Subacromial Impingement Syndrome: Biomechanics, Pathology, Diagnosis, and Treatment

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OVERVIEW

One third of all physician office visits for musculoskeletal pain are attributable to the shoulder.[1] Shoulder pain is reported by 7-34% of the population, and subacromial impingement syndrome (SIS) is the most common cause, accounting for nearly two-thirds of all shoulder pain diagnoses.[2] SIS is an umbrella term that encompasses a spectrum of shoulder pathologies, including: bursitis, rotator cuff tendinitis/tendinosis, and both partial and full-thickness rotator cuff tears.[3] Rotator cuff arthropathy alone is reported to affect 9.7% of persons aged 20 years and younger and increases to 62% of persons of 80 years and older.[4] SIS diagnosis is most prevalent within 40 to 60 years of age.[5]

Impingement occurs in the subacromial space which is defined inferiorly by the humeral head and superiorly by the coracoid process, the coracoacromial ligament, and antero-inferior acromion.[2,6] The typical distance between the acromion and humeral head in a healthy adult is 10-15 mm, and situated within the subacromial space are the bursa, rotator cuff tendons, and the long head of the biceps tendon.[6] Trauma, degeneration, or other disturbance to the relationship of these structures may lead to SIS.[2,6] Most often SIS refers to rotator cuff degeneration or mechanical impingement of the rotator cuff against the superior margin of the subacromial space. If left untreated, SIS advances to partial and full rotator cuff tendon ruptures.[2-4]

The glenohumeral joint is the most mobile joint in the human body, but this same characteristic also makes it the least stable joint. Stabilization at the glenohumeral joint is unique in that it relies not only on passive capsular and ligamentous structures but also on the active compression by the rotator cuff muscles.[7] Arm elevation occurs by a muscular force-couple mechanism: active inward and downward rotator cuff activation approximates the humeral head as the deltoid muscle force is directed upward and outward with respect to the humerus.[8] The supraspinatus both stabilizes and initiates arm elevation, and works synergistically with the more powerful deltoid throughout range of motion.[9] In a healthy shoulder, balanced moments between subscapularis and the combined forces of infraspinatus and teres minor compress the humerus into the glenoid as illustrated in Figure 1.[7,8]

Full overhead range of motion in shoulder flexion, scaption, and abduction also requires activity at the scapulothoracic joint. The scapula is the foundational base of support for coordinated, functional movements at the glenohumeral joint.[10] Roughly one third of full arm elevation occurs via upward rotation and elevation of the scapula.[11] These motions are accomplished synergistically through concentric actions of the peri-scapular muscles: trapezius muscles, serratus anterior, and levator scapula; and eccentrically by rhomboids major and minor.[10-12] The upper trapezius and serratus anterior upwardly rotate the scapula while the lower and middle trapezius and serratus anterior stabilize the scapula against the thorax (Figure 2). The upper trapezius and levator scapula are responsible for scapular elevation. The serratus anterior has the important task of posteriorly tipping the scapula, positioning it close to the thorax during overhead movement and preventing scapular winging (Figure 3).[12] Anterior tipping approximates the acromion and humeral head which decrease available subacromial space. As the humeral head rolls and translates, these movements of the scapula maintain optimal

congruency between the glenoid fossa and humeral head and preserves the length-tension ratio of the rotator cuff muscles (Figure 4).[11,13] Hence, normalized kinematics of the shoulder are dependent upon scapulohumeral rhythm, coordinated movement between the humerus and scapula. The reliance on muscular balance and optimal alignment throughout overhead arm range of motion makes the glenohumeral joint susceptible to pathomechanics and injury.

PATHOLOGY

There are various theories as to why shoulder SIS occurs, and the causes for SIS are often multifactorial and depend upon patient-specific characteristics such as age, movement habits, and individual anatomical morphology. Some sources distinguish between primary and secondary impingement. Primary causes of SIS are considered inflammation of the subacromial space, rotator cuff tendon degeneration, osteophytes under the acromioclavicular joint, type III hooked acromion, and glenohumeral instability; whereas, secondary causes may include abnormal glenohumeral and/or scapulothoracic arthrokinematics, thoracic kyphosis, muscle weakness/fatigue, muscular hypomobility, posterior capsule tightness, and adhesive capsulitis.[14] However, most investigators distinguish between extrinsic compression and intrinsic degeneration in SIS.[2,11] Extrinsic compression refers to inflammation and degeneration of the rotator cuff tendon(s) as a result of mechanical compression by some structure external to the tendinous cuff; whereas, intrinsic degeneration attributes SIS to degenerative processes related to tendon age, avascularity, tension overload, overuse, or trauma. The question is "which of these two major etiologies occurs first?"[11] Patients typically present with tendinopathy that is already in the presence of positive extrinsic factors such as subacromial osteophytes, muscle weakness/incoordination, and abnormal scapulohumeral rhythm, and so it is difficult to

determine which occurred first.[2,11,13] SIS is complex, and the clinician will likely need to consider both extrinsic and intrinsic factors.

Extrinsic Compression

The aforementioned causes, acromioclavicular morphology, poor postural/structural alignment, and pathomechanics, all give credence to the idea that extrinsic compression causes SIS. Neer's ideas pioneered much of the thinking about the extrinsic compression model, and he described three distinct stages of impingement: (1) a benign, self-limiting syndrome that involves acute bursitis with subacromial edema and hemorrhage; (2) repeated compression of the bursa causing it to lose its lubrication ability and the underlying rotator cuff is exposed to friction, fraying the underlying tendons, eliciting fibrosis; and (3) progression of partial to full-thickness rotator cuff tearing and development of antero-inferior acromial bony spurs.[8,14] Generally, these three stages are associated with specific age ranges: under 25 years of age, between 25 and 40 years, and over 40 years of age, respectively.[2] These stages should not be thought of as discrete phases but rather blend together along the spectrum of SIS severity.

External compression may occur against any structure within the superior margin of the subacromial space, yet the most commonly implicated structure is the acromion. There are three major variations of acromion shape (although some sources refer to a fourth type),[15] and they are classified as flat (type I), concave (type II), or hooked (type III), all illustrated in (Figure 5).[16] There is evidence to suggest that acromion morphology significantly influences the incidence of SIS with type III acromia increasing the chance of impingement.[2,15,16] Rotator cuff tears occur more frequently in individuals with type III acromia, and those tears tend to be larger.[15] However, there is debate as to whether acromion shape is congenital or a degenerative phenomenon. It is thought that over time tensile strain from the coracoacromial ligament may

cause thickening of the antero-inferior acromion, thereby changing its shape to type III.[2] Chambler reported acromial enthesophyte growth at the coracoacromial ligament's insertion in 15 consecutive patients undergoing modified open acromioplasty and rotator cuff repair (mean age = 62.2 ± 1.75 years).[17] It was reasoned that the stimulus for bone growth was twofold: (1) constant static tensile loading from the coracoacromial ligament; and (2) periods of increased dynamic tensile loading when the ligament is forced upwards during impinging movements. Hence, in shoulders with an existing rotator cuff tear, impinging motions provoke further inferior acromial bone deposition which escalates a viscous cycle of impingement, giving further credence to the notion that impingement affects acromial morphology.

Mechanical compression of the rotator cuff into the superior margin of the subacromial space can also be precipitated by abnormal scapulohumeral rhythm. Muscular insufficiency due to weakness, fatigue, or incoordination imbalances muscular force coupling, thus disrupting stability, alignment, and length-tension relationships.[10,13,18] For example, weakness of the rotator cuff (especially infraspinatus and subscapularis) will lead to excessive superior or anterior translations of the humeral head on the glenoid fossa since there is not a sufficient counteraction to the deltoid force.[18] Inadequate humeral external rotation (primarily infraspinatus weakness) also fosters SIS because if the arm is lifted while oriented with too much internal rotation, the greater tuberosity of the humerus will not clear the superior margin of the subacromial space.[18] Weakness of the glenohumeral external rotators may impact scapular kinematics as well. In an interesting study by Joshi et al, healthy volunteers followed protocols for fatiguing themselves in glenohumeral external rotation.[13] Thereafter, upon EMG testing, the researchers observed fatigue-induced alterations of the lower trapezius (4% decreased activation) and infraspinatus

(4% increased eccentric activation), highlighting the interdependence between the rotator cuff and scapular stabilizers.

Further consideration for scapular kinematics reveals that weakness of the lower trapezius and/or serratus anterior permits anterior tipping of the scapula which causes the acromion and humeral head to approximate.[18] Decreased serratus anterior activity is associated with increased upper trapezius activation.[18] Since the serratus anterior and upper trapezius normally co-contract to facilitate scapular upward rotation, the upper trapezius compensates for serratus anterior weakness. Unfortunately, the upper trapezius fiber orientation obligates the anterior pull/tilt of the scapula in the absence of a counter force along the medial border of the scapula.[12] Muscular incoordination or mistiming also promotes these aberrant kinematics. A delay in middle and lower trapezius activation has been observed in SIS patinets.[13] It is also worthwhile to note that in SIS, scapular tipping becomes more prominent with greater humeral elevation.[18]

Components of sagittal plane posture strongly influence scapular kinematics. Forward head posture and excessive thoracic kyphosis predispose individuals for SIS and are regularly observed in incidents of mechanical impingement.[5] These postures are associated with limited range of motion into thoracic extension, and excessive thoracic kyphosis essentially forces the scapula to maintain an anteriorly tipped orientation since the scapula must follow the contour of the thorax (Figure 6). Individuals who often position themselves in forward head posture and hyperkyphotic positions may develop adaptive shortening of certain soft tissue structures such as the pectoralis minor and the shoulder capsule.[19-21]

A short pectoralis minor will cause anterior tipping of the scapula and decrease the subacromial space.[19,21,22] This muscle is considered a prime antagonist to normal kinematics of

overhead motion since the fiber orientation promotes the following muscle actions: scapular internal rotation, downward rotation, and anterior tilt.[21,22] A shortened pectoralis minor creates excessive passive tension, resulting in forward shoulder posture, and contributes to a tight, shortened anterior capsule of the glenohumeral joint.[19,22]

Tightness of either the anterior or posterior capsule may lead to impingement. It has been described that antero-inferior capsular insufficiency promotes SIS-like symptoms.[20] Abduction and external rotation quickly take the slack out of the anterior band and the tension pushes the humeral head into the suprahumeral space.[20] Furthermore, an insufficient posterior capsule will translate the humeral head anteriorly and/or superiorly with arm elevation.[31]

Intrinsic Degeneration

While it is convenient to think of each of the rotator cuff muscles as having well-defined insertions, in reality there is no clear insertional footprint because the whole area serves as an insertion zone for all four muscles as well as the joint capsule.[23] The tendons interdigitate and blend together, forming a common, continuous insertion onto the humerus.[24] In SIS the supraspinatus portion of the tendinous duff is the most vulnerable to degeneration. In advanced tendinopathy of the rotator cuff, tearing advances in an anterior to posterior direction. If the supraspinatus is completely ruptured then the adjacent interwoven fibers of infraspinatus are vulnerable. Should the infraspinatus rupture completely, then the teres minor is next in line for injury.

Nowadays, more emphasis is being placed on the degeneration of the rotator cuff tendons.[25] There is evidence that the rotator cuff tendons themselves are the cause for SIS due to intrinsic factors which include tendon age, avascularity, tension overload, overuse, and trauma;

that rotator cuff deterioration causes the muscular weakness that manifests as altered shoulder mechanics. After all, older individuals with SIS are known to experience tendinopathy in the absence of degenerative changes to the acromion.[2] Tendon has been described as having a constant ultimate strength throughout the first six decades of life, but there is a rapid decline in the seventh decade.[26] Given the age demographics for SIS prevalence this biomechanical change could help explain SIS in some older individuals who do not present with acromial degeneration. On the other hand, most SIS cases are reported in individuals 40-60 years of age, and this age range is still susceptible to slow healing due to hypovascularity, tension overload, overuse, and trauma.

It is difficult for hypovascular tissues to heal. Blood to the rotator cuff is supplied by up to six arteries. Three of them (suprascapular and both anterior and posterior humeral circumflex arteries) are common to most people, but the other three (thoracoacromial, suprahumeral, and subscapular arteries) are sometimes absent.[8] Supraspinatus receives its blood supply from the thoracoacromial artery which anastomoses with the anterior and posterior circumflex arteries; however, since that artery is sometimes absent, supraspinatus, and to a lesser extent infraspinatus, are often hypovascular compared to the rest of the tendinous cuff.[8] Supraspinatus may be particularly susceptible to injury since tendon injury is often reported at sites of poor blood supply.[26] It is also reasonable to consider that biomechanically, the supraspinatus tendon accumulates years of tensile stress, compressive stress, and frictional abrasion which leads to a "wringing out" effect of the shoulder vascularity.[27] In other words, hypovascularity may be brought about as a natural adaptive consequence of arm use. Ischemia heralds intrinsic tendon degeneration.[28] The avascular area of the supraspinatus (and infraspinatus) is so significantly associated with rotator cuff tearing that it is dubbed the "critical zone."

DIAGNOSIS

History and Differential Diagnosis

Early recognition and management of SIS is important to prevent or limit pain, reduce activity, and avoid subsequent partial or complete rotator cuff tears. Long duration of symptoms prior to treatment (>3 months) is associated with poorer outcomes.[25] Accurate diagnosis of SIS is achieved through a thorough evaluation that includes history taking, systems review, and clinical examination. SIS pain will present as either acute (traumatic event) or more often with an insidious onset. It is typically reported as persistent anterolateral arm pain, and aggravating activities may consist of lying on the affected shoulder, stretching, and reaching for overhead objects.[2] The patient with SIS is also likely to report shoulder pain at night which may interfere with sleep hygiene.[3] Risk factors for SIS include:

- Overhead athletes and persons whose occupations require overhead work[13]
- Age of 40+ years (tendinopathy)[5]
- Age of 65+ years (intrinsic degeneration)[4,5]
- Forward head posture[5,19-22]
- Excessive thoracic kyphosis[5,19-22]
- Type III, hooked acromion[2,15,16]
- Weak/imbalanced shoulder muscles[12,13,18]
- Scapular dyskinesia[12,13,18]
- Unfavorable psychosocial status (low perception of control, low social support, low job satisfaction, and high pressure to perform)[25]

Of course, it is critical early on in the evaluation that neurological etiologies are ruled out and determined that the patient in fact "belongs" to PT and is not in need of urgent medical attention.[29] Differential diagnosis is broad and may include acromioclavicular joint injury, bicipital tendonitis, brachial plexus injury, cervical disc injuries, cervical discogenic pain syndrome, cervical radiculopathy, cervical spine sprain/strain injuries, claviclular fractures, contusions, myofascial pain, osteoarthritis, shoulder dislocation, SLAP lesions, suprascapular neuropathy, thoracic disc injuries, thoracic discogenic pain syndrome, and thoracic outlet syndrome.[1,3,8,30]

The clinician should also distinguish between SIS and internal impingement of the shoulder. Note that a thorough consideration of this form of impingement is beyond the scope of this paper. In short, internal impingement is another form of shoulder impingement which may have some similar symptoms. Internal impingement is less common and believed to occur most often in overhead throwing athletes and swimmers all of whom require extensive glenohumeral external rotation, but it may certainly present in other populations.[31] Generally, internal impingement is associated with posterior shoulder pain (especially during late cocking phase of throwing with end range glenohumeral external rotation and 90° abduction),[31] anterior glenohumeral instability,[32] tight posterior glenohumeral capsule,[33] and scapular dyskinesia.[31-33] The postero-inferior aspect of the labrum impinges on the underside of the rotator cuff, and in isolation this is not a pathological process, but prolonged frequency of internal impingement will damage the articular surface of the rotator cuff and the labrum.[2] Optimal treatment for internal impingement will diverge from SIS treatment.

Clinical Examination

The patient will likely exhibit normal or close to normal passive range of motion of the affected shoulder.[2] As many as 80% of SIS patients will experience shoulder pain upon passive abduction.[3] SIS patients generally demonstrate less passive internal rotation and passive posterior shoulder range in the painful shoulder compared to the unaffected shoulder.[5] Full active range of motion may be limited due to pain, and a positive finding for the painful arc between 60° and 120° of glenohumeral abduction may be indicative of SIS.[2] Manual muscle testing will vary depending upon the severity of the tendinopathy associated with the SIS, but the clinician is likely to note weakness in resisted external rotation. Scapular dyskinesis is a likely finding, but physical assessment of the scapula has not been shown to be a reliable means for differentially diagnosing shoulder pathology.[34] Scapular dyskinesis is found in both painful and non-painful shoulders.[34]

There are numerous special tests that have been developed for ruling in or ruling out subacromial impingement. Impingement tests are designed to reproduce symptoms or pain by compressing the greater tuberosity against the superior margin of the subacromial space. No single test is accurate enough to diagnose SIS, but using a combination of special tests increases the post-test reliability of diagnosis.

Michener et al examined the reliability and diagnostic accuracy of five such tests: Hawkins-Kennedy, Neer, painful arc, empty can (Jobe test), and external rotation resistance.[1] Conveniently, 2 of 5 of the above tests should already be part of the initial musculoskeletal examination (painful arc and resisted external rotation), and thus these special tests offer an efficient means of diagnosis. The tests have been found to have fair to substantial strength of interrater reliability as described in Table 1. The investigators found that positive findings on at

least 3 of the 5 tests have good diagnostic accuracy: sensitivity = 95% CI, 0.75 (0.54–0.96); specificity = 95% CI 0.74 (0.61–0.88), +LR = 95% CI 2.93 (1.60–5.36); and -LR = 95% CI 0.34 (0.14–0.80).[1] Individual sensitivity, specificity, and likelihood ratios for each of the five tests is listed in Table 2.

Brief summaries of the tests are based upon descriptions by Magee and are as follows:[14]

- For the Hawkins-Kennedy test (Figure 7) the patient stands as the clinician flexes the arm to 90° and brings the arm to 10-20° of horizontal abduction. The arm is then passively internally rotated. A positive finding is pain provocation at the shoulder. Anecdotally, if there is no positive finding in the positioning described, then adaptations to this test include testing at 0° and then 20° of horizontal adduction, and it is thought that pain provocation in more abducted positioning relates to greater severity of supraspinatus injury. The test can alternatively be performed by abducting the arm to 90° and then horizontally adducting the glenohumeral joint with concurrent maximal internal rotation to capture all possible combinations of horizontal adduction.
- For the Neer test (Figure 8) with one hand the clinician passively and forcibly elevates the patient's upper extremity in the scapular plane with the arm in medial rotation. During this test it is important that the clinician use his other hand to stabilize the ipsilateral scapula to prevent scapular elevation since this is a provocative test used to elicit familiar symptoms. In this position the greater tubercle of the humerus is pressed against the anteroinferior acromion. A positive test results in shoulder pain, indicating either supraspinatus or long head biceps tendon irritation.

- For painful arc test (Figure 9) the patient actively abducts his/her arm, and increased pain between 60-120° indicates a positive finding.
- The empty can test (Figure 10) entails having the patient actively abduct his arm to 90° in the scapular plane and then maximally internally rotate the arm (like he is pouring out the contents from a 12 oz can). The therapist then asks the patient to maintain the position while inferiorly directed manual force is applied. Pain or weakness indicates a positive finding. Modifications of this test have entailed first having the arm in 30° of elevation with maximal internal rotation to see if symptoms are provoked before bringing the patient into a position of maximum impingement. If familiar impingement symptoms are not provoked, then the test may be repeated by bringing the arm up higher between 30-90°.
- Finally, for the external rotation resistance test (Figure 11) while standing the patient flexes his neutrally aligned forearm to 90°. The clinician asks the patient to maintain the position as force is applied to the patient's distal forearm, prompting the patient to apply a glenohumeral external rotation moment. Pain or weakness indicates a positive finding.

The Dutch Orthopaedic Association recommends that positive findings for three specific tests from up above are sufficient for SIS diagnosis: Hawkins-Kennedy, painful arc, and infraspinatus (external rotation) tests.[25] Park et al corroborate that collectively these same three tests are effective in diagnosing SIS.[12] Additional tests for detection of tendinopathy in SIS may include the Drop Arm test, the Lift-Off test, and Yocum's test:

- The Drop Arm test (Figure 12) is performed by having the therapist abduct the patient's arm to 90° and then asking him to slowly lower his arm. A positive finding is indicated if

the patient is unable to control the eccentric motion or severe pain is reported in attempting to lower the arm. A positive Drop Arm test suggests supraspinatus involvement.

- To initiate the Lift-Off test (Figure 13) the patient internally rotates his arm, flexes the arm to 90°, and places the dorsum of the hand at the contralateral low back. The patient is then instructed to lift his hand away from the low back. Pain or an inability to lift the hand 5-10 cm without extending the elbow indicates a subscapularis lesion. (Sensitivity = 68%; Specificity = 50%)[3]
- To perform the Yocum's test (Figure 14), the patient is instructed to place his hand (from the same side as the affected shoulder) on the contralateral shoulder. The clinician then flexes the patient's arm to 90°. Pain indicates a positive test and a supraspinatus or long head of the biceps tendon lesion.

The clinician should be aware that positive findings for rotator cuff tendinopathy may not necessarily infer SIS since shoulder tendinopathy can occur in the absence of SIS. SIS and tendinopathy often go hand in hand, but are not necessarily counterparts.

In a 2018 publication Ferenczi et al proposed a new 2-part test for detecting SIS in patients with degenerative rotator cuff disorders, the counter test with elevation in lateral rotation (CELR) and is shown in Figure 15. The test is similar to the empty can test. For the first part of this test the arm is set in medial rotation and passively raised in the anterolateral scapular plane. If pain is elicited, then the second part of the test is performed. The clinician sets the arm in lateral rotation and then again raises the arm in the antero-scapular plane. The test is considered positive if there is no pain during the second half of the test. In this nascent consideration of CELR, the investigators found that this test had moderate reproducibility suitable for clinical

practice, similar to the Hawkins test, but only detected 41-44% of ultrasound-confirmed cases of SIS.[35] The CELR test needs further investigation.

Imaging

Numerous authors recommend routine radiography in SIS diagnosis.[2,30] Radiography has been determined to be a reasonable diagnostic tool in the painful shoulder to rule out other pathologies as it can help to determine the presence/absence of osteoarthritis, osseous abnormalities, acromion morphology, os acrominale, and calcium deposits.[2,25] It has been suggested that at a minimum an anteroposterior view, an axillary view, and a scapular outlet view should be taken, but in some cases 30° caudal tilt and 15° cephalad tilt views should be taken to rule out bone spurs.[2] Radiograph images are useful in determining mechanical impingement and discerning the distance between the humeral head and acromion.[30] Generally, additional imaging is reserved unless there is suspicion of "repairable" shoulder pathologies such as rotator cuff tearing, labral tearing, or operable instability.[2]

Ultrasonography is recommended when conservative intervention fails.[25] It is a sensitive and specific method for detecting or excluding rotator cuff tendinopathy, subacromial bursitis, biceps tendon rupture, and tendinosis calcarea.[25] The diagnostic accuracy may be as high as an MRI, but ultrasound is highly dependent upon the expertise of the operator.[2,25] Ultrasonography has the distinct advantage over MRI in that it is much more cost-effective.

Considered the gold standard in diagnosing many musculoskeletal disorders, MRI is routinely prescribed in the US for suspected SIS, and it consistently displays a specificity and sensitivity of approximately 90% across numerous investigations, allowing for visualization of bursitis and partial/full thickness rotator cuff tears.[2,3] However, MRI results should be interpreted

with caution since a significant number of individuals have been shown to have rotator cuff tears despite pain-free, asymptomatic shoulders. It is likely that MRI is overprescribed in the US. Considering the high specificity and sensitivity that the physical exam exhibits for identifying SIS and high cost of MRI, it should be reserved for patients who fail to respond to conservative treatment and for those contemplating surgical intervention.

INTERVENTION

Treatment outcomes for SIS depend upon a variety of factors including age, activity level, extent of tendinous degeneration, patient expectations, and general health. The general goals of treatment are to reduce patient pain and improve function. Conservative treatment is widely regarded as the first line of treatment and should entail some combination of relative rest, reduction of aggravating activities (especially overhead activities), NSAIDs, and physical therapy management. There is a lack of compelling evidence that surgical intervention is more effective than conservative treatment for pain management and functional restoration for most instances of SIS.[25] When one considers these reasons, plus the cost and risks associated with surgery, as a general guideline surgical intervention should be pursued only if the patient does not respond to exhaustive non-operative treatment.

In addition to using VAS pain and function scales, there are a number of outcome measures used to assess patient status and effectiveness of treatment in SIS. By in large, these measures rely on patient self-reports and have moderate to strong psychometric properties in assessing SIS. A full description of each of these measures is beyond the scope of this paper, but

for the sake of convenience and completeness here is a list of validated measures frequently used in both operative and non-operative treatments of SIS:

- Constant Shoulder Score (CSS)[36]
- Disabilities of the Arm, Shoulder, and Hand (DASH)[37]
- Oxford Shoulder Score (OSS)[38]
- Rotator Cuff Quality of Life (RC-QOL)[39]
- Shoulder Pain and Disability Index (SPADI)[5,35,38,40]
- Simple Shoulder Test (SST)[40]
- UCLA Shoulder Test[40]
- University of Pennsylvania Shoulder Score (PSS)[41]
- Western Ontario Rotator Cuff Index (WORC)[36,42,43]

Corticosteroid Injections

Corticosteroid treatment for SIS is controversial. It has been shown to be more effective than placebo injections, physical therapy, or no treatment in reducing pain and improving shoulder function within the first 8 weeks of diagnosis, but these injections are no more effective at reducing pain than NSAIDs.[25] Numerous investigations have determined that NSAIDs and corticosteroids produced equivalent outcomes in pain management.[30] There are certainly cases in which corticosteroid injection with physical therapy have resolved signs and symptoms of SIS.[2] Yet the outcomes may be mixed. Hart determined that corticosteroid injections were beneficial (but similar to placebo) at producing short term pain relief of tendinopathy but that long-term deleterious consequences were likely.[44] Adverse side effects were observed in as many as 82% of corticosteroid injections.

In the author's opinion, patients should be cautioned against corticosteroid injection as an initial treatment due to the catabolic effects of corticosteroids. Tendon rupture is associated with physical activity following corticosteroid injection.[26] Furthermore, corticosteroid injection in the absence of physical therapy does nothing to address the pathomechanics involved in SIS. Thus, the patient may resume full activities in a pain-free manner that actually promote impingement since there will be no noxious deterrent. Such patient behavior could exacerbate the degenerative changes associated with SIS and lead to partial/full-thickness rotator cuff rupture.

Orthobiologics

There is growing research concerning the use of orthobiologic agents such as platelet-rich plasma (PRP) for the management of rotator cuff tendinopathy associated with SIS.[45] Orthobiologic agents address the intrinsic degeneration component of SIS. PRP derived from autologous blood samples are injected into the tendon and thought to promote healing by adding growth factors to the injection site. PRP injections enhance tenocyte proliferation and collagen production through processes of angiogenesis, epithelialization, cell differentiation, proliferation, and the formation of extracellular matrix and fibrovascular callus.[46]

A recent study compared PRP and corticosteroid injections.[47] At 6 and 12 weeks follow-ups the PRP group displayed more favorable outcomes that the corticosteroid group, but at 6 months follow-up there was no significant difference between the groups. These results suggest that PRP injections may be a better alternative, especially for persons for whom corticosteroids are contraindicated.

PRP has also been compared to exercise therapy in SIS. Both groups showed significant improvements, but early on (1 and 3 months) the exercise group showed greater improvements in pain, shoulder flexion and abduction, and functionality.

Stem cell therapy is another orthobiologic treatment that is gaining research and clinical interest. Stem cell therapy is a rapidly evolving facet of health science literature but very few trials have been conducted in humans and none have been conducted on human rotator cuff tendons.[46] Still, stem cell research appears promising and may be a direction for future treatments.

Surgical Intervention

While there are no hard guidelines for selecting surgical repair for SIS, it has been suggested that for surgery to be indicated one of two criteria must be met: failure of non-operative management or presence of a full-thickness rotator cuff tear.[2] The 2013 Neer Award recognized Dunn et al for their contributions for understanding failure of non-operative management of SIS.[48] The most important finding was that a patient having low expectations for physical therapy was the strongest determinant for selecting surgery. Expectations were significantly more predictive of surgical intervention compared to VAS pain scores and rotator cuff tear size. They also found that non-smokers and individuals who engage in higher levels of activity are more likely to select surgery. Patients who opt for surgical intervention often do so early in the intervention process. Other salient considerations for opting for surgery are younger age and higher levels of activity.[48] It is thought that persons who engage at higher levels of activity are less likely to observe relative rest prescriptions and will continue work or sport behaviors that promote impingement, thus undermining non-operative intervention.

Prior to surgery, MRI imaging and/or arthroscopy are common procedures used to inform the orthopedist and patient.[30] The exact nature of the repair depends on the precise location of the lesion and acromial morphology. Generally speaking, the goal of surgical management is to reestablish a strong tendon-bone integration and restore normal biomechanics to the joint.[49] There are two main types of surgery in SIS: open surgery and arthroscopic procedures.[2] Open anterior acromioplasty with concurrent rotator repair (if necessary) was the surgery pioneered by Charles Neer.[50] A retrospective investigation (n=32) revealed that at mean follow up of 25 years (range 21 to 27 years) for patients who received this surgery, 72% reported slight or no pain and 88% expressed satisfaction with the operation.[51] Overall, these numbers are favorable. However, 15.6% of patients still required a follow-up surgery. An additional drawback to open acromioplasty is that it requires a longer post-op hospital stay.[52]

In recent decades, open surgery has been replaced by less invasive techniques involving arthroscopy. Arthroscopy has the advantage of avoiding the risks of open surgery while still allowing the surgeon to inspect the inter-articular surfaces, long head of the biceps tendon, the bursa, and subacromial margin.[2,53] A typical lesion may look like scuffing of the acromion and fraying of the rotator cuff tendon(s) (Figure 16). The major arthroscopic surgical management in SIS includes subacromial decompression that may include rotator cuff repair, acromioplasty, or debridement without acromioplasty.[2,52] Subacromial decompression is a procedure that relieves accumulated pressure within the subacromial space. This is often accomplished through a bursectomy, removal of fibrotic tissue, and the antero-inferior aspect of the acromion may be sanded flat (acromioplasty). Subacromial decompression allows for increases in the available joint space for the rotator cuff tendons, decreasing the likelihood of impingement. The outcomes

for the procedure are generally favorable but about 10% of patients continue to report pain postoperatively.[2]

Recent research has questioned the necessity for arthroscopic acromioplasty. For example, a double-blind RCT (n=56) compared bursectomy with acromioplasty to bursectomy alone and at 12 year follow-up found no difference between the groups for all outcome measures (Constant score, Simple Shoulder Test score, and two VAS scores: pain and function), calling into question the effectiveness of acromioplasty.[54] Bursectomy alone may be sufficient to reduce SIS pain.

Surgical repair failure rates vary widely, depend upon reporting source, and are attributable to numerous factors, but some estimates put failure rates as high as 20-70%.[46] It must be underscored that in most cases, surgery should be reserved as a last resort and used if the patient fails to respond to conservative intervention. Fewer than 5 % of all patients with rotator cuff tears and SIS undergo surgery in the US, but this still accounts for 75,000 to 250,000 surgical repairs annually.[48]

Physical Therapy

Physical therapy is a crucial component to both non-operative and operative management of SIS, and indeed the majority of SIS cases are successfully managed through conservative interventions that include physical therapy. A prospective study of 103 rotator cuff tears treated non-operatively showed continued pain relief at 13 years' follow-up, and 72% of patients reported no problems with activities of daily living.[48]

There are numerous therapeutic options available for physical therapists to use in SIS rehabilitation and may include therapeutic exercise, joint mobilizations, extracorporeal shock

wave therapy, hot/cold therapy, taping, ultrasound, transverse friction massage, and manual therapy.[55,56] Many of the aforementioned modalities are best suited for controlling pain and advancing tendon healing, but retraining proper arthrokinematics and strengthening through exercise therapy is the hallmark treatment in SIS.[18,30,44] Hence, active patient participation is an essential ingredient in successful rehabilitation.

Patient Education

It could be argued that the single most critical aspect of initiating an exercise therapy in SIS is establishing patient "buy-in" early on in the rehabilitation process.[57] Ultimately, long-term self-management is the key to success. Conservative treatment of SIS requires a long game approach, and it is the patient who must be diligent about posture, regularly perform rehabilitation exercises as instructed, and self-monitor participation in physical activities that could have implications for the shoulder. Patient expectations and activity levels are the greatest predictors of outcomes in physical therapy management of SIS.[48] Hence, it is critically important to get patient "buy-in" early in the rehabilitation process. Patient's baseline status for tendon tear size, poor shoulder kinematics, pain, or weakness are far less important predictors.[48] Good outcomes are likely if the patient puts in the effort. Physical therapy programs have been shown to be highly effective in alleviating patient symptoms despite some patients continuing to have tears of the rotator cuff.[48] The patient should be educated on these points as reassurance to trust in the process of rehabilitation. It has been suggested that being able to produce immediate pain relief will increase SIS patient "buy-in".[57]

Manual Therapy

Manual therapy may be a good starting point. Thoracic manipulation has been shown to have statistically significant effects on reducing pain in SIS at 48 hours after administration.[58] Posterior to anterior directed thoracic manipulation is thought to reduce pain through neurophysiologic mechanisms. Glenohumeral mobilizations have also been found to have a significant effect in decreasing SIS pain at 24 hours.[59] Conroy and Hayes describe using posterior, anterior, and inferior glides as part of a comprehensive treatment protocol that demonstrated better initial outcomes on pain than without the mobilizations. It should be noted that glenohumeral mobilizations are only indicated in the hypomobile shoulder. Should the shoulder demonstrate instability such as a positive sulcus sign or if the patient is positive for the Beighton hypermobility index, then joint mobilizations should be avoided.[60]

Patients with chronic SIS may develop pain central sensitization that causes them to experience pressure pain hyperalgesia and hypersensitivity to muscles within the C4-C6 nerve distribution: the levator scapulae, supraspinatus, infraspinatus, subscapularis, biceps brachii, pectoralis major, and tibialis anterior (TA is not within the C4-C6 nerve distribution but TA hypersensitivity is found to have high correlation with SIS).[58,61] Moreover, these same muscles (and others) may contain trigger points. Manual treatment of these trigger points in SIS reduces spontaneous pain and pressure hypersensitivity and induces segmental anti-nociceptive effects.[58] Six treatment sessions of manual physical therapy have been found to be just as effective as one to three 40 mg of triamcinolone acetonide (corticosteroid) at reducing SIS pain even at one year follow up.[62] These manual therapy techniques may help to establish patient trust and confidence in non-operative SIS management.

Exercise Therapy

Exercise is the centerpiece of a comprehensive conservative rehabilitation program for the SIS patient.[57] The goals of exercise therapy are to increase pain-free range of motion, improve muscular strength, enhance the integrity of the rotator cuff tendons, restore proper scapulohumeral kinematics, and optimize function. Exercise improves patient outcomes through a variety of mechanisms. Therapeutic exercise is thought to relieve pain, reduce muscle spasm, and reverse abnormal force-couple imbalances which will restore pain-free ROM, and eventually improve function.[57] Exercise promotes tendon healing by increasing metabolic turnover of tendon.[26] Reducing thoracic kyphosis has been shown to increase the range of shoulder flexion and scapular plane abduction in those with SIS.[5] Flexibility and AROM exercises can be used to lengthen the pectoralis minor and improve thoracic kyphosis and forward head posture. Despite widespread agreement that exercise is an invaluable aspect of treatment there is insufficient evidence to support any one particular exercise protocol (type, intensity, frequency, and duration).[57,63] However, there is endorsement of strategies throughout the literature.

Herein, guidelines for consideration of exercise selection and progression will be presented. These guidelines are presented as recommendations rather than absolute requirements. Physical therapy rehabilitation for SIS can be considered in four stages. Much of this characterization is based upon the SUPPORT trial:[56]

- Stage I: acute phase rehabilitation with a focus on reducing pain and initial neuromuscular reeducation. This entails scapular stabilization exercises and active movement with no external loading, addressing scapulothoracic arthrokinematics. Scapular stabilization retraining progresses from prone to seated to standing and initially

involves short lever arms (flexed elbow) and later a longer lever arm (extended elbow) for additional resistance.

- Stage II: subacute phase, progressing ROM and strengthening program. The clinician should direct ROM exercises, isometric strengthening, and emphasis on developing pain-free motion. Herein, cardinal planes of motion are directed (such as flexion, abduction, and external/internal rotation) while maintaining scapular stability and normalized scapulothoracic arthrokinematics. Progressions include resistance bands or manually directed resistance (or self-directed resistance).
- Stage III: progressive strength training with emphasis on proper mechanics.
- Stage IV: return to activity with unrestricted, symptom-free movement.

ROM

Range of motion (ROM) assessment should direct ROM treatment. Many patients with SIS demonstrate reduced internal rotation which fosters posterior capsule tightness and subsequent anterior and superior translation of the humeral head.[60] This is common in overhead throwing athletes. Ellenbecker et al recommended passive (manual) supine internal rotation stretching with the arm placed in the scapular plane. The scapula should be stabilized so as to prevent compensation.

Akkaya et al found that both weighted and unweighted pendulum exercises were effective in increasing ROM and decreasing pain via distraction during the oscillations.[64]

Pain-free ROM should be emphasized in all planes so as to prevent secondary complications such as excessive fear avoidance behavior and adhesive capsulitis.[57] ROM exercises may also include glenohumeral external rotation, medial rotation and abduction to

approximately 90°. These exercises are progressed from gravity neutral and "stick-assisted" to ROM against gravity.[65]

Pectoralis Minor

Since adaptive shortening of pectoralis minor is thought to influence posture and alter scapular kinematics that determine SIS, it follows that this muscle should be emphasized as part of a stretching routine when the patient presents with characteristic forward shoulder posture.[22] Borstad and Ludewig compared three stretching techniques of the pectoralis minor: the unilateral corner stretch, the seated manual stretch, and the supine manual stretch. They found that the unilateral self-stretch accomplished the most elongation of the pectoralis minor muscle (nearly three times the lengthening compared to the unilateral sitting manual stretch), followed by a unilateral supine manual stretch.[66] The manual supine stretch is likely a suitable initial option for the painful, acute shoulder and more aggressive passive stretching through the unilateral corner stretch is more appropriate with reduced levels of painful inflammation.

Rosa et al assigned a 6-week daily protocol of 4 repetitions of 1-minute stretches with a 30-second interval between repetitions, and found that it was effective at reducing pain and improving shoulder function in SIS.[22] Surprisingly, the investigators did not observe an increase in resting pectoralis minor length or significant effects on scapular kinematics. They postulated that because the stretching was unsupervised, the participants may not have sufficiently stretched at end range or did not fully comply with the protocols.

Scapular Stabilization

Patients with SIS may demonstrate scapular neuromuscular incoordination, muscular weakness, or both. It has been suggested that for patients who demonstrate neuromuscular

incoordination, a 4 week program that emphasizes pain reduction and scapular retraining is best practice in SIS.[60] In early scapular training, the patient should be taught conscious muscle control of their scapulae to enhance proprioception and normalize scapular resting position.[60] Once scapular orientation is complete the patient may progress to dynamic and isometric exercises including "low row", "inferior glide", "lawnmower", and "robbery" exercises.[56,60,67] Exercises that retrain the serratus anterior and lower/middle trapezius while minimizing upper trapezius activity will enhance normalized kinematics about the scapula, promoting normalized upward rotation and posterior tilt and normalized force coupling.[60,67]

Rotator Cuff Strengthening

SIS patients consistently show strength imbalance between internal and external rotation at the glenohumeral joint.[56,60] Electromyographic investigations in SIS reveal weakness of supraspinatus and infraspinatus, and isokinetic dynamometry shows that individuals with SIS are only 60-70% as strong in glenohumeral external rotation compared to internal rotation.[60] Once the patient's acute pain has resolved there are numerous unilateral exercises that emphasize supraspinatus strengthening and glenohumeral external rotation such as full can, side lying external rotation, and tubing/cable external rotation variations.[56,60]

Of course, clinicians are not limited to only prescribing isolation exercises. Once a patient demonstrates reduced/eliminated pain and satisfactory neuromuscular control, scapulohumeral compound movements can (and should) be used. Examples include prone scapular retraction with external rotation (elbow flexed to 90°) and face pulls (standing/seated rowing motion with additional external rotation in the contracted position). Clinicians have also had success utilizing PNF patterns. Al Dajah found that combining 5 repetitions of the contract-relax PNF technique for the shoulder internal rotator muscles, followed by 5 repetitions of a PNF

facilitated abduction and external rotation diagonal pattern, reduced pain and improved glenohumeral external rotation and overhead reach.[68] Heron et al used closed chain exercises: double-arm wall press ups, press ups in four point kneeling, and "an exercise whereby the participant adopted a seated position and pressed their hands into the chair, as if trying to lift their body".[65] These exercises were progressed such that the participant could eventually perform the movements with the involved UE and without the uninvolved UE.

Parameters

Adding to the difficulty of formulating a single best SIS exercise protocol is the fact that numerous clinicians have used different protocols and still achieved positive outcomes. The SUPPORT trial endorsed 8-10 total exercises and recommends that initial set and rep ranges will vary depending upon patient tolerance, but 3 sets of 10 per exercise is the prescribed target.[56] Ellenbecker recommends 3 sets of 15-20 repetitions per exercise. Lovering has suggested that since rotator cuff muscular fatigue/weakness is often implicated in pathological glenohumeral arthrokinematics, higher rep ranges may be preferred.[17] Training these muscles for endurance through resistance exercise will facilitate transition of type II muscle fiber into type I, enhancing resilience to fatigue. Kim et al have found that eccentric loading of supraspinatus in abduction produces similar strength gains as concentric loading, but that the additional tensile strain imposed by greater eccentric loads may be more beneficial for maintaining fiber bundle length, remodeling, and healing.[69]

CONCLUSIONS

The shoulder is the most mobile joint in the human body, and with that freedom of movement comes complexity and an invitation for an insidious pathology, subacromial impingement syndrome. SIS most likely develops as a result of both extrinsic and intrinsic factors. No single test is accurate enough to diagnose SIS, but using a combination of special tests increases the post-test reliability of diagnosis. There are many different treatment strategies available to the patient. The most widely accepted intervention is conservative management that includes physical therapy. Surgical management of SIS should be reserved for failure of non-operative treatments or full thickness rotator cuff tendon ruptures. Patient education, manual therapy, and exercise are the most common components of a physical therapy protocol for SIS. Despite the accumulation of evidence that supports exercise intervention, there is no standard protocol for exercise rehabilitation in SIS. Underlying guidelines should direct treatment. Physical therapy intervention should seek to reduce pain, increase ROM, improve posture, normalize scapulohumeral kinematics, and increase shoulder strength and stability.

APPENDIX

Figure 1.

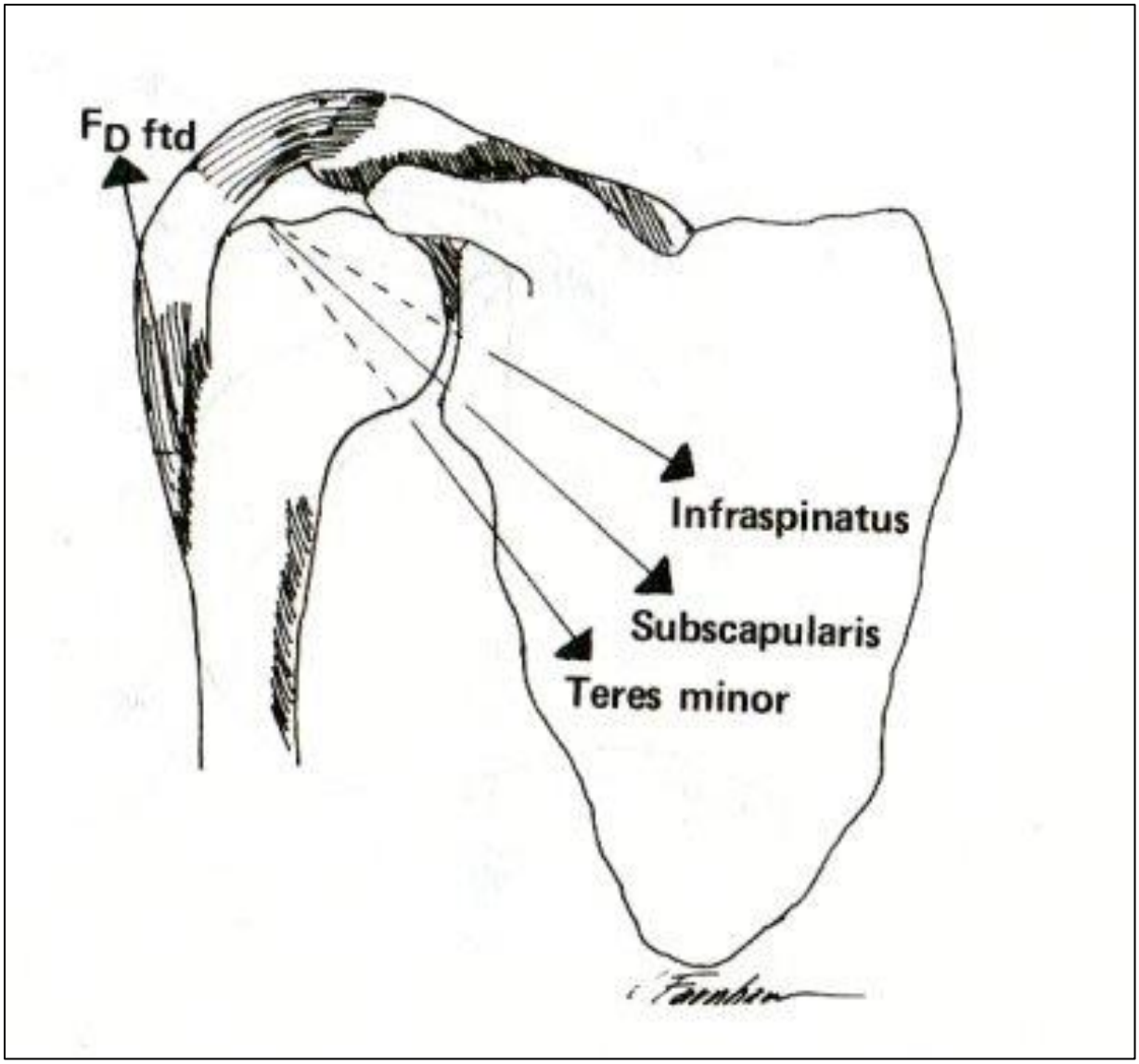


Figure 2. Approximation of the humeral head to the glenoid by balanced force coupling moments between subscapularis and both infraspinatus and teres minor during early abduction.

Image source: http://3.bp.blogspot.com/-LNBjYjWllwo/VmmP8SGuAZI/AAAAAAAACZ4/_4vfmnJcXVI/s1600/shoulder%2Bcentration.jpg

Figure 2.

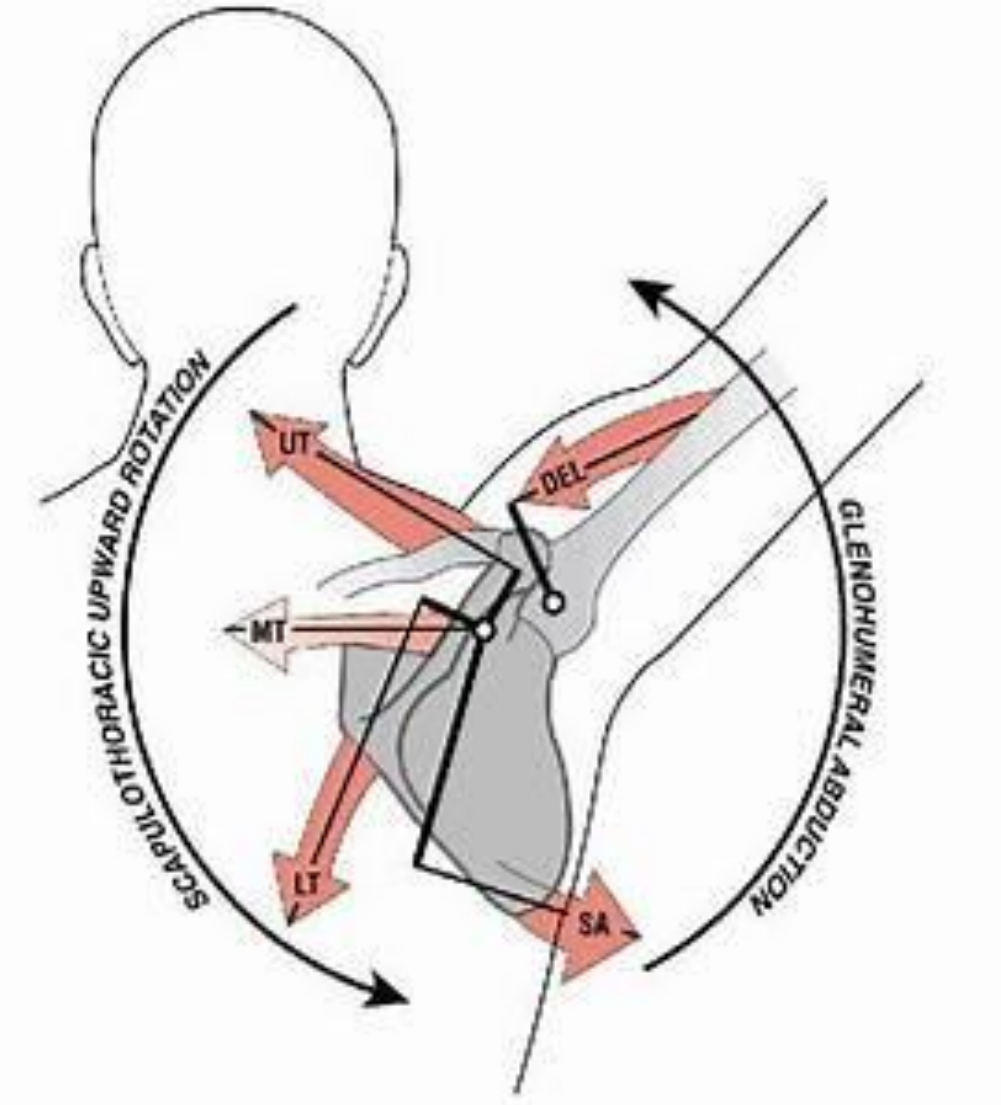
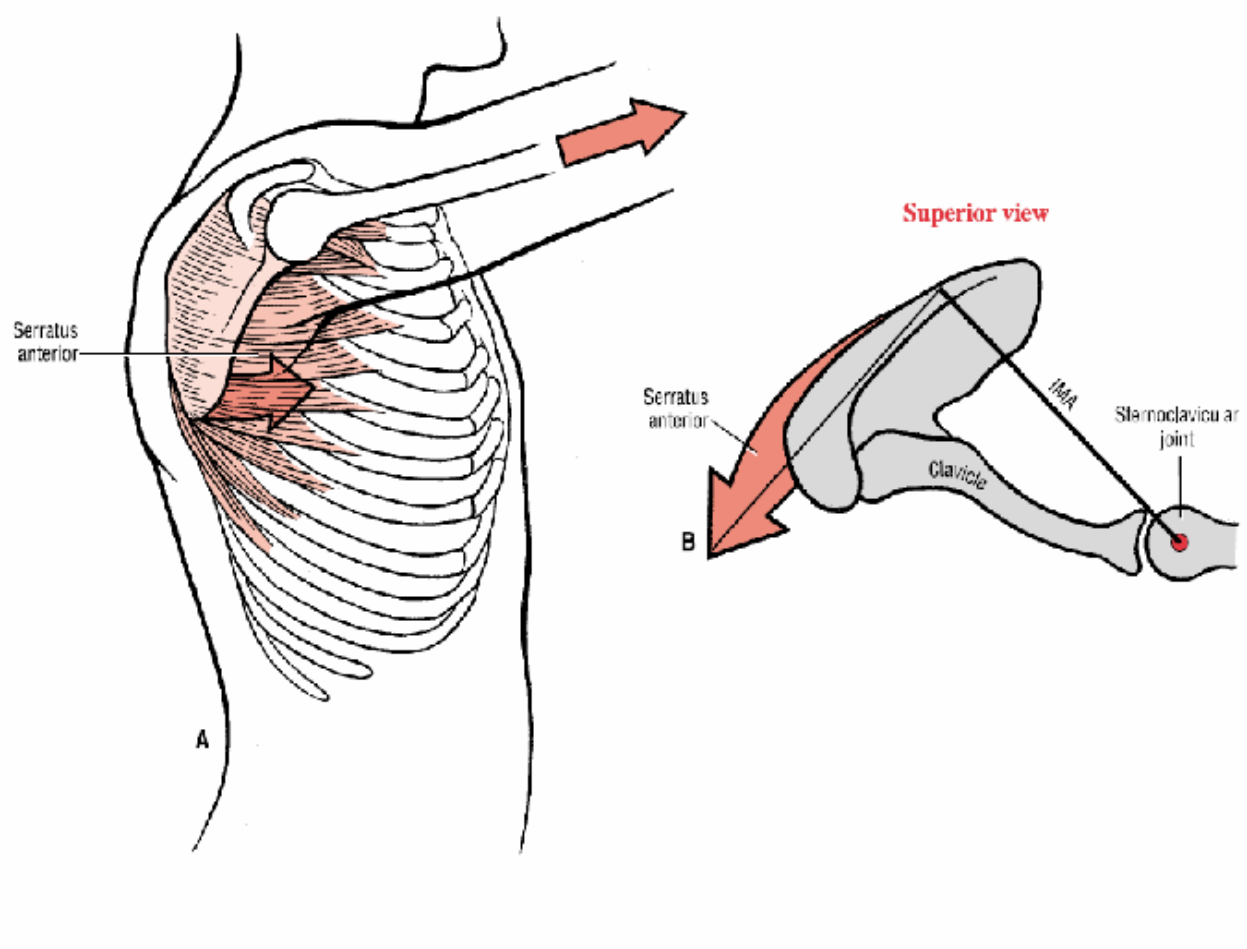


Image source:
http://classconnection.s3.amazonaws.com/295/flashcards/522295/jpg/upward_rotation_scapula1322547571250.jpg

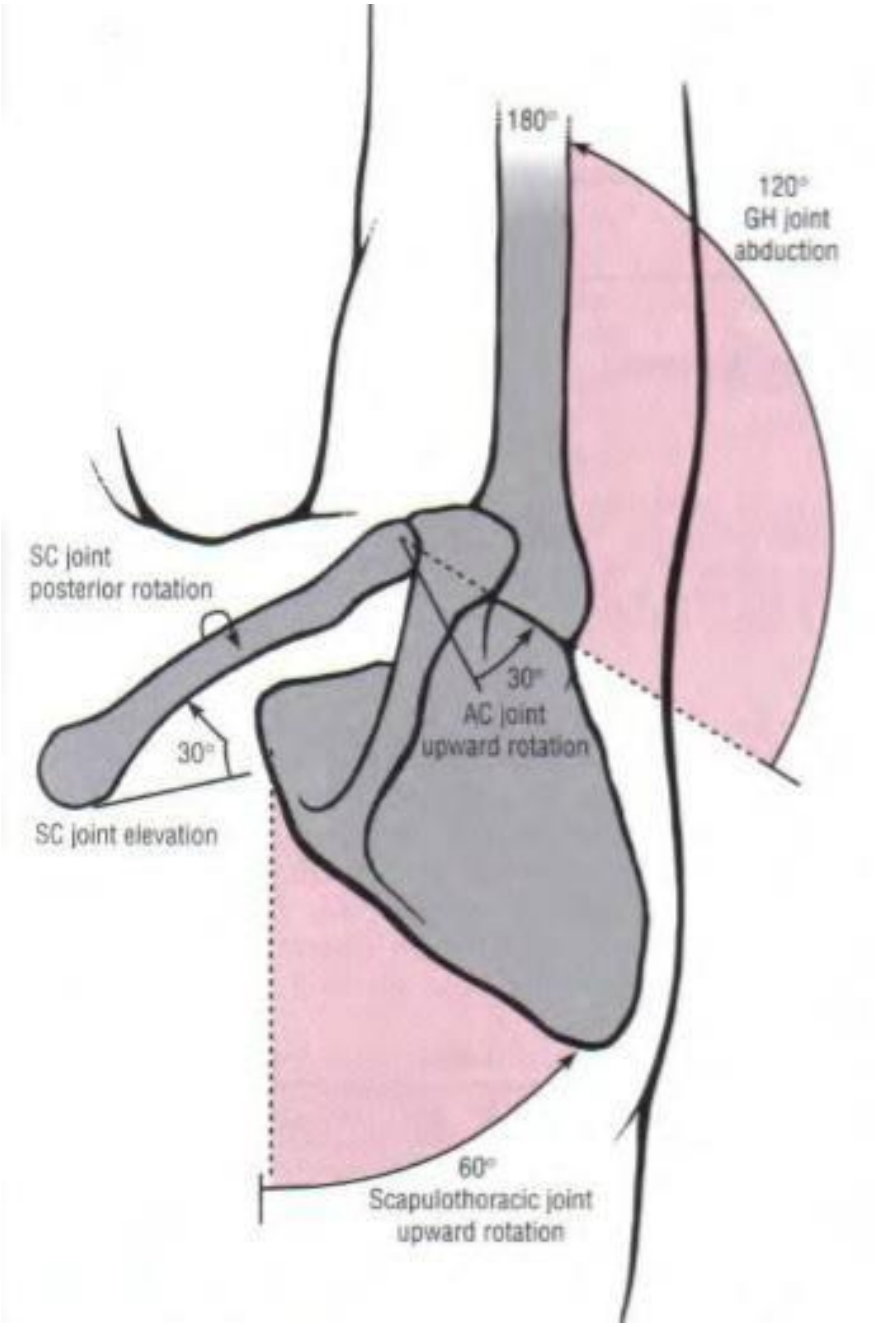
Figure 3.



Normal posterior tipping of scapula by serratus anterior in a healthy shoulder.

Images source: https://3.bp.blogspot.com/-CHJV0BD3URk/VCP8mPE9_5I/AAAAAAAAAoE/wQREmoWGDcA/s1600/lateroposterior_muscles_acting_on_scapula1322546741866.png

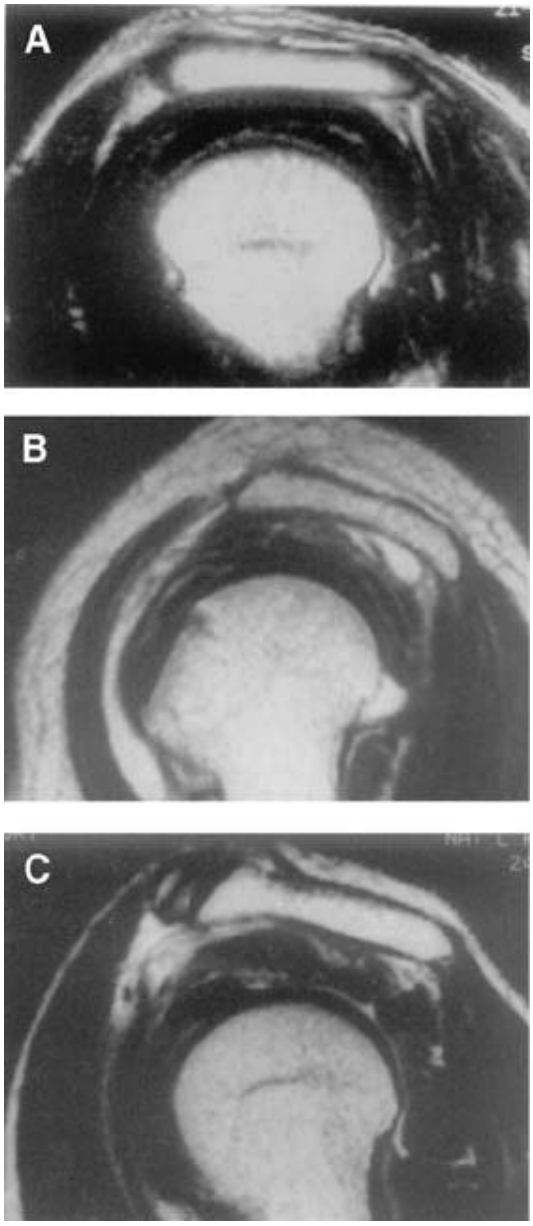
Figure 4.



Full overhead arm elevation requires cooperation between movements of the scapula and humerus. Roughly one third of the movement occurs via scapulothoracic upward rotation and elevation while the glenohumeral joint contributes two thirds of the ROM. In a healthy shoulder, concurrent movement between these bones maintains optimal length-tension ratio for the rotator cuff muscles.

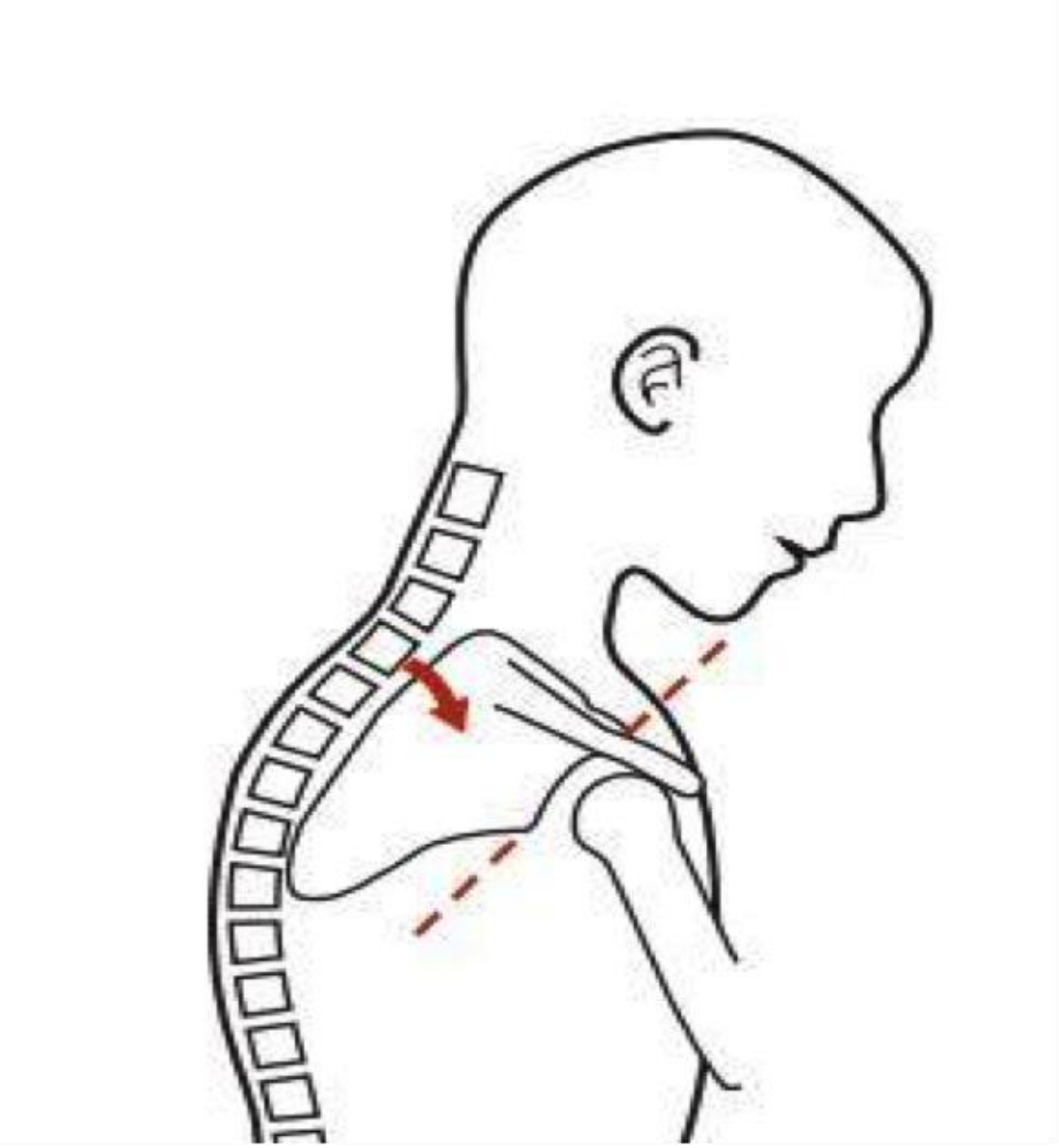
Image source:
http://postfiles2.naver.net/20150215_225/movescience_1423972624300v3qTI_JPEG/scapulohumeral_rhythm.JPG?type=w2

Figure 5.



Acromial shapes determined by the sagittal oblique plane on MRI: type I (flat) (**A**), type II (curved) (**B**), and type III (hooked) (**C**). The longest dimension of the acromion was used to assess type.[15]

Figure 6.



Thoracic kyphosis obligates the scapula to anteriorly tip relative to the humerus, approximating the superior margin of the subacromial space and humeral head.

Image source: https://brentbrookbush.com/articles/research-corner/upper-body/the-link-between-kyphosis-and-subacromial-impingement-syndrome/

Figure 7. Hawkins-Kennedy Test

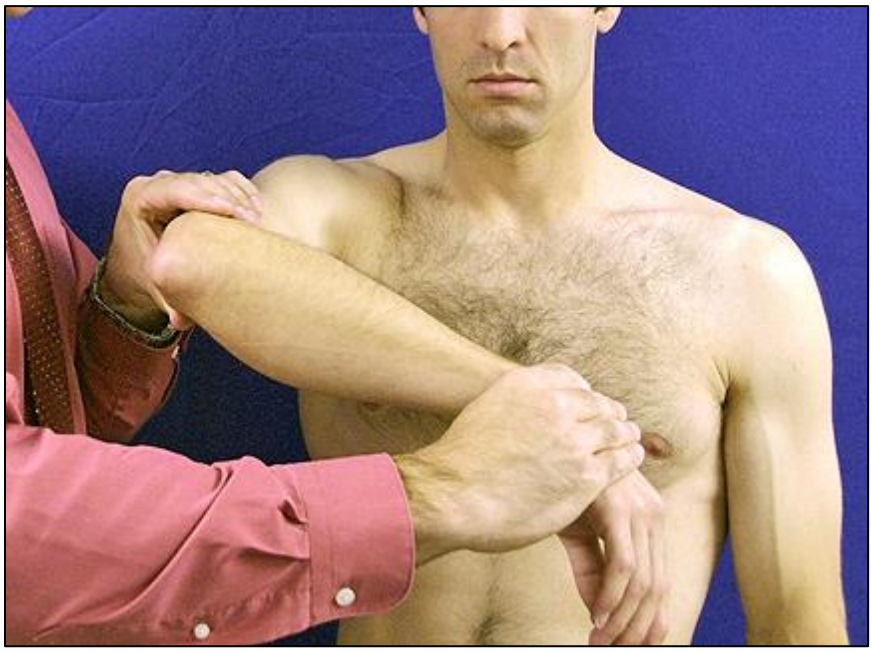


Image source: http://s0www.utdlab.com/contents/images/EM/60425/Hawkins_Kennedy_test.jpg?title=Hawkins+Kennedy+test+for+shoulder+impingement

Figure 8. Neer Test.

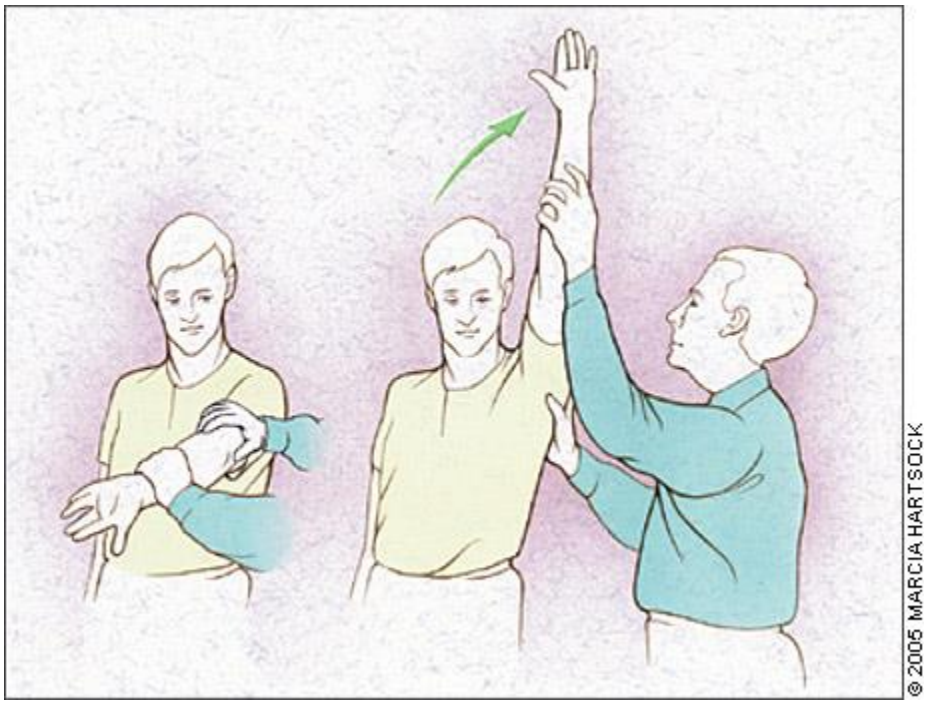


Image source: https://i.pinimg.com/originals/6c/2b/a6/6c2ba65e7e7363d94c3aa28e80c34428.jpg

Figure 9. Painful Arc Test

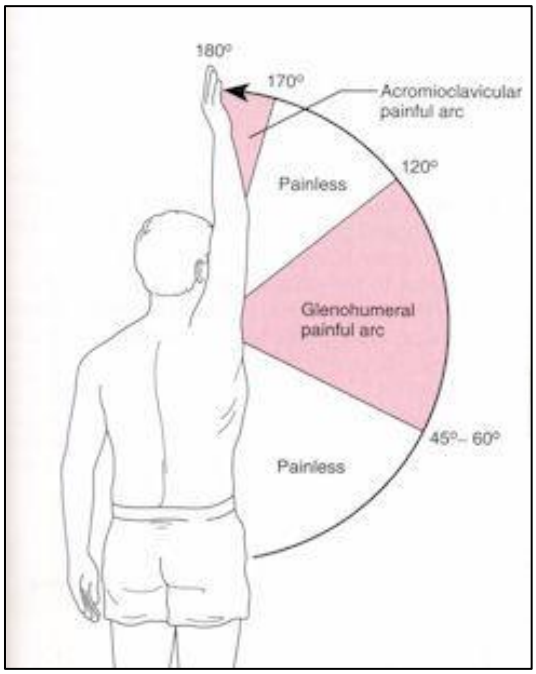


Image source: https://i.pinimg.com/originals/79/97/eb/7997eb866bbf8a8fee02055e3a4715da.jpg

Figure 10. Empty Can Test[24]

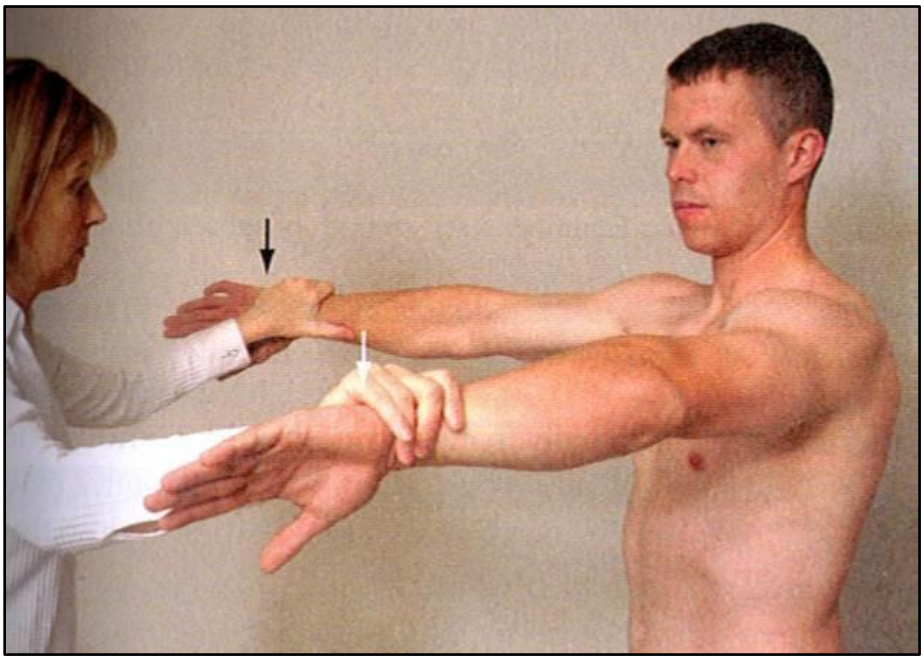


Figure 11. External Rotation Resistance Test

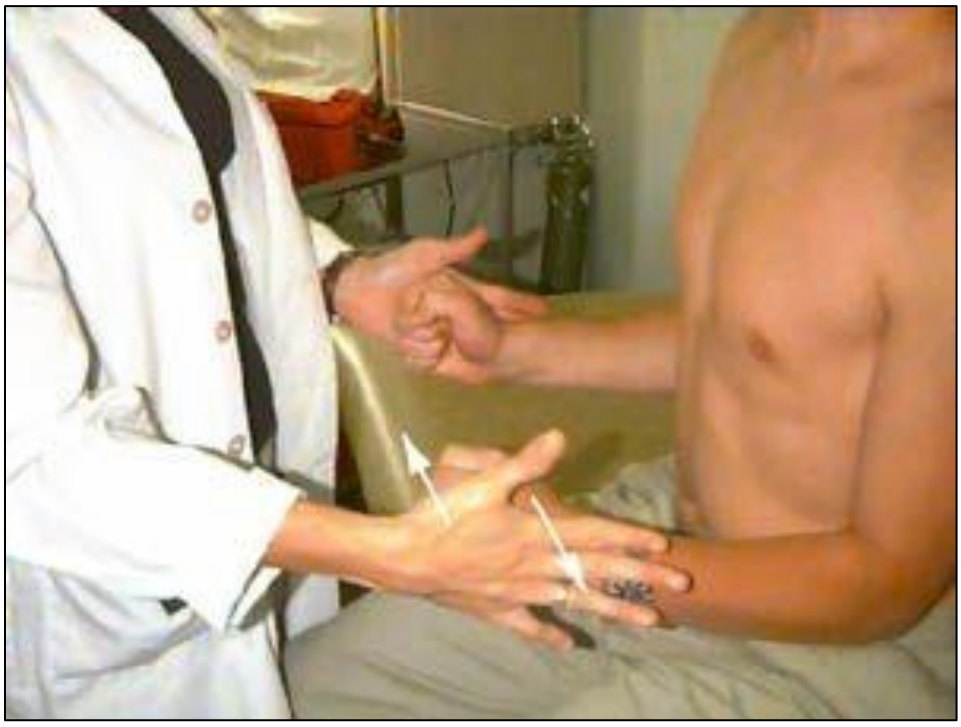


Image source: http://www.nismat.org/data/images/sexam1352199481017.jpg

Figure 12. Drop Arm Test

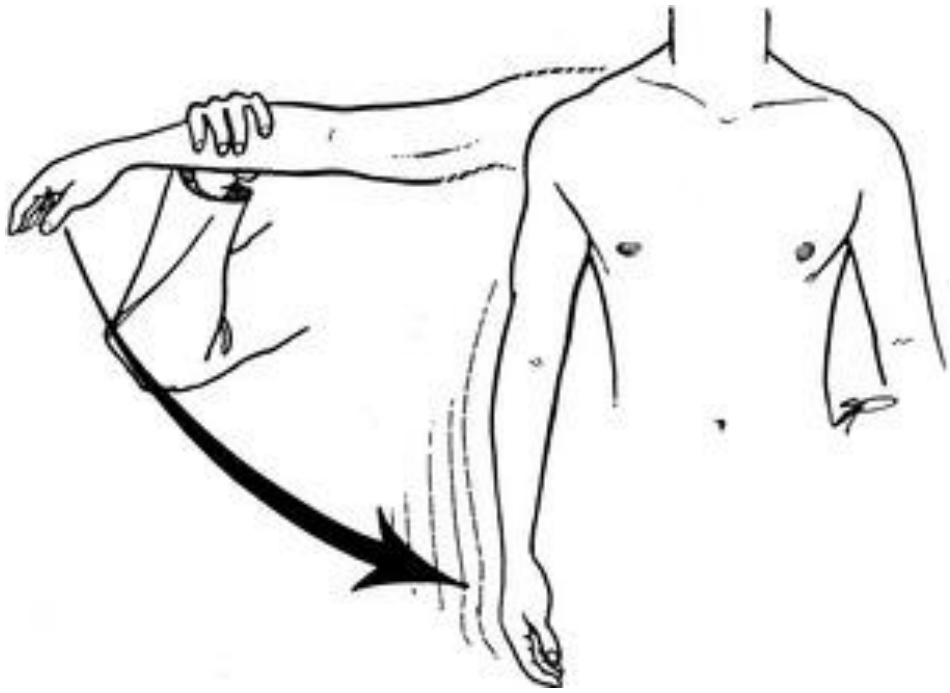


Image source: https://clinicalgate.com/wp-content/uploads/2015/03/B9780323036184100059_gr14.jpg

Figure 13. Lift-Off Test

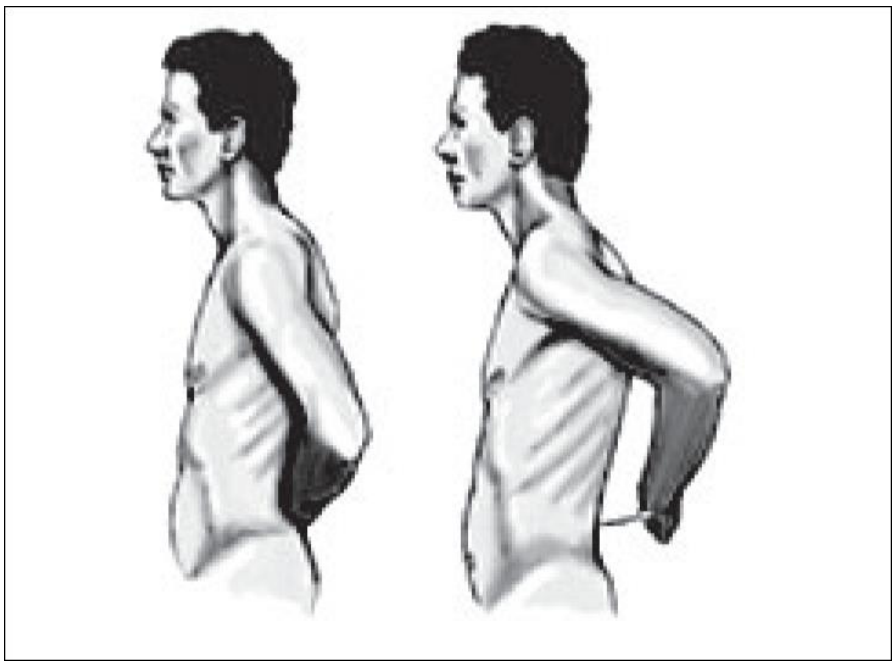


Image source: http://www.internationalshoulderjournal.org/articles/2007/1/1/images/IntJShoulderSurg_2007_1_1_16_30674_4.jpg

Figure 14. Yocum's Test

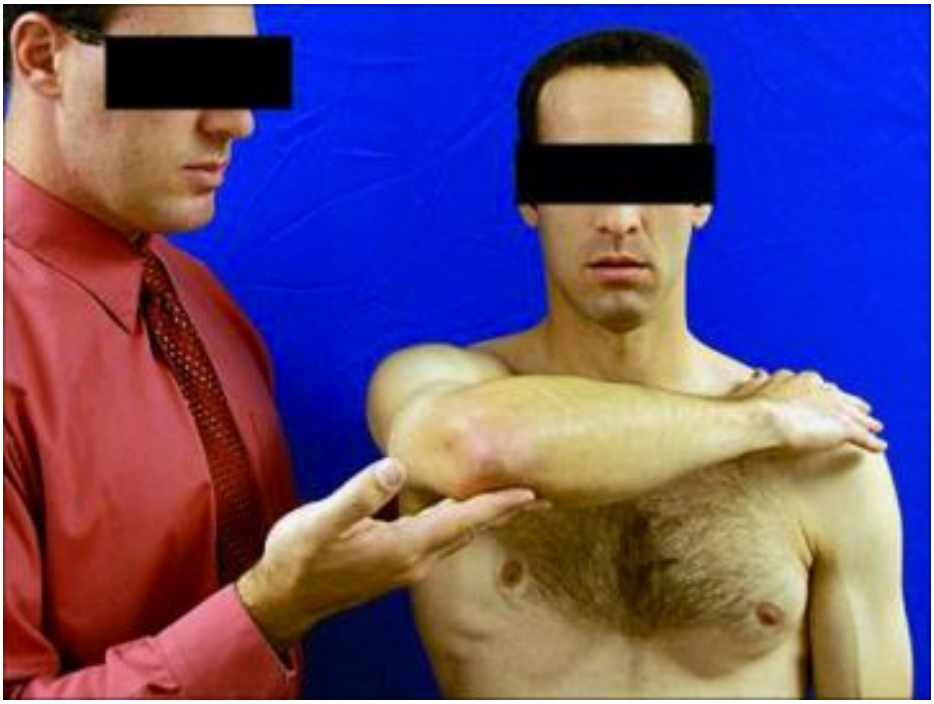


Image source: https://classconnection.s3.amazonaws.com/1412/flashcards/809708/png/yo-thumb400.png

Figure 15. The counter test with elevation in lateral rotation (CELR)[35]

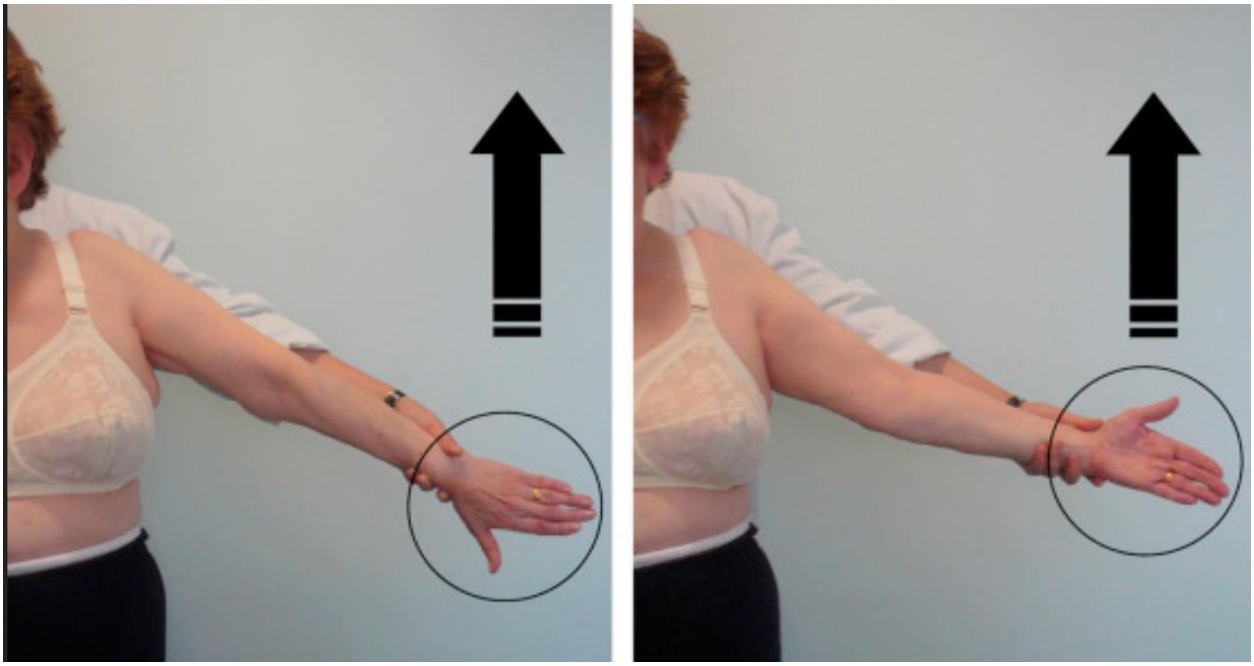
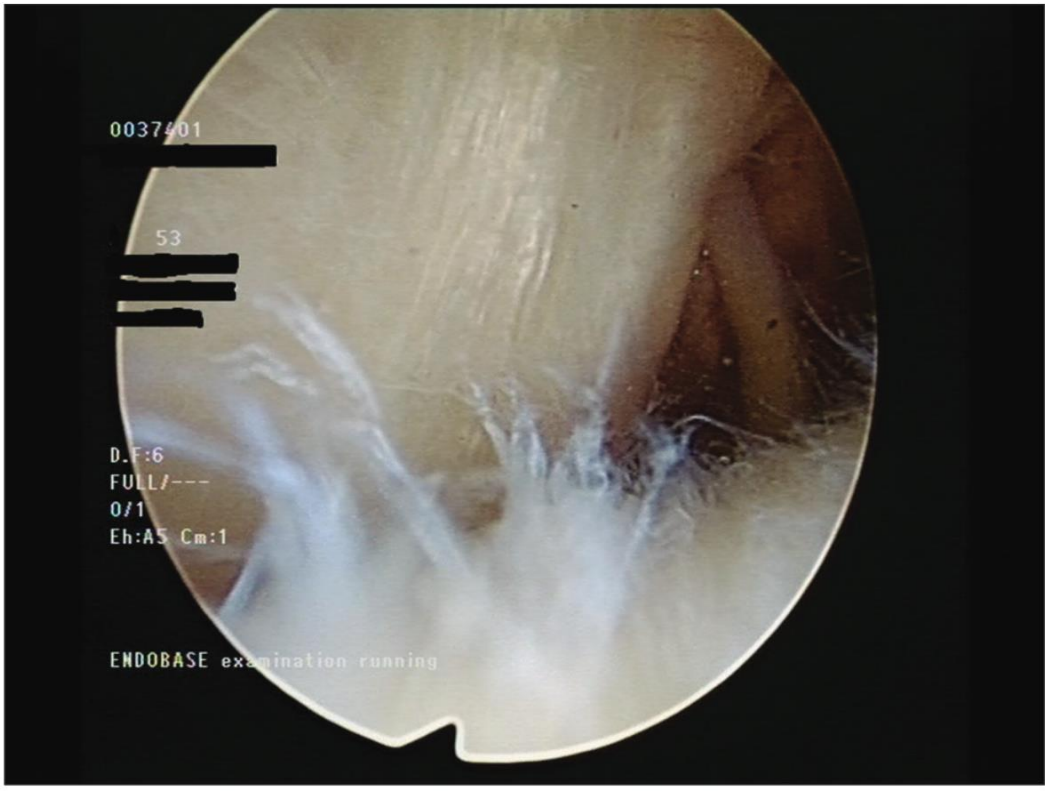


Figure 16. Arthroscopic view showing impingement lesion.[2]



This view shows the long head of the biceps tendon on the right side of the image and fraying of the rotator cuff tendons in the foreground. The rest of the image shows intact rotator cuff tendon over the humeral head.

Table 1. Interrater Kappa Reliability Coefficients and Agreements of 5 special tests for subacromial impingement syndrome.[1]

Test	Kappa Coefficient (95% CI)	Percentage Agreement
Hawkins-Kennedy	.39 (.12–.65)	69
Neer	.40 (.13–.67)	71
Painful arc	.45 (.18–.72)	73
Empty can (Jobe)	.47 (.22–.72)	76
External rotation resistance	.67 (.40–.94)	87

Table 2. Diagnostic Accuracy of Subacromial Impingement Shoulder Tests[1]

Test	Sensitivity (95% CI)	Specificity (95% CI)	+LR (95% CI)	−LR (95% CI)
Hawkins-Kennedy	.63 (.39–.86)	.62 (.46–.77)	1.63 (.94–2.81)	.61 (.31–1.20)
Neer	.81 (.62–1.0)	.54 (.38–.69)	1.76 (1.17–2.66)	.35 (.12–.97)
Painful arc	.75 (.54–.96)	.67 (.52–.81)	2.25 (1.33–3.81)	.38 (.16–.90)
Empty can (Jobe)	.50 (.26–.75)	.87 (.77–.98)	3.90 (1.50–10.12)	.57 (.35–.95)
External rotation resistance	.56 (.32–.81)	.87 (.77–.98)	4.39 (1.74–11.07)	.50 (.28–.89)

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