The external rotation limit is greater than the abduction limit, which is greater than the internal rotation limit. In addition, the ratio of these limits is approximately 3:2:1, respectively.[5] For example, a moderate capsular pattern at the glenohumeral joint could present as a 45-degree limitation in external rotation, a 30-degree limitation in glenohumeral abduction (with the scapula stabilized), and a 15-degree limitation for internal rotation. On the other hand, the capsular pattern at both the acromioclavicular and sternoclavicular joints (ACJ & SCJ) is pain produced at the end-range in all movement directions at the shoulder, without any detectable motion limits. For example, a patient with an ACJ traumatic synovitis would present with C4 pain at the end-range of normal arm elevation, external rotation and internal rotation.

A clinician should pay particular attention to a patient's historical specifics when a capsular pattern has been observed. As suggested, this pattern can represent either synovitis, which can be traumatic or nontraumatic in nature, or arthrosis associated with degenerative changes on the articular surfaces. Traumatic synovitis can be related to a single macrotraumatic event, or associated with a repetitive microtrauma, as sustained while participating in a throwing sport or tennis. In addition, a patient can develop a nontraumatic synovitis when afflicted with septic arthritis[31] or a systemic disease, such as rheumatoid arthritis, psoriasis, or Reiter's syndrome. Furthermore, a capsular pattern will slowly emerge as result of a primary arthrosis, which is associated with the time-based degenerative events that accompany age. Finally, a capsular pattern can be caused by secondary arthrosis, which arises when degeneration is accelerated in response to disease, a previous intra-articular trauma.[5]

Traumatic synovitis and primary arthrosis can be effectively managed when the grade of the condition is considered. A minor affliction, or grade I, can present with a subtle capsular pattern of mildly limited (10° limit) and painful external rotation and, slightly limited (<5° limit) abduction, and painful internal rotation at end-range. The pain is mild and localized to the shoulder, allowing continued function and sleep on either the involved or uninvolved side. This affliction is effectively managed with joint specific mobilization, neuromuscular re-education, home exercises and unloaded activities (such as pulley exercises). A grade II affliction presents with a moderate capsular pattern, inability to sleep on the involved side, and moderate pain often referring into the elbow. This condition can also be managed as a grade I, but may additionally require intra-articular steroid injection. Finally, a grade III affliction presents with severe pain that can refer distal to the elbow and a remarkable capsular pattern where the patient may not be capable of producing any external rotation. The grade III lesion is best managed with rest, mild unloaded exercise and a sequential intra-articular injection series (discussed below), only allowing progressive exercise when the symptoms have begun to recede.

A glenohumeral capsular pattern limitation will also be observed when the patient suffers from idiopathic

adhesive capsulitis (IAC). While the limitations are similar to those observed with other synovial and chondropathic conditions of the glenohumeral joint, the pathology of this nontraumatic capsular transformation appears to be different from other capsular pattern lesions. Although the etiology is not well understood, recent investigations have examined various features of this condition, lending insight into the pathological processes and development of clinical management strategies. For example, recent histological and immunocytochemical findings demonstrate that these processes include active fibroblastic proliferation, accompanied by limited transformation of a portion of capsular tissue into a smooth muscle phenotype likened to myofibroblasts.[32,33] These fibroblasts lay down collagen as a thick nodular band or fleshy mass that is very similar to that witnessed in Dupuytrens disease of the hand, with no inflammation or synovial involvement. This process appears to be most prevalent at the anterior-superior region of the glenohumeral capsuloligamentous complex, especially in the region of the coracohumeral ligament and the rotator interval.[34] Bunker et al suggested that this event cascade might be triggered by production of cytokines and growth factors, which appear to be expressed locally in the capsular tissue.[33] In addition, adhesive capsulitis can result in significant humeral bone loss, which appears to be transient in nature.[35]

This affliction is most common in females (70%) between the ages of 40 and 60. It is more commonly observed in the dominant versus nondominant limb and is more prevalent in diabetic patients.[7] This self-limiting affliction of unknown etiology appears only in the glenohumeral and coxafemoral joints, transitioning through three distinct stages: freezing, frozen and thawing. During the freezing stage, the patient experiences gradually increasing painful limitations in glenohumeral motion. With the examination of passive motion during this stage, the examiner will provoke the patient's symptoms before the end-range of motion is reached, suggesting a painful, empty end-feel. More specifically, the patient will demonstrate a large limitation of elevation through flexion. In addition, external rotation will be more limited in an adducted versus abducted testing position.

After two to nine months time, the patient's condition transitions into the frozen stage, where the limitations and pain plateau. During this stage, the examiner will reach the patient's end-range of motion, where pain is immediately provoked. After an additional four to twelve months, the condition finally reaches the thawing stage where the patient experiences a gradual resolution of pain and variable restoration of movement. During this stage the examiner is able to move the patient's shoulder to the end range with minimal provocation of symptoms.

Careful consideration of the patient's place in the various stages may assist the clinician with designing an effective management strategy. Clinicians have attempted to manage this condition with a variety of different approaches, including injection, manipulation under anesthesia (MUA), distention hydroplasty, and nonoperative rehabilitation. Vanderwindt et al found at 7 weeks post-onset of IAC that 77% of the patients treated with injections were considered to be treatment successes compared with the 46% out of those treated with physical therapy.[36]

While injection has been shown to address the sequelae of idiopathic adhesive capsulitis, the dosage may influence clinical outcomes. Dejong et al obtained greater symptom relief in the treatment of patients' frozen shoulders with a triamcinolone acetonide intra-articular injection of 40 versus 10 mg.[37] These investigators found that the injection produced a greater effect on the reduction of pain and sleep disturbances versus amelioration of motion deficits.

Clinicians have frequently incorporated manipulation under anesthesia as a treatment measure for IAC. Anderson evaluated this measure and early post-procedure continuous passive motion (CPM) on 24 patients with arthroscopically verified frozen shoulders. The investigators found that 79% of the patients reported slight or no pain after the procedure. In addition, 75% returned to work at a mean of 9 weeks after treatment.[38]

Investigators have reported effective management of IAC when incorporating a hydraulic distention of the GHJ capsule (*Distention Hydroplasty*) followed by early post-procedure CPM.[39–43] This procedure is accomplished by introducing 5 ml of 1% lidocaine followed by a bolus of sterile, chilled saline (up to 40 ml) into the GHL capsular compartment through an 18-gauge 1.5 inch needle. Laroche et al found that the mean pain severity of patients suffering from IAC significantly decreased after distention hydroplasty. In addition, passive abduction and external rotation each increased significantly in the first five days after the procedure, whereas the change between day 5 and day 30 was not significant.[42] Similarly, van Royen and Pavlov evaluated the effect of distention hydroplasty with 24 frozen

Table 2. The Effect of Distention Hydroplasty with 24 Frozen Shoulders in 22 Patients at 3-Months Post-Procedure, Reporting % Return of Each Motion Compared to the Opposite Side

93% for shoulder abduction
84% for glenohumeral abduction
94% for forward flexion
96% for backward extension
75% for internal rotation
73% for external rotation

shoulders in 22 patients. The range of motion values at 3 months after the procedure, when compared with the unaffected shoulder, can be witnessed in Table 2.[43] Finally, Halverson and Maas found that 94% of their patients receiving distention hydroplasty for idiopathic arthritis demonstrated immediate and sustained improvement in shoulder pain and range of motion.[44]

Investigators have incorporated arthroscopic release into the management of IAC.[45,46] Ogilvieharris conducted a prospective cohort study on 40 patients suffering from IAC, 20 of which were treated with manipulation and arthroscopy and 20 of which the contracted structures were divided through arthroscopy. The arthroscopic division was performed in 4 sequential steps: (1) Resection of the inflammatory synovium in the rotator interval; (2) Progressive division of the anterior superior glenohumeral ligament and anterior capsule; (3) Longitudinal separation of the subscapularis tendon; and (4) Longitudinal separation of the inferior capsule. Both groups reported similar levels of satisfaction during a follow up at 2–5 years post procedure. However, the patients in the arthroscopic division had significantly greater pain relief and restoration of function.[46]

Diagnosis and Management of Non-Capsular Pattern Limitations

A noncapsular pattern (NCP) of limitation is any pattern of limitation other than the capsular pattern.[5] While any combination of noncapsular pattern limitations can be potentially observed in the shoulder complex, selected patterns appear to be more frequent. Patients commonly present in the clinic with shoulder internal rotation limitation based on functional external rotation requirements, such as throwing and reaching. This condition can be witnessed in athletes participating in throwing or racket sports, and frequently perpetuates impingement behaviors based on altered joint kinematics.[47,48] This condition is best managed with joint specific mobilization to the glenohumeral joint in the posterior translatory direction with the shoulder positioned in adduction and submaximal internal rotation. These activities should be followed with a home stretching program that includes passive shoulder internal rotation and horizontal adduction while the scapula is stabilized.

Patients who suffer from acute subacromial bursitis will commonly demonstrate painfully limited passive and active arm elevation, with internal and external rotation relatively normal. Acute bursitis will present with a rapid onset of severe pain in the C5 distribution that can potentially refer to the wrist and inability to elevate the arm in the direction of abduction or possibly flexion. This condition is related to rupture of a pasty calcific material within a rotator cuff tendon. The pasty, calcific material is caustic to the synovial lining of the subacromial bursa, triggering an aggressive synovial reaction. While this condition is commonly self-limiting, the patient's discomfort can be reduced with rest and a steroid injection into the subacromial bursa.

Diagnosis and Management of Pain without Limitation

Many painful conditions of the shoulder complex present without limitation. Afflictions of the acromioclavicular joint (ACJ) and sternoclavicular joint (SCJ) produce pain in the C4 distribution without limitation. Conversely, pain in the C5 distribution can emerge as the result of nerve entrapment, tendinitis, tenosynovitis, bursitis, impingement, labral affliction and clinical GHJ instability. Each of these conditions presents with distinctive provocation patterns during the functional examination. A comparison of resistive, modified resistive, passive and special tests can lead the clinician to a diagnostic conclusion and appropriate management strategy.

Acromioclavicular joint synovitis (arthritis) develops after macro-or microtrauma, producing pain in the C4 dermatome located directly over the joint. Pain is reproduced at the end-range of all passive shoulder movements and resisted adduction of the arm is often painful. The most pain can be provoked with passive horizontal adduction and resisted horizontal abduction with the shoulder flexed to 90°. Synovitis at the ACJ can present with a hypomobility or be the consequence of joint instability. Hypomobility at the ACJ is a consequence of joint effusion, as well as eventual capsular adaptations, and is best treated with intra-articular steroid injection

followed in 7–10 days by mobilization and or manipulation. On the contrary, instability is typically the consequence of macrotrauma and subsequent failure of the acromioclavicular or coracoclavicular ligaments. This condition presents with increased anterior-posterior or craniocaudal ACJ movement, respectively. Coracoclavicular ligament deficiency additionally leads to a possible prominent distal clavicle associated with a dropped scapula ("Piano key sign"). Instability of the ACJ is best treated with intra-articular steroid injection, shoulder girdle bracing and eventual shoulder girdle strengthening so to utilize the investment of deltoid and trapezius insertions at the capsule of the ACJ.

Patients can develop posterior C5 shoulder pain in the region of the scapula associated with suprascapular nerve entrapment. This condition develops as result of trauma, neuritis, or slow progressive compression at the suprascapular or spino-glenoid notch.[49] Investigators have reported iatrogenic suprascapular nerve injury during Bankhart repair of rotator cuff lesions.[50] The nerve can be injured as result of macrotrauma, as witnessed during proximal humerus fractures.[51]

Microtrauma can lead to suprascapular nerve injury, as repeated shoulder horizontal adduction with scapular protraction during volleyball slams or water polo returns can traumatize the nerve. Here, the nerve is repeatedly traction-loaded proximally through contralateral cervical sidebending and distally through arm and shoulder-girdle prepositioning. Additionally, pitchers can develop this condition after repeated follow-through maneuvers, by virtue of nerve irritation associated with increased supra- and infraspinatus activity during deceleration. This condition is provoked when the athlete's shoulder is placed behind the back and passively loaded in adduction, followed by contralateral cervical sidebending. Furthermore, suprascapular nerve symptoms can develop when a ganglion cyst forms on the posterior labrum in response to a degenerative horizontal tear. Finally, this posterior shoulder pain should be differentiated from posterior labral tear and internal impingement of the supraspinatus against the glenoid labrum and limbus (discussed later).

Suprascapular nerve lesions may demonstrate neurophysiological dysfunction, but a painful injury can present with normal EMG findings. This condition is best detected through the suprascapular neural tension test, whereby the patient's cervical spine is laterally flexed away from the painful side, while the involved arm is placed into horizontal adduction in front of the body or in adduction with the arm behind the body. This lesion can be treated conservatively and or surgically. If this position reproduces the patient's pain, a positive test is confirmed when the pain is reduced with a return of the patient's neck to neutral, thus reducing tension on the nerve. Conservative management can include NSAIDs, steroid injection in the region of irritation, and eventual implementation of a rotator cuff strengthening program. Surgical intervention includes open decompression and possible possible neurolysis.[49]

Patients who suffer from rotator cuff tendinitis typically experience the most pain provocation in the C5 dermatome at the region of the deltoid during resistive testing (See Table 1).[5] Resistive testing should be performed isometrically and with the patient's arm at the side, so to isolate strain within the tendon substance.[1] Diagnosis of subscapularis tendinitis presents a conundrum to clinicians. This tendinitis is provoked with resistive shoulder internal rotation, while other resistive tests are negative. This is an important distinction, due to the contributions of pectoralis major, latissimus dorsi, and teres major to internal rotation and adduction. If any of these other muscles were involved in a painful resistive internal rotation, then resisted adduction would be painful as well. However, painless resistive adduction (with the arm at the side) isolates the subscapularis as the tendon responsible for painful resisted internal rotation.

Because of the powerful contributions of the pectoralis major, latissimus dorsi, and teres major to internal rotation, a patient suffering from a subscapularis tear or rupture may demonstrate a false negative resisted internal rotation test when the arm is positioned at the side and the elbow positioned to 90° of flexion. Thus, investigators have reported the use of additional tests to identify subscapularis involvement, including the lift-off test[52–54] and internal rotation lag sign (IRLS).[55] The lift-off test involves placing the forearm behind the patient's back and asking the patient to maintain the forearm off of the back's surface. If the patient is unable to maintain the position, then the clinician should suspect a complete rupture of the subscapularis (see Figure 2). If the patient is able but the maneuver is painful, the clinician should suspect either tendinitis or a partial tear (suspected if the patient has a history of trauma). Finally, if the patient is limited in shoulder motion and unable to place the test arm behind the back, then the clinician can ask the patient to place the hand of the test arm against his / her stomach and the clinician can proceed to lift the forearm and hand off of the stomach (modified lift-off test).

Figure 2. The Lift-Off Test. (a) Starting position, whereby the patient places the test arm behind the back and the clinician stabilizes the distal humerus; (b) Test movement, whereby the clinician asks the patient to lift the test arm off of the back, while stabilizing the distal humerus and preventing shoulder extension.

When a patient demonstrates pain provocation during any resistive test accompanied by findings of a painful arc during active arm elevation, the clinician should differentiate between tendinitis and subacromiodeltoid bursitis, as activation of any rotator cuff muscle can increase acromiohumeral interval pressure and bursal pain. This is achieved through the "Pull Test",[5] where the clinician repeats the same resistive tests while pulling on the humerus, thereby separating the acromiohumeral interval (see Figure 3). This procedure should resolve bursal pain produced during the previously positive resisted tests, due to eliminating bursal compression. On the other hand, the "Pull Test" would increase pain associated with a tendinitis, as tension loading is advanced within the tendon substance with the pull.

Patients can suffer from tenosynovitis of the biceps long head, where the tenosynovial sheath is inflamed around the tendon as it courses through the intertubercular sulcus.[56] Biceps tendon lesions are commonly associated with degenerative changes in the glenohumeral joint and seldom occur in isolation of other lesions about the joint system.[57] An affected synovial sheath is most painful with tension, thus the patient experiences the greatest pain provocation when the clinician first flexes the elbow with the patient's arm at the side, pronates the forearm, extends the shoulder in the glenoid plane (slightly outward), and finally extends the elbow (see Figure 4). This sequence is emphasized, so to avoid internally rotating the shoulder and producing a false positive test associated with a winding-up of the GHJ capsule. Additionally, tenosynovitis is differentiated from biceps tendinitis at the shoulder, the latter of which produces the greatest shoulder pain during resisted elbow flexion and resisted forearm supination.

While the treatment of symptoms associated with the previously mentioned painful, nonlimited shoulder conditions can include transverse or longitudinal friction techniques, modalities and or tissue-specific injection (to be discussed), these treatments may not completely ameliorate the patient's overall condition. Consequently, the clinician is encouraged to address causation, so to reduce the risk of symptom return or persistence. Tendinitis, tenosynovitis, and bursitis at the shoulder can emerge in response to impingement and or instability. Therefore, accurate identification of these underlying entities should be exercised in concert with pain-relieving management techniques.

Figure 3. The "Pull Test" for differentiating rotator cuff tendinitis from subacromiodeltoid bursitis. For this test, the clinician repeats the previously performed resistive abduction test while pulling on the humerus, thereby separating the acromiohumeral interval.

Shoulder Impingement

As previously mentioned, patients can develop shoulder pain without limitation as result of numerous soft tissue lesions, including tendinitis, tenosynovitis, and bursitis. Frequently, these afflictions can emerge in response to an impingement event in the confined spaces about the glenohumeral joint. Impingement is the most frequent cause of shoulder pain and has served as a diagnostic "waste-basket" for classifying shoulder pain.[58] Nordt et al stated that ". . . It (impingement) has become, to some degree, a catch-all diagnosis for numerous disorders that have diverse causes but similar presentations . . ."[59]

Impingement is an event whereby structures around the glenohumeral joint are friction- or impact-loaded during glenohumeral motion. Impingement has been classified as either external or internal, based on its location and responsible mechanisms. During external impingement the subacromiodeltoid bursa and or external surface of one or more rotator cuff tendons are compressed by surrounding structures, such as the acromion, coracoacromial ligament, or coracoid process. Conversely, internal impingement involves compromise to the deep fibers of the rotator cuff as a consequence of impact loading of those fibers between their insertion on the greater tubercle and the superior and or posterior glenoid limbus and labrum.

External impingement has been comprehensively evaluated and investigators have attempted to associate selected anatomical factors with the incidence and severity of subacromial external impingement (see Table 3). Edelson and Taitz suggested that increased acromion length could propagate increased incidence of impinge-

Table 3. Factors Contributing to Shoulder Impingement

Anatomical Etiologies	Biomechanical Etiologies	Traumatic Etiologies	Degenerative Etiologies	Vascular Etiologies
Acromion Morphology	*Scapulothoracic Instability*	*MacroTrauma*	*Rotator Cuff Degeneration*	*Zone of Lability*
Type I	Winging	Partial RC Tear	Fibrosis	
Type II	Tipping	Complete RC Tear	Hyperplasia	*Ranaud's*
Type III	Upward Elevation			Wringing Out'
	Downward Rotation	*MicroTrauma*		Early Fatigue
Acromion Direction		Inc'd Eccentric Load		
Flat	*GHJ Hypomobility*	Dec'd Stress Tolerance		*Dec'd Stress Tolerance*
Intermediate	Diablo Effect			Inc'd Injury Potential
Steep				Risk for MacroTrauma
	GHJ Hypermobility			
Acromion Length	Instability			
Short	Inc'd Humeral Translation			
Long				
	Rotator Cuff Imbalance			
Os Acromiale	Firing Order Disturbance			

Figure 4. Biceps Stretch Test for biceps tenosynovitis. (a) Starting position, whereby the clinician first flexes the elbow with the patient's arm at the side, pronates the forearm, passively extends the shoulder in the glenoid plane (slightly outward); (b) Test movement, whereby the clinician passively extends the elbow, thus stretching the biceps tendon through the intertubercular sulcus at the shoulder.

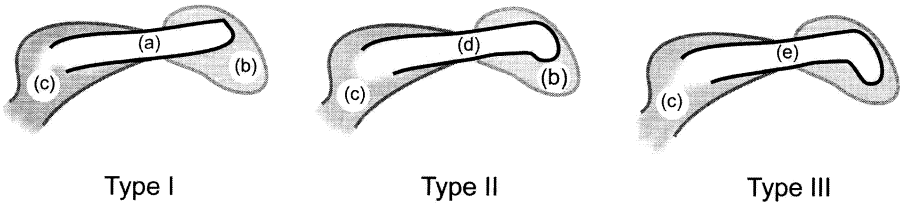

Figure 5. Variations in the acromion process, from an outlet view. (a) Type I: Straight acromion process; (b) Clavicle; (c) Scapular spine; (d) Type II: Mild inferior "hook" on the edge of the acromion; (e) Type III: Prominent "hook" on the edge of the acromion.

ment.[60] In addition, a horizontally oriented acromion is at greater risk for contributing to impingement.[60] Finally, investigators have reported that Os Acromiale, or nonunion and persistence of the lateral acromion past 18 years old, could serve as a probable cause of impingement.[61–64]

Historically, Bigliani classified the shape of the acromion process (Types I–III; see Figure 5) and suggested that differences could influence the incidence of impingement and any subsequent rotator cuff tearing.[65] Later, Bigliani et al suggested that 78% of all full thickness rotator cuff tears were associated with Type III acromia.[65] Investigators have suggested that differences in the acromion are acquired, resulting from altered tension loads imposed by the coracoacromial ligament and deltoid.[66,67] Getz et al observed that Type III acromia were more common in female patients and discovered that Type II acromia were

related to posterior capsule adaptive shortening of the glenohumeral joint.[68]

Although external impingement has been associated with advances in age, the relationship between age and incidence of Type III acromia is controversial.[68,69] In addition, other investigators have identified a poor relationship between acromial classification and rotator cuff lesions. For example, Zuckerman et al observed a poor clinical agreement between impingement and clavicle type and Banas et al observed a similar outcome between clavicle type and MRI-determined rotator cuff disease.[70,71]

Although surgeons have attempted to reduce the clinical sequelae of impingement by surgically removing the coracoacromial ligament and Type II or III acromion projections, symptoms can persist after such a procedure. In addition, Levy et al found that the coracoacromial ligament can regenerate after excision, questioning the efficacy of these measures in managing impingement.[72] However, while the role of subacromial architecture in impingement has remained controversial, Flatow et al identified other factors that could decrease the acromiohumeral interval, leading to increased interval pressure and subsequent symptoms.[73] These factors include rotator cuff hypertrophy, a protracted resting scapular position, and posterior capsular adaptive shortening.

The supraspinatus and infraspinatus demonstrate the potential for hypertrophic changes, especially in concert with excessive overhead activity. Excessive overhead activity can lead to inflammation and subsequent hypertrophic thickening of the rotator cuff, resulting in friction and impingement of the tendons or bursa.[74,75] These changes can be witnessed in occupational or sporting endeavors, such as swimming, throwing, and weightlifting.

Impingement can be exacerbated by scapulothoracic instability, which can develop as result of static or dynamic dysfunction in the lower trapezius or serratus anterior. Static dysfunction results in a winged, tipped, or downwardly rotated scapular position with the arm at the side or in a position of 90° to 100° abduction. Conversely, dynamic dysfunction demonstrates the same scapular behavior during an elevation movement sequence, where the scapula wings, tips, or downwardly rotates while the arm is elevated or returned from elevation to the starting position.[21] In any case, malpositioning of the scapula produces decreased AHI space, increased intr-interval pressure, and subsequent symptoms.

Investigators have identified numerous other pathomechanical behaviors in the shoulder complex that could perpetuate an impingement event. Laxity or instability of the glenohumeral joint may produce excessive aphysiological translation of the humeral head, potentially increasing interval pressure in a similar fashion as previously described.[74,76] Similarly, rotator cuff imbalance could lend to poor control of humeral head position leading to excessive superior translation, repeating the AHI pressure elevation and symptom production.[77] Hypomobility in the glenohumeral joint could perpetuate impingement, as well. A common example is witnessed with glenohumeral internal rotation limits, where the posterior capsule is tight and the humeral head is forced to migrate cranially during elevation activities. While this affliction can be accompanied by anterior capsular laxity, the subsequent migration lends to increased interval pressure and pain.[6]

Chronic external impingement can induce rotator cuff degeneration.[78] While cuff degeneration is multifactorial, chronic tendon injury can produce calcium phosphate accumulation and subsequent narrowing of the interval with accompanying elevated interval pressure.[79] In addition, degeneration can result in decreased stress tolerances and altered strain behaviors of a tendon, thus lending to further degenerative changes.[80] Progressive degeneration can lend to both glenohumeral

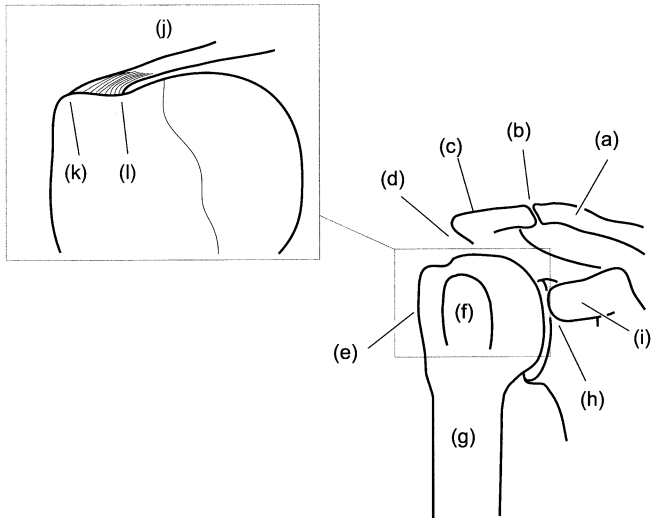

Figure 6. (a) Clavicle; (b) Acromioclavicular joint; (C) Acromion process; (d) Acromiohumeral interval; (e) Greater trochanter; (f) Lesser trochanter; (g) Humeral shaft; (h) subcoracoid interval; (j) Supraspinatus tendon approaching the insertion into the greater tubercle of the humerus; (k) External insertion of the supraspinatus; (l) Internal insertion of the supraspinatus.

instability and rotator cuff hyperplasia, resulting in an impingement event. These degenerative processes appear to be predisposed by hypovascularity in the mid-tendinous region of the cuff. This "zone of lability" has been known for its contribution to compromised stress tolerance and subsequent degenerative tearing.[81]

External impingement can be localized to the subacromial or subcoracoid spaces of the shoulder complex (see Figure 6). A subacromial external impingement event is more common with individuals greater than 30 years of age and is associated with chronic rubbing of the supraspinatus and infraspinatus tendons, the proximal insertion of the biceps brachii (long head) and the subacromiodeltoid (SAD) bursa.[78] The bursa appears to be the most frequent pain generator involved in external impingement by virtue of the unequalled sensory nerve supply.[82–85] However, the rotator cuff can also be compromised, where gradual external tearing may ensue. Clinically, the patient with external impingement presents with pain in the C5 distribution that manifests as a mid-range painful arc (80°–130°) during execution or return from arm elevation through flexion and or abduction.[86] Although the patient may present with additional findings of tendinitis or bursitis in the examination (previously discussed), this painful arc is the clinical hallmark of external impingement and its presence can be a strong diagnostic confirmation.[58] The diagnosis can be confirmed with the external impingement test, whereby the clinician stabilizes the shoulder girdle, then internally rotates the humerus, passively flexes the shoulder to 90°, horizontally adducts the arm, and finally stresses the shoulder into end-range internal rotation (see Figure 7). In doing so, symptoms are produced as the greater tubercle is forced cranially into the

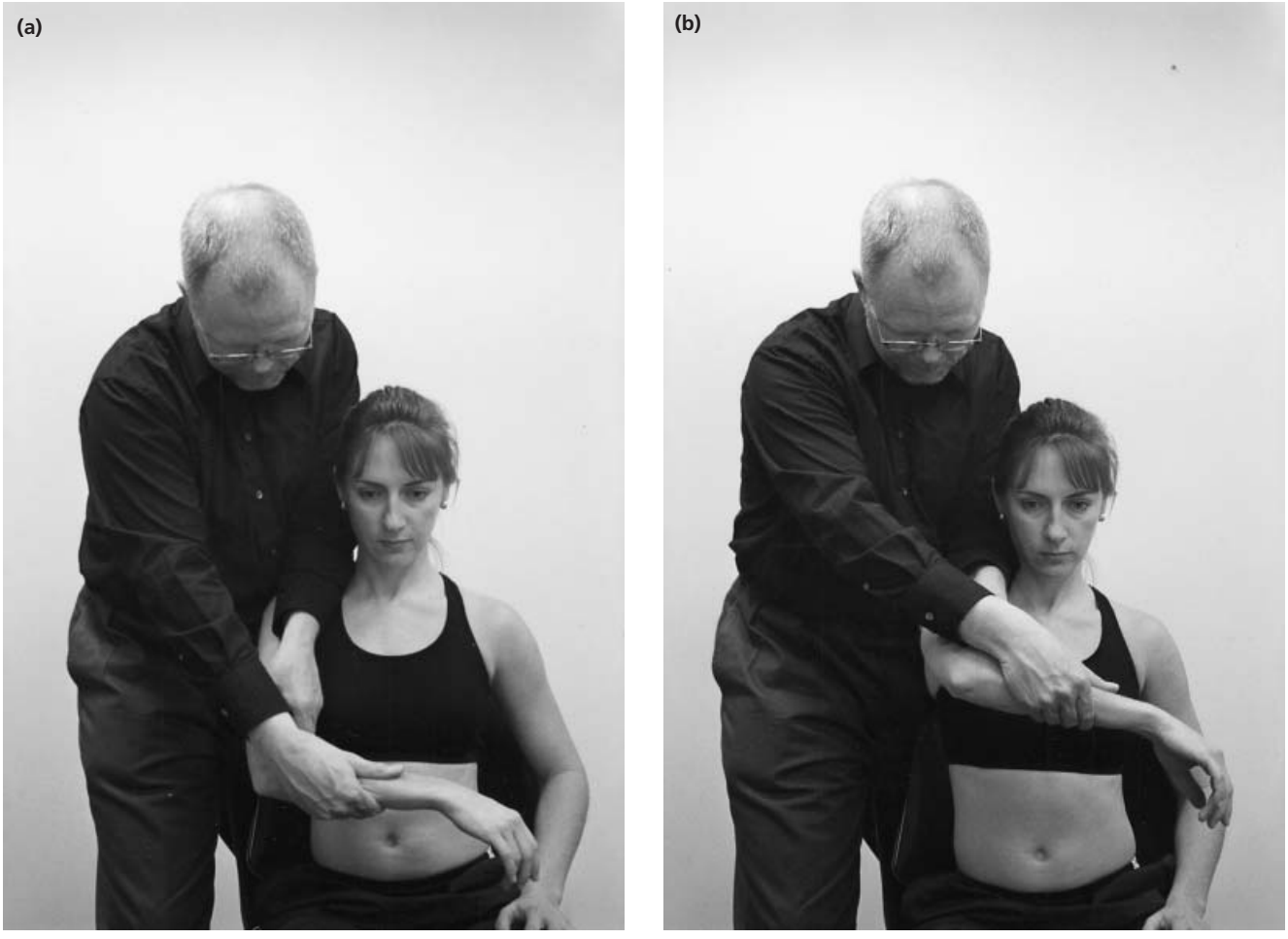

Figure 7. The External Impingement Test, whereby (a) the clinician stabilizes the shoulder girdle, then internally rotates the humerus, and (b) passively flexes the shoulder to 90°, horizontally adducts the arm, and finally stresses the shoulder into end-range internal rotation.

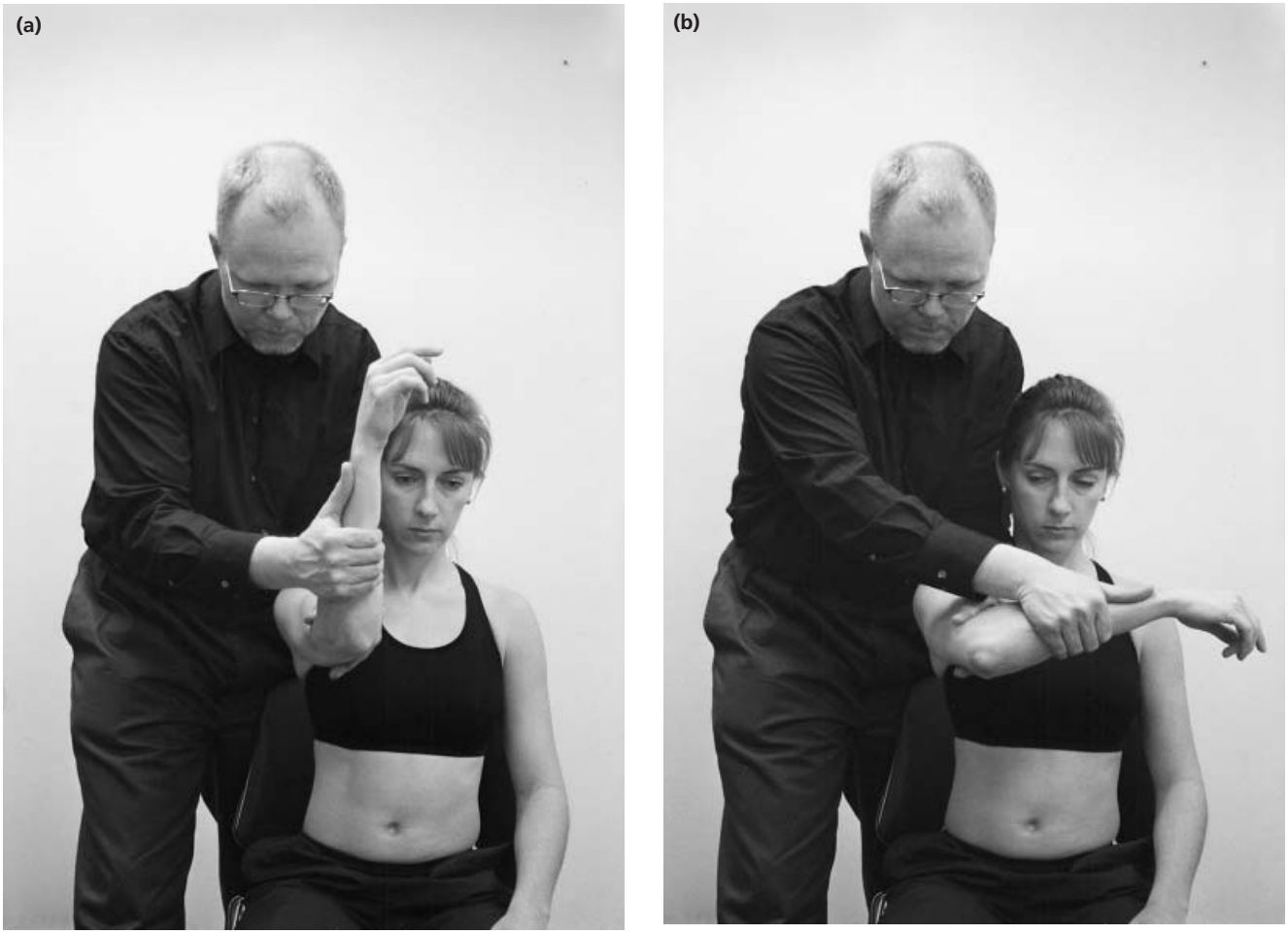

Figure 8. The Anterior (or Subcoracoid) Impingement Test, where (a) the clinician stabilizes the shoulder girdle, flexes the shoulder to 90°, horizontally adducts the arm and then (b) internally rotates the shoulder.

acromion, subsequently escalating pressure within the AHI.

External impingement can develop anterior to the glenohumeral joint in the subcoracoid interval (see Figure 6). This subcoracoid impingement commonly involves an impingement of the anterior soft tissue structures against the coracoid process and appears to be related to the extent and angle of coracoid process projection.[87] Clinically, the patient suffers from shoulder pain in the C5 dermatome and exhibits a mid-range painful arc during flexion elevation between 80° and 130° as the coracoid compresses the subcoracoid bursa, fibers of the subscapularis, biceps tendon or biceps tenosynovium.[53] This affliction can best be provoked with the anterior (or subcoracoid) impingement test, where the clinician stabilizes the shoulder girdle, flexes the shoulder to 90°, horizontally adducts the arm and then internally rotates the shoulder (see Figure 8). This anterior impingement test resembles the External Impingement Test, while differing in the test sequence. Whereas internal rotation is performed early in the External Impingement Test to ensure lesser tubercle clearance prior to elevation, it is the final step of the anterior impingement test, so to produce impingement of the same tubercle against the coracoid process.

Internal impingement is more common in individuals who are less than 30 years of age. This affliction occurs when the deep fibers of the supraspinatus or infraspinatus impact loads against either the superior or posterior glenoid labrum and limbus (see Figure 9).[88–90] Additionally, this impact loading can produce an accompanying bone contusion when accompanied by macrotrauma.[91] The most common etiologies for this condition include throwing[2] and overhead sports such as water polo.[92] Internal impingement of the deep

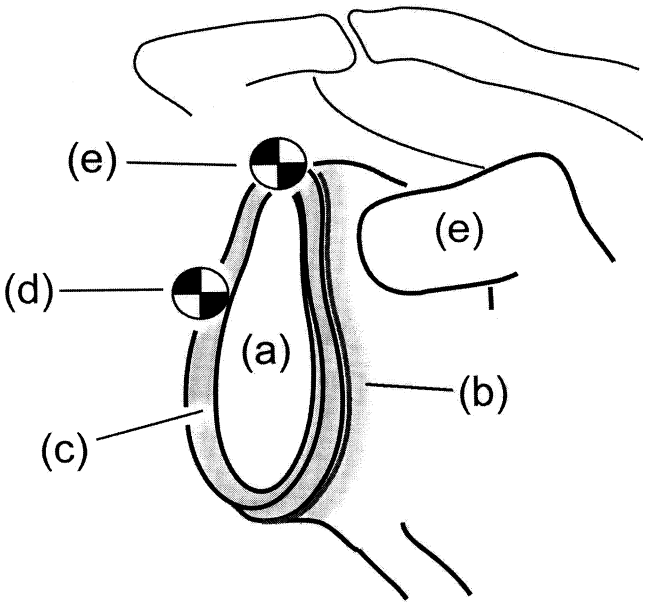

Figure 9. Location of internal impingement events at the glenoid labrum and limbus. (a) Glenoid fossa; (b) Glenoid limbus, or rim; (c) Glenoid labrum; (d) Site for internal impingement on the posterior labrum and limbus; (e) Site for internal impingement on the superior labrum and limbus.

supra- and infraspinatus tendon fibers can occur against the superior glenoid labrum and limbus during overhead elevation activities.[93–94] While C5 shoulder pain is once again produced, the symptoms are provoked during passive end-range elevation. The clinician can provoke the symptoms by passively elevating the shoulder in the abduction direction, then exerting a passive overpressure force to the humerus in a posterior-medial direction while stabilizing the shoulder girdle. The clinician should note that in instances of internal impingement, there is no painful arc during active arm elevation.

For posterior internal impingement, patients experience posterior shoulder pain in the deltoid region that is provoked in the terminal cocked position of the throwing sequence (full GHJ Abduction / external rotation).[92,93,95] A clinician can suspect this affliction when the greatest pain is produced during passive overpressure to glenohumeral external rotation in this position. Associated lesions can frequently develop, including labral cyst, axillary nerve entrapment, greater tubercle sclerosis, limbus erosions, humeral head osteochondral defects, and Bennett's lesion.[92] A posterior labral cyst can emerge as a sequelum of a posterior labral tear or GHJ instability and can result in nonspecific posterior shoulder pain.[96] While imaging can be useful for the detection of erosions, sclerosis, and chondral defects, any associated labral cysts and axillary nerve lesions can be elusive to the diagnostician as they can be difficult to view on imaging studies.

Finally, pain in the C4 dermatome can be provoked during terminal elevation activities when a patient suffers from impingement of the greater tubercle against the inferior surface of the acromioclavicular joint (ACJ; see Figure 6). This pain will commonly be produced at 160°–180° of flexion or abduction elevation. The clinician must differentiate this affliction from superior internal impingement, as both afflictions produce pain at terminal elevation range. Thus, an aid to this differentiation is identifying whether the pain is produced in a C4 or C5 distribution during terminal passive elevation.

When managing impingement, the clinician should execute measures for symptom alleviation and correction of any causative factors. Symptoms that are associated with bursal irritation are best treated with injection. Injections should be administered to the entire subacromiodeltoid bursal compartment, as the bursa can demonstrate septally-divided compartmentalization and the corticosteroid may not reach the inflamed region with an injection in a single area of the space.[5] Iontophoresis can also be used to reduce inflammation, alternating electrode placement to insure access to maximum bursal regions. Placing the electrode anterior to the acromion and positioning the shoulder in slight extension with internal rotation can allow the iontophoresis to access the anterior region. Conversely, the shoulder should be flexed to 60°, horizontally adducted and slightly externally rotated when the electrode is placed posterior to the acromion.

After three to four days rest, clinicians can follow injection or iontophoresis with "bursal massage" For this technique, the patient is positioned supine with his or her arm at the side. The clinician performs small arc passive oscillations to the shoulder in the direction of internal-external rotation, while maintaining a longitudinal pull to the humerus in order to increase the subacromial space. This activity should be performed for several minutes to activate the subacromial gliding mechanism,[82] to decrease inflammation and reduce pain via mechanoreceptor stimulus. The patient can produce a similar activity at home by performing active small arc internal-external rotation oscillations while gently squeezing a towel roll between the arm and trunk, thus activating the adductors of the shoulder. This resisted adductor activity increases the sub-acromial space in a similar fashion to a long axis humeral pull, thus reduc-

ing any friction between the synovial surfaces during the repetitive motion.

Winkel et al has suggested several measures to address the tendopathies that develop with external or internal impingement.[5] First, both transverse and longitudinal friction can be applied to the supraspinatus, infraspinatus, subscapularis, or tenosynovium of the biceps long head within the bicipital groove. It has been suggested that this measure can promote pain reduction via mechanoreceptor stimulation, collagen reorganization, and increased fibroblastic activity. In addition shoulder positions have been described that relocate the rotator cuff tendons out from under the acromion.[5] The supraspinatus can be located just anterior to the acromion when the shoulder is prepositioned in full internal rotation (the patient's forearm is positioned behind his or her back). Conversely, the infraspinatus insertion is best located 1–1.5 inches caudal to the posterior acromion angle with the patient's shoulder positioned in 60° flexion, slight horizontal adduction and external rotation. In this position, the insertion is palpated on the posterior ridge of the greater tubercle on an imaginary line between the posterior acromion angle and the axilla. Friction massage can be applied to any tendon for several minutes in directions both transverse and longitudinal to the orientation of tendon fibers.

Friction techniques should be followed by gentle stretching in both the clinic and as a component of the home exercise program.[97] Finally, injection using 0.5–1.0 mL corticosteroid and local anesthetic can be administered to insertion tendopathy of the rotator cuff, accessing the insertions with the previously described prepositions. The biceps tenosynovium can be injected as well, while care must be taken to not infiltrate the tendon substance. Considering the catabolic influence of corticosteroids and the relative hypovascularity of long tendons, an infiltration to the midsubstance of the biceps tendon may place that tendon at risk for rupture or tendinosis.

Once symptom alleviation is under way, the clinician should incorporate measures to eradicate causative factors. Associated muscle imbalances should be resolved (Jerosh et al, 1989), especially when the impingement is accompanied by laxity or instability of the glenohumeral joint or scapulothoracic junction.[77] It behooves the clinician to begin with retraining of the scapular stabilizers, specifically the rhomboids, lower trapezius, and serratus anterior. Serratus anterior exercises should emphasize eccentric control in the closed chain with the shoulder girdle positioned in retraction. Secondly, the clinician can initiate resisted shoulder adduction, as this will decompress the subacromial space and reduce impingement. Following this, the patient can begin training of the shoulder internal rotators in the closed chain with the shoulder girdle positioned in retraction, based on the importance of subscapularis for elevation activities.[98,99] Additionally, infraspinatus appears to play a significant role in the elevation behaviors of the shoulder and, therefore, should be activated.[98,100,101] However, due to the potential deleterious influence supraspinatus activity can have on the impingement event, resisted external rotation is initiated last and resisted shoulder abduction is completely avoided.[99,102] Finally, because neuromuscular re-education and coordination appear to supercede hypertrophic change when normalizing the shoulder function of the impingement patient, exercises should emphasize endurance versus strengthening. With this in mind, the clinician should ask the patient to ultimately perform the exercises for 100–200 repetitions at 25% maximum voluntary contraction (MVC). In addition, the exercise program should emphasize eccentric contractions, as this format appears to be more relevant to the rotator cuff's action during functional activities.

When an impingement patient presents with posterior capsular limitations, the clinician should implement joint-specific mobilization and home exercise to stretch the posterior capsule and restore normal glenohumeral internal rotation.[103–105] More specifically, superior posterior capsular limitations can be treated when the clinician translates the humeral head in a posterior, slightly lateral, and slightly cranial direction with the patient supine and the shoulder positioned in internal rotation and slight abduction (see Figure 10).[103,106] The patient can follow this with a simple home exercise of placing the hand in the rear pants pocket or behind back; followed by pulling the elbow forward while the scapula is kept retracted against the wall (see Figure 10). Conversely, a posterior-inferior capsular limitation is addressed when the clinician performs a posterior-superior-lateral glide of the humeral head with the patient supine and the shoulder positioned in 60° flexion, horizontal adduction, and minimal IR (see Figure 11). For the home exercise, the patient lies on involved side in a semi-supine position such that the scapula is flat on mat and the shoulder elevated to 60° (see Figure 11). In this position, the patient uses the other hand to push the involved forearm toward the mat while the elbow is kept at 90°, thus internally rotating the shoulder. While the clinician performs joint-specific

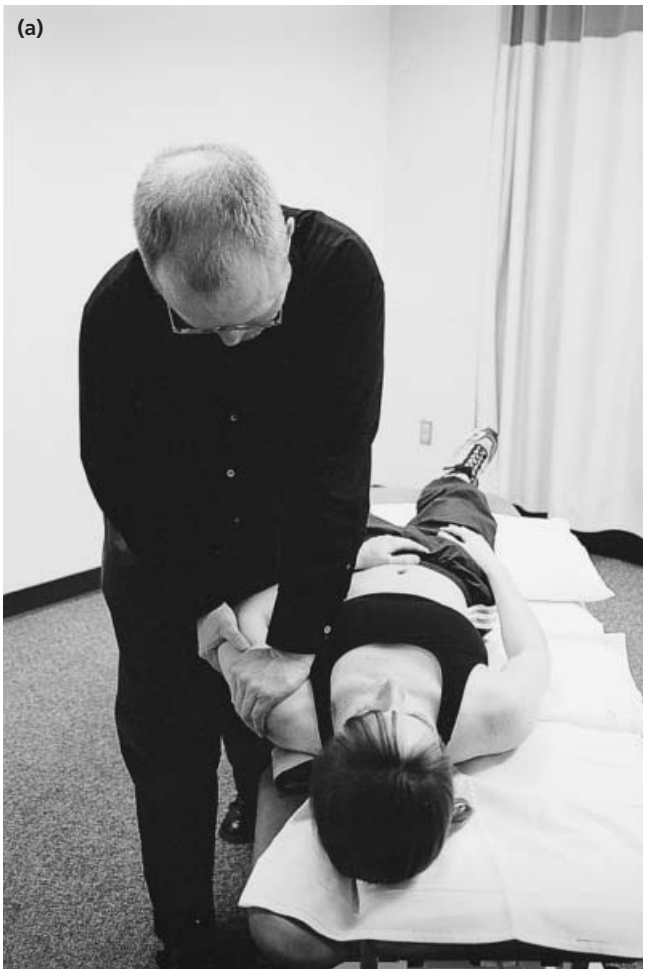

Figure 10. (a) Manual treatment of a superior posterior capsular limitation, where the clinician translates the humeral head in a posterior, slightly lateral, and slightly cranial direction, while the patient is supine and the shoulder is positioned in internal rotation and slight abduction. (b) Home exercise where the patient places the hand in the rear pants pocket or behind back; followed by pulling the elbow forward while the scapula is kept retracted against the wall.

mobilizations several times a week to coax the capsule to adapt, the patient can perform the 20 repetitions of the home exercises several times per day.

Limitations in the sternoclavicular and acromioclavicular joints (SCJ, ACJ) can influence shoulder girdle function and result in end-range elevation limitations. These limitations can contribute to an impingement event, causing abnormal biomechanical behaviors in the elevation chain with resultant elevated AHI pressure. Thus, resolving limitations in the SCJ and ACJ can alleviate impingement-related symptoms. To achieve normal movement in these joints, One can use joint-specific mobilization in a position of end-range arm elevation. These techniques include anterior curved slide of clavicle on acromion at the ACJ, as well as caudal (slight lateral and slight ventral) slide of the clavicle on sternum at the SCJ. End-range elevation accounts for the backward clavicular spin and subsequent twisted capsules at each joint during elevation.[20]

Instability

Patients can develop instability in the shoulder that lends to a variety of clinical sequelae, including tendopathy,[107] impingement,[58] degeneration,[108,109] and labral afflictions.[96] Prior to effectively examining patients for shoulder instability, one must understand the difference between joint laxity and instability. Laxity is a nonpathological state of increased mobility in a particular direction due to decreased tension or resistance to movement that is normally provided by the bony or soft tissue structures. Instability is a pathological condition that is linked to dissociation of the articular surfaces, producing aphysiological loading and subsequent pain in from an associated soft tissue structure. Whereas

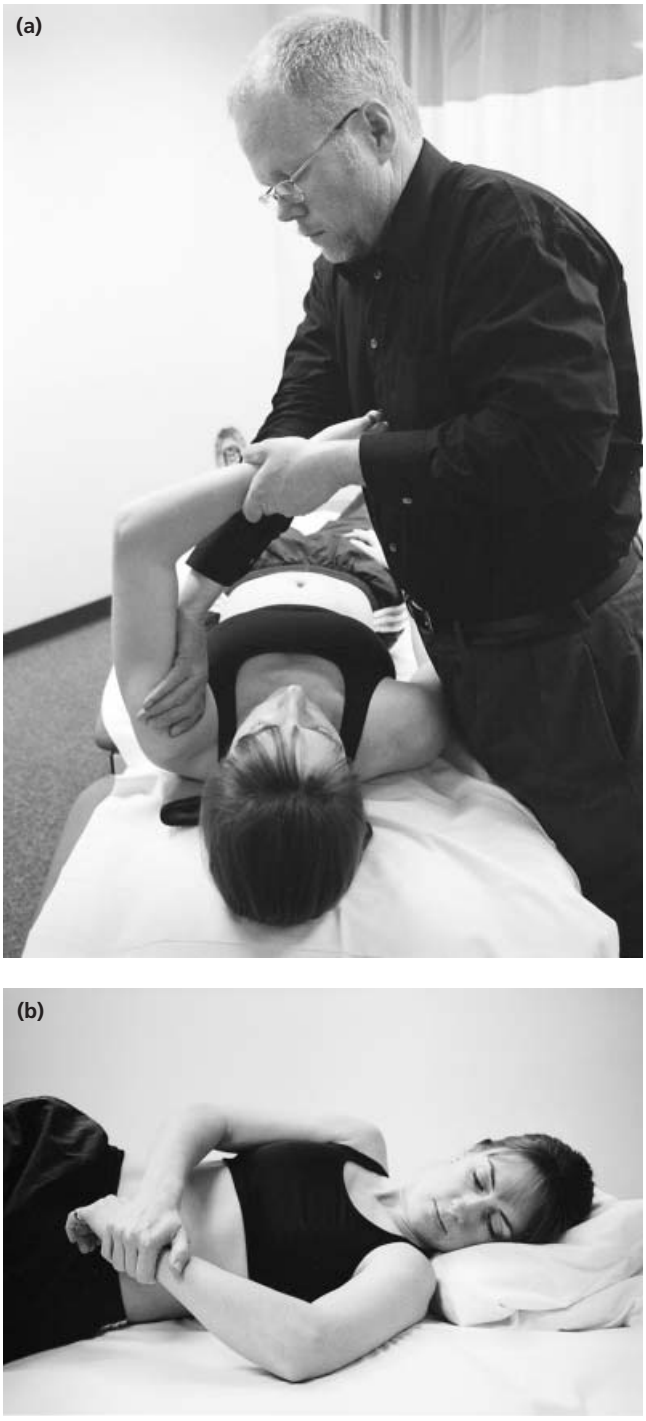

Figure 11. Manual treatment of a superior posterior capsular limitation, where the clinician performs a posterior-superior-lateral glide of the humeral head, while the patient is supine and the shoulder is positioned in 60° flexion, horizontal adduction, and minimal IR. (b) Home exercise where the patient lies on involved side in a semi-supine position such that the scapula is flat on mat and the shoulder elevated to 60°. In this position, the patient uses the other hand to push the involved forearm toward the mat while the elbow is kept at 90°, thus internally rotating the shoulder.

laxity does not necessarily lead to instability, an unstable shoulder can emerge from a lax state. Both laxity and instability can take place in concert with shoulder impingement, where the impingement event occurs either in response to laxity or in concert with uni- or multidirectional instability.[58] Therefore, the clinician is charged with the task of differentiating the patient's symptoms between pain related to impingement versus instability, as each may require different, albeit interrelated, management strategies.

The stability of a joint relies on both static and dynamic supportive mechanisms. The sources of static stability in the glenohumeral joint include the bony architectural configuration and the integrity of the capsuloligamentous structures that have been previously reviewed. Dynamic mechanisms not only include the activity of the muscles around the joint, but also involve the capsule once again, as the rotator cuff dynamizes the capsule through collagenous investment. Failure in any of these components may result in a compromise of glenohumeral stability, resulting in aphysiological loading within the joint and ensuing signs and symptoms of instability. Whereas the glenohumeral joint normally exhibits arthrokinematic rolling, spinning and translation, an unstable mechanism produces increased articular translation and potential for subluxation or dislocation. Although all instabilities do not result in frank articular perching or dislocation, these behaviors are consistent with increased severity of the unstable condition.

The etiological categories of glenohumeral instability include macrotraumatic, microtraumatic, nontraumatic, acquired, and congential lesions.[110] Macrotrauma can involve an over-stretch or tearing of the capsuloligamentous complex, resulting in reduced motion constraint. These lesions, which are more common in individuals under 45 years of age, can be accompanied by a Bankhart lesion that can disrupt the labrum and bony limbus, or Hill Sach's Lesion that produces a dent in the cartilage and subchondral bone after the humeral head is impact-loaded over the labrum and limbus. Consequently, radiographic studies should include special views to delineate these specific lesions.[111] On the other hand, microtraumatic lesions will result from a gradual overstretch to the capsule, as witnessed during throwing or other overhead activities. Acquired nontraumatic instability can develop as result of neuromuscular afflictions, such as poliomyelitis, suprascapular nerve entrapment, or cerebrovascular accidient. In these cases, the locomotor system has failed to serve as a dynamic

constraint to movement, resulting in laxity and painful instability. Moreover, the passive constraint of the capsuloligamentous mechanism can also be compromised by enzymatic degradation that accompanies systemic arthritides, such as rheumatoid arthritis.

Nontraumatic glenohumeral instability can emerge in response to congenital deformities. The capsuloligamentous complex can be overstretched during normal movement when the glenoid fossa is more vertically inclined, as well as in concert with torsional deformities at the scapular neck. In addition, architectural disproportion can contribute to instability. For example, the constraints on the glenohumeral joint are compromised when the radius of the glenoid fossa is proportionally small or the humeral head is either too large or small in relation to the glenoid. Furthermore, humeral antetorsion will excessively stretch the anterior glenohumeral capsule over the humeral head in external rotation.[112] Inherited general laxity conditions, such as Marfan's or Ehler's Danlos, can compromise the collagen within the capsule and ligaments, rendering it vulnerable to injury and failure. Finally, females appear to be predisposed to glenohumeral instability, in response to increased joint laxity.[113]

Instability can emerge after either greater tubercle fracture or tear of the rotator cuff and or biceps long head. Individuals can develop partial or complete tears in the rotator cuff in response to either macro- or microtrauma. These tears can extend through the supraspinatus,[114] infraspinatus,[115] and subscapularis [116] and can either present as full thickness or partial thickness,[117,118] where the tear is observed on either the bursal or articular sides of the tendon.[119] Rotator cuff tears can be acute, where they develop in younger individuals as result of abrupt macro trauma.[120] Neer suggested that single-trauma tears are more common with individuals under 40 years of age and can occur due to either a traction or dislocation force.[121] These tears frequently involve the anterior cuff, especially at the rotator cuff interval, and can lead to multidirectional instability.[122]

A rotator cuff tear can be chronic in the 50–60 year old male as a consequence of a repetitive microtraumatic event, such as the recurrent external impingement that is related to glenoid ostephytic changes[123] or exostotic changes in the underside of the acromion process.[119] Degenerative rotator cuff tears appear to be more frequently initiated on the articular side near the insertion of the tendon,[116] most likely related to the compromised blood supply at that region.[119] While rotator cuff tears can be the source of significant shoulder pain,[124] they can also present asymptomatically,[16,125] lending to biomechanical changes that may manifest as a painful condition in later years, such as subacromial bursitis.[125]

The onset of symptoms associated with rotator cuff tear may be related to advancement of the tear and associated biochemical changes within the joint.[125,126] Large rotator cuff tears appear to accelerate cartilage degradation, as evidenced by increased matrix metalloprotease markers in the synovial fluid. Considering the vital role the rotator cuff plays in the stability of the glenohumeral joint, one can recognize the relationship that rotator cuff failure has with instability and subsequent degeneration.[122]

Pathologically, rotator cuff tears present with fatty degeneration within the tendon[114] and amyloid deposition within the region of the tear.[127] These changes lend to irreversible structural changes, tendon retraction and subsequent joint degeneration. Degenerative joint changes appear to be accelerated by the release of metalomatrix proteinases (MMP-1 and MMP-3) that degrade cartilage. Yoshihara et al observed increased levels of these proteinases, accompanied by increase levels of glycosaminoglycans (GAGs), in the synovial fluid of the glenohumeral joint after cuff tear.[126] In addition, they observed a correlation between the extent of the tear and the concentration of these molecules, suggesting a relationship between the extent of the tear and the potential for degenerative changes in the joint.

Rotator cuff tears can be difficult to diagnose. Clinically, traumatic rotator cuff tears can present with a significant capsular pattern limitation as a consequence of traumatic synovitis. However, a hallmark of these injuries is the patient's inability to actively elevate the arm when the either the infraspinatus and or the subscapularis tendons are torn. As the swelling and inflammation subside, full thickness tears can present with normal passive motion accompanied by the previously mentioned resistive tests that are weak and painless. However, motions can eventually become increasingly painful as degenerative changes progress.[125] Moreover, partial tears are commonly symptomatic, potentially producing painful weakness during the same resistive tests. Because rotator cuff tears can resemble tendopathies about the shoulder, they should be ruled out with the appropriate imaging studies after a macrotraumatic event and or impingement symptoms persist in the aging individual.

Patients suffering from a degenerative tear will frequently present with signs of impingement, including painful arc and possible subacromial crepitus. In either case, the patients may feel weak with shoulder use and may demonstrate a "Shrugging Sign", where the shoulder girdle elevates during attempts to elevate the arm. If either a tear of the infraspinatus or subscapularis accompanies the supraspinatus tear, then the patient will demonstrate a positive Drop Arm Test, where the patient is unable to maintain a previously positioned shoulder in 90° abduction.[128]

Several imaging techniques have been suggested as concomitants in the diagnosis of rotator cuff tears. Multidirectional plain-film imaging could be useful in detecting osseus lesions, such as glenoid osteophytes and or acromion abnormalities.[123] High-resolution sonogram has been proven to be useful in the accurate detection of a cuff tear lesion.[125,129] While Miniachi et al suggested that nonenhanced MRI is of limited value for the clinical detection of rotator cuff injury, they did suggest its utility with full thickness rotator cuff tears.[16] Similarly, Motamedi et al found that MRI could be useful in the detection of full thickness, recurrent rotator cuff tears in the post-surgical shoulder.[117] However, these investigators suggested that the MRI tended to over-diagnose cuff tears and was limited in determining the size of the lesion.

Trauma can injure the patient's glenoid labrum along with the capsuloligamentous complex, thus complicating the clinical picture. Labrum lesions promote instability, due to compromise of the negative intra-articular pressure and successive increased humeral head translation. Patients can suffer from Bankhart lesions, seen especially at the inferior anterior labral region.[122] These lesions have been classified as: (1) Type 1, or an avulsion of the labrum from the glenoid limbus; (2) Type 2, or avulsion of the labrum and limbus; and (3) Type 3, or avulsion of the labrum limbus and fragment of the glenoid body. These lesions can be detected with appropriate imaging and are best managed with surgical repair and or articular augmentation.

Another type of labral lesion from which patients can develop instability is a lesion to the superior labrum, or SLAP lesion (Superior Labrum Anterior-to-Posterior). Investigators have reported various classification systems that reflect location, involved tissues, and or causation.[130–131] Type I lesions involve a fraying and degeneration of the superior labrum, whereas Type II lesions demonstrate a detachment of the biceps and superior labrum from the glenoid. Type III lesions demonstrate a clean bucket-handle tear limited to the labrum and Type IV lesions demonstrate an extension of the bucket handle into the biceps tendon substance. A Type V lesion is a superior extension of a Bankhart lesion with a separation of the biceps tendon from the glenoid, while Type VI lesions demonstrate a flap tear of the labrum. Finally, the labral injury of a Type VII lesion extends caudally into the middle glenohumeral ligament.

Causes of a SLAP lesion include forced biceps activity under load, falls to the outstretched hand, traction injuries, and shear loads associated with throwing.[122,132,133–134] McGough et al suggested that high tensile load tolerance of the proximal biceps tendon predisposes the labral-limbus interface to disruption.[135] Additionally, Pradhan et all found that labral strain is highest during the late cocking phase of throwing, where the shoulder is positioned in maximum external rotation. This strain is followed by eccentric biceps contraction during follow through, where the muscle decelerates rapid elbow extension.[136] These adjacent strain behaviors predispose the tendon-labrum interface to injury. Furthermore, Healey et al reported that the long head of the biceps (LHB) insertion into the supraglenoid tubercle provides 52% of the linear stiffness of this tendon / cartilage / bone interface, while the labrum offers 15% of the linear stiffness. These authors reported that both the bony and the labral insertions must be disrupted in order to create a Type II SLAP lesion.[137] Consequently, Patton and McCluskey suggested that these relationships merit new surgical techniques that preserve the biceps-labral complex.[138]

Glenohumeral instability can be classified as voluntary or involuntary. Patients suffering from voluntary instability possess the ability to sublux or dislocate their afflicted shoulders on command. These individuals frequently gain social benefit from this capacity and represent poor surgical candidates, due to the propensity for post-operative dislocation. Conversely, the clinical symptoms of involuntary instability occur spontaneously, causing sharp pain and a dead-arm feeling. This event occurs when the patient is engaged in functional activities, especially when cognitively distracted and or moving in a brisk fashion. The functional activities that trigger this response are dependent on the direction of the instability, which includes anterior (most common), posterior, inferior, or multidirectional. An example of an involuntary anterior instability can be observed in pitchers during the late cocking phase of the throwing

sequence. These individuals will experience the sharp pain and dead arm when the arm reaches the fully cocked position, just prior to acceleration. Other athletes that may be predisposed to this affliction include powerlifters, volleyball or waterpolo players, and swimmers.

Instabilities can be unidirectional or multidirectional. Shoulders can be unidirectionally unstable in either the anterior or posterior directions. Curl and Warren suggested the 'Circle Concept', where the compromise in the anterior and posterior capsulo-ligamentous structures is required for instability in either the anterior or posterior directions.[139] Anterior instability is frequently related to a lesion of the anterior-middle[140] and or anterior-inferior glenohumeral ligaments.[141] This predisposition may be related to increased mineralized fibrocartilage and disorganization of ligament fibers in those regions.[142] Posterior instabilities, which are uncommon and appear to be related to trauma or seizure activity, emerge as a consequence of failure in both the anterior and posterior capsular regions. On the other hand, multidirectional instabilities involve a symptomatic inferior subluxation or dislocation related to a redundant inferior capsular recess and failure in the anterior and posterior capsular regions.[122,143]

Patients suffering from instability present with several distinctive clinical characteristics. Historically, younger patients can develop instability due to laxity and or trauma, whereas the middle to older age population experience instability in concert with degeneration or trauma. Instability produces a clinical triad of: (1) deep, sharp shoulder pain in the C5 dermatome during sudden or unexpected movements, followed by a "dead arm"; (2) possible noncapsular pattern of limitation accompanied by excessive motion on another direction; and (3) a pathological end-feel during passive motion assessment (either excessively soft or firm, depending on joint congruency and function). This lesion can be accompanied by a painful click when the labrum is involved in the instability. Patients are commonly apprehensive to certain movements, especially with external rotation. The pain can be located in the anterior or posterior shoulder, due to either compression or tension loading of the labral and or capsuloligamentous structures. If produced while throwing, the patient may experience the pain during the cocking, acceleration or throwing phases.[144]

The examination of a patient suffering from glenohumeral instability may be misleading or completely negative, based on the elusive nature of the condition. Therefore, several special tests have been proposed for detection of a clinical instability at the shoulder. These tests can be categorized as either laxity tests or provocation tests, based on application and expected outcomes. Glenohumeral joint laxity can be detected through joint play testing in an anterior-posterior direction along the plane of the glenoid, while respecting the glenoid labrum (see Figure 12). The testing should be performed with mild humeral proximal loading ('load and shift') to increase translation.[111,145] In addition, the test can be performed in three shoulder pre-positions: (1) maximum loose packed position, with the shoulder positioned at 55° abduction in the scapular plane; (2) with the arm at the side, so to test the superior glenohumeral capsuloligamentous complex;[146] and (3) at 90° abduction, so to test the anterior inferior capsuloligamentous structures.[147]

A laxity test is considered positive when the involved side demonstrates increased translatory movement compared to the un-involved side. Hawkins has classified instability as: (1) Type I: where the humeral head demonstrates mild subluxation without perching over the labrum; (2) Type II, where the humeral head perches on top of the glenoid labrum when the joint is fully rotated; and (3) Type III, where the humeral head luxates, or dislocates, over the labrum.[148] Type III is potentially the easiest to diagnose, as the patient may present with a frank dislocation. However, the differences between Type I and II may be more unclear. Although a Type II may be visualized during stress imaging, clinical laxity tests may produce glenohumeral perching, providing the patient relaxes and does not exhibit muscle guarding.

While the previous tests indicate laxity in the glenohumeral joint, joint laxity does not necessarily equal instability. Therefore, a diagnosis of instability needs to include positive provocation testing. Recent investigations have proposed the relocation and modified relocation tests for clinical diagnosis of shoulder instability.[149,150] During the relocation test in supine, a patient suffering from anterior instability will demonstrate increased symptoms when the shoulder is positioned at 90° abduction and 90° external rotation. Consequently, these symptoms will decrease when the clinician exerts a passive posterior force onto the anterior humeral head. Conversely, a patient suffering from internal impingement may demonstrate increased symptoms when the clinician exerts the posterior force, making the test a useful tool for differentiating posterior impingement from instability.[95,151]

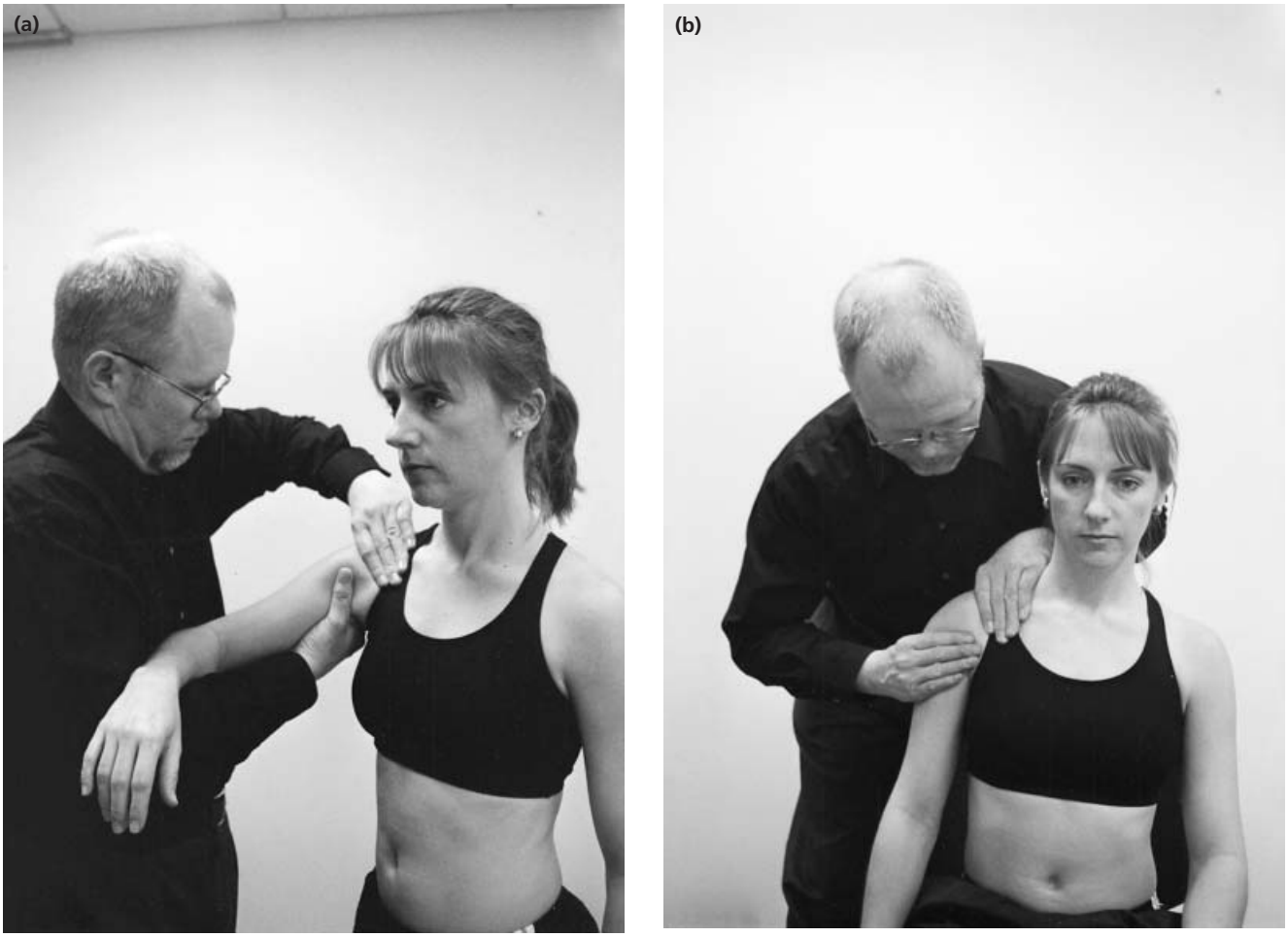

Figure 12. Glenohumeral joint laxity testing with the shoulder positioned in (a) maximum loose packed position, with the shoulder positioned at 55° abduction in the scapular plane, and (b) with the arm at the side, so to test the superior glenohumeral capsuloligamentous complex.

As previously mentioned, patients may suffer from labral lesions in context with glenohumeral instability. In the case of anterior-inferior instability, the patient may demonstrate a painful click during the previously mentioned tests. In addition, these patients will be resistant to conservative management, as the painful click will persist and symptoms will not rescind. Selected superior labral lesions can be detected through MR imaging, which is moderately sensitive and specific for as Type I, II, and III SLAP lesions.[12] Additionally, when the clinician suspects a SLAP lesion, he or she may choose to test the patient with the Crank test[153] and or active compression test of O'Brien.[154] The crank test, demonstrating 93% specificity and 91% sensitivity for SLAP lesions, produces a painful click with passive rotation of the shoulder in an elevated position and the scapula stabilized. For the active compression test, pain is produced when the clinician exerts a downward pressure to the patient's shoulder that is positioned in 90° flexion, slight horizontal adduction, and full internal rotation (see Figure 13). The same test will be negative when repeated while the shoulder is re-positioned in full external rotation. Finally, a patient with a Type II SLAP lesion and shoulder instability, will likely demonstrate a positive biceps load test. This test, which demonstrates 90.9 sensitivity and 96.9 specificity for the lesion,[131] produces pain during resisted elbow flexion when the forearm is fully supinated, the elbow is flexed to 90° and the shoulder is prepositioned in 90° abduction and full external rotation.[155,156]

Recently, clinicians have incorporated a variety of measures to reduce the increased glenohumeral translation associated with shoulder instability. Clinicians will first incorporate a nonoperative stabilization program

Figure 13. Active compression test for SLAP lesion, whereby the clinician exerts a downward pressure to the patient's shoulder that is positioned in (a) 90° flexion, slight horizontal adduction, and full internal rotation and (b) 90° flexion, slight horizontal adduction, and full external rotation.

prior to any invasive procedures. If instability is accompanied by dislocation, the rehabilitation program may be preceded by short-term immobilization. After reduction, this problem was traditionally immobilized in an internally rotated position, so to reduce stress on the anterior capsular structures. However, Itoi et al recently reported increased glenohumeral coaptation and reduced aphysiological translation when the glen humeral joint is immobilized in external rotation (mean = 35°).[157] They suggested that this reduction was related to pretension in the subscapularis, thus creating a tension-loaded restraint to anterior translation of the humeral head.

Kibler suggested that there is no evidence indicating the most efficacious rehabilitation protocol for shoulder instability.[21] Regardless, a comprehensive rehabilitation program is aimed at improving strength in the parascapular muscles, deltoid and rotator cuff, while reducing coordination disturbances and deficits in proprioception.[158] The program should be initiated with NSAIDs and education in pain-reducing strategies. Patients then progress to exercises involving the scapular stabilizers, especially the serratus anterior, latissimus dorsi and trapezius. The exercise program can be further advanced to include activation of the rotator cuff, with particular emphasis on the subscapularis. Initially, patients should avoid end-range external rotation, abduction, and horizontal abduction. Additionally, rotator cuff activation may be enhanced when exercises are performed in the closed chain[159] and with electromyographic biofeedback (EMGBF).[160] Moreover, conservative rehabilitation programs appear to be more effective when used with patients with a nontraumatic etiology versus those with a traumatic etiology.[161]

Nonsurgical rehabilitation has been proven to be beneficial to patients suffering from full thickness rotator cuff tears.[118] According to Brewster and Schwab, this program should initially focus on reducing inflammation with activity modification, local modality treatments, and posterior capsule stretching.[162] As inflammation begins to subside, the patient can engage in resisted exercise to shoulder internal and external rotation, as well as extension and scapular elevation and retraction. Later, resisted exercises can be incorporated which emphasize improving both strength and endurance of the shoulder complex. Finally, functional retraining is incorporated, including plyometrics, throwing, and or functional weightbearing. The success of a nonsurgical program on the rehabilitation of a rotator cuff tear appears to be improved by a significantly worse post-morbid functional state and a lack of either litigation or worker's comp status prior to implementation, a high level of patient motivation, and involvement of the dominant upper extremity.[163]

Conservative measures are not always successful in treating the sequelae of glenohumeral instability.[120] Numerous authors have reported more invasive procedures aimed at stabilizing the joint, including capsulorrhaphy techniques that serve to shrink the glenohumeral capsule. Heat-induced collagen denaturation can be triggered by thermal energy at radiofrequency temperatures between 65° and 80°C.[164,165] Medvecky et al suggested that radiofrequency energy creates ionic agitation and frictional heating within the capsular tissue.[110] This heat reduces heat-sensitive bonds in the collagen, leading to collagen fibril uncoiling and random transformation. The coils ultimately re-contract in a disorganized fashion, producing a capsular shrink-

age. This conformational change is accompanied by fibroblastic proliferation, fibrosis and connective tissue disorganization, which all contribute to tissue maturation in a shrunken state. Tissue strain and yield strain change, lending to increased tissue stiffness. As a consequence, aphysiological translation within the glenohumeral joint decreases, reducing the clinical consequence of instability.[164]

This process has been incorporated to tighten an intact capsule that has stretched after microtrauma or as an adjunct to surgical repair.[164] Levitz et al reported the use of electrothermal energy to internally shrink the glenohumeral capsules in the shoulders of baseball players who had previously failed conventional arthroscopic debridement and repair.[170] These authors found that the procedure reduced symptoms and re-injury associated with internal impingement. Wilk suggested that this procedure should be treated with two weeks of immobilization followed by progressive passive movement, limiting external rotation to 50° and abduction to 135° for four additional weeks.[167] Finally, clinicians are warned that thermal capsulorrhaphy can produce axillary nerve injury in response to heat in close proximity to the nerve.[168,169]

Laser assisted capsular shrinkage has been proposed as an alternative to the radiofrequency thermal procedure. Wilk suggested that these patients should be treated with two weeks of immobilization followed by progressive passive range, limiting external rotation to 15° and abduction to 90° in the scapular plane for four additional weeks.[167] Lyons et al reported that 96% of their patients' shoulders treated with laser-assisted capsulorrhaphy remained persistently stable and asymptomatic at 24 months post-procedure.[170] However, Levy et al reported a 36.1% failure rate in patients who were treated with laser shrinkage, versus a 23.7% failure rate in patients treated with radiofrequency thermal shrinkage.[72]

Surgical re-stabilization is aimed at balancing the capsular tension on all sides of the glenohumeral 'circle'.[158] In general, the procedures involve overlapping and securing redundancies in the capsule, so to promote scarring and subsequent capsular tightness. While these corrections can be made from any approach to the joint, anterior inferior capsular shift procedures appear to be more frequently implemented.[158,171,172] Anterior-inferior capsular shift can reduce anterior and posterior translation, thus reflecting the 'circle' concept and serving as a useful tool in the management of multidirectional instability.[139] Deutch et al evaluated the biomechanical differences between glenoid-based versus humeral-based anterior-inferior capsular shift. While no differences in post-operative anterior or inferior translation were observed, certain mechanical features appeared superior with the glenoid-based shift.[172] Finally, outcomes from arthroscopic restabilization with suture anchors may equal or surpass traditional open techniques in select cases.[173]

Partial thickness rotator cuff tears have been managed through arthroscopic debridment with or without acromioplasty.[115,119] Additionally, full thickness tears have been successfully repaired through both arthroscopic and open procedures.[115,174] Longitudinal analyses of such procedures have demonstrated persistent success between one and 10 years post-op.[175,176] While range of motion and muscle strength deficits may persist[176] functional outcomes appear to improve over time.[175] Repair outcomes appear to improve when suture anchors are incorporated and the superior glenohumeral capsule and or coracohumeral ligament are released at the time of the repair.[120,177]

In the event of a Bankhart lesion to the labrum, surgeons have incorporated a Bankhart reconstructive procedure to reattach the defect to the scapular limbus. While this procedure has proven to be an effective measure for restabilizing this shoulder lesion,[178] differences in post-surgical outcomes have been noted. First, Itoi et al. observed better post-operative external rotation when the surgeon performed a horizontal versus vertical incision through the joint capsule.[146] Second, an arthroscopic approach to the procedure, while equal to open procedure on most accounts, produced significantly greater external rotation in an abducted shoulder position, which could serve functional restoration in activities such as throwing.[179]

SUMMARY

Effective management of the painful shoulder is closely linked to a tissue-specific clinical examination. A patient's shoulder pain presentation, while commonly vague and referred in nature, can give the clinician clues regarding the joint responsible for the painful affliction. Painful shoulder conditions can present with or without limitations in passive and or active motion. Limits in passive motion can be classified as either capsular or noncapsular patterns, where capsular patterns are predictable, repeatable patterns of passive motion limitation that are distinctive for each joint and represent synovitis or arthrosis. Common noncapsular patterns

of the glenohumeral joint commonly accompany acute bursitis or impingement-related chronic bursitis and tendopathy.

Conversely, patients can present with shoulder pain that demonstrates no limitation of motion. Bursitis, tendopathy and rotator cuff tears can produce shoulder pain that is challenging to diagnose, especially when they are the consequence of underlying impingement and or instability. Furthermore, patients can develop shoulder pain as a consequence of nerve entrapment, lending to a more complicated clinical presentation. Numerous nonsurgical measures can be implemented in treating the painful shoulder, reserving surgical interventions for those patients who are resistant to conservative care.

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Appendix A: Clinical Examination of the Shoulder

Chief Complaint:

Cervical Quick Tests

Flexion	Extension
(R) Sidebending	(L) Sidebending
(R) Axial Rotation	(L) Axial Rotation

Shoulder Girdle Tests

Elevation	Depression
Retraction (15 sec hold)	Protraction

Scapulothoracic Junction (look for wing, tip or downward rotation)

- Static: Arms at Side (Neutral)
- Static: Arms actively elevated to 90° flexion with shoulder IR
- Static: Arms actively elevated to 90° abduction
- Dynamic: Active full arm elevation in flexion and abduction

Arm Elevation

- Test for painful arc: Active full arm elevation in flexion, abduction and scaption
- Passive elevation: With shoulder girdle fixation + posteromedial overpressure
- Passive elevation: With scapular fixation + posteromedial & posterior overpressure

Passive Tests

- Internal Rotation
- External Rotation
- Glenohumeral Abduction

Resistive Tests

Resisted Adduction	Resisted Abduction
Resisted Internal Rotation	Resisted External Rotation
Lift-Off Test	
Resisted Elbow Flexion	Resisted Elbow Extension

Extra Tests

"Pull Test" with Resisted Abduction, External Rotation, & Internal Rotation	
External Impingement Test	
Anterior (Subcoracoid) Impingement Test	
Posterior Internal Impingement Test	Suprascapular n. neural tension test
Modified Crank labrum test	Active Compression labrum test
Biceps stretch test	Inferior posterior capsule test

Instability Tests

- Anterior Laxity tests in MLPP, arm at side, and @ 90° Ab + full ER
- Inferior Laxity tests with arm at side and @ 90° Ab + full ER
- Posterior Laxity tests in MLPP, arm at side, and @ 90° Ab + full ER
- Relocation Test for anterior instability

Instructions: Please answer the following questions. Please note that the alternatives are alphabetically arranged for each question.

1. All of the following conditions typically produce vague, diffused symptoms, except for:
   a. Acromioclavicular instability
   b. Glenohumeral arthritis
   c. Sternoclavicular synovitis
   d. Subacromiodeltoid bursitis
2. All of the following indicate scapulothoracic instability when exhibited during the eccentric and or concentric phases of upper extremity elevation, except for:
   a. Scapular downward rotation
   b. Scapular tipping
   c. Scapular upward rotation
   d. Scapular winging
3. If resisted shoulder external rotation is the MOST painful procedure during the shoulder basic functional examination, which tendon insertion is most likely the pain generator?
   a. Supraspinatus
   b. Infraspinatus
   c. Subscapularis
   d. Deltoid
4. Which of the following patterns of glenohumeral limitation can be considered a capsular pattern?

	External Rotation Limitation	Abduction Limitation	Internal Rotation Limitation
a.	30 deg	80 deg	10 deg
b.	20 deg	40 deg	60 deg
c.	60 deg	40 deg	20 deg
d.	80 deg	20 deg	40 deg

5. You have diagnosed your patient with Idiopathic Adhesive Capsulitis (IAC). When you examined her passive external rotation, you noted that she experienced her C5 pain prior to reaching the end of her range of motion. Which stage of this disorder is she in?
   a. Freezing stage
   b. Frozen stage
   c. Melting stage
   d. Thawing stage
6. All of the following management strategies are recommended for a patient with Idiopathic Adhesive Capsulitis, except for:
   a. Arthroscopic release
   b. Hydraulic distention of the glenohumeral joint
   c. Intra-articular Injection
   d. Stretching and resistive ROM exercises
7. Which of the following noncapsular patterns of limitation most frequently accompanies subacromial impingement?
   a. Glenohumeral abduction limits
   b. Glenohumeral adduction limits
   c. Glenohumeral external rotation limits
   d. Glenohumeral internal rotation limits
8. Your patient is a 38-year-old male who plays in a weekend volleyball league on a regular basis. He has developed posterior shoulder pain that is aching in nature and increases with increased slamming of the ball over the net. You have noticed that his symptoms are provoked when you passively internally rotate and adduct his arm behind his back, followed by passive cervical sidebending to the contralateral side. Which disorder do you suspect?
   a. Acromioclavicular arthritis
   b. Bennett's lesion
   c. Posterior glenohumeral labral tear
   d. Suprascapular nerve entrapment
9. Your patient is a 42-year-old male with pain in the region of the deltoid that began when he started to build a fence in his back yard 6 weeks ago. Now his pain is sharp, followed by a dull aching and increases when he elevates and lowers his arm during activity. He demonstrates a midrange painful arc when he elevates his arm. You are able to provoke his symptoms with resisted shoulder abduction. However, the same test is negative when you perform it while pulling on his humerus along its long axis. How will you diagnose your patient?
   a. External impingement with subacromiodeltoid bursitis
   b. External impingement with supraspinatus tendinitis
   c. Internal impingement with infraspinatus tendinitis
   d. Internal impingement with supraspinatus tendinitis
10. Your 27 y.o. female patient presents in the clinic with glenohumeral instability. Upon examining her imaging, you noted that she demonstrates a dent in the posterior humeral head. How would this dent be classified?
    a. Bankhart Lesion
    b. Bennett's lesion
    c. Gray's Lesion
    d. Hill Sach's lesion
11. Your patient suffers from a detachment of the superior glenohumeral labrum and biceps tendon from the glenoid fossa after a traumatic, forced eccentric load to his biceps while lifting. What lesion does he likely suffer from?
    a. SLAP Type 1
    b. SLAP Type 2
    c. SLAP Type 3
    d. SLAP Type 4
12. All of the following are clinical a part of the functional triad associated with clinical shoulder instability, except for:
    a. A pathological end-feel during passive motion assessment (either excessively soft or firm, depending on joint congruency and function)
    b. Deep, sharp shoulder pain in the C5 dermatome during sudden or unexpected movements, followed by a "dead arm"
    c. Dull aching shoulder pain at rest that increases without movement and decreases with movement.
    d. Possible non-capsular pattern of limitation accompanied by excessive motion on another direction
13. According to Hawkins, how would you classify a patient's instability if during the laxity test the humeral head perched on top of the glenoid labrum when the joint was in a fully rotated position?
    a. Type I
    b. Type II
    c. Type III
    d. Type IV

14. Which of the following tests positive for a SLAP lesion incorporates a downward pressure to the patient's shoulder that is positioned in 90° flexion, slight horizontal adduction, and full internal or external rotation?
    a. Active Compression Test
    b. Clunk Test
    c. Crank Test
    d. Relocation Test
15. You have applied an electrothermal capsulorhapphy technique to your patient's anterior glenohumeral instability. Please review the following physiological events associated with this procedure:
    I. Collagen fibril contraction
    II. Capsular ionic agitation and frictional heating
    III. Collagen fibril uncoiling and transformation
    IV. fibroblastic proliferation and connective tissue disorganization
    V. Reduction in collagen heat-sensitive bonds

Now answer the following question: In what order do the previously mentioned events occur in response to this treatment?
    a. I, II, III, IV, V
    b. II, I, IV, V, III
    c. II, V, III, I, IV
    d. III, V, I, V, IV

Answers:
1. a
2. c
3. b
4. c
5. a
6. d
7. d
8. d
9. a
10. d
11. b
12. c
13. b
14. a
15. c