

Anatomy, Kinesiology, Pathomechanics, and Diagnosis of Shoulder Impingement Symptom

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Abstract : Musculoskeletal complaints with the highest prevalence of patients coming to the hospital one of which is shoulder pain. Based on several studies the current prevalence of complaints of shoulder pain ranges from 7-36% of the population. The most common cause of complaints of shoulder pain coming to the Orthopedic clinic or Medical Rehabilitation is a patient with Shoulder Impingement Syndrome (SIS). Patients with SIS will experience functional limitations making it difficult to do work as usual and difficult to lift weights above the head. SIS that occurs continuously will become a functional disability and decreased quality of life. Initial treatment in this case is given non-operatively, making rehabilitation the first choice as therapy.

1 INTRODUCTION

Shoulder pain is a common presenting complaint from patients of all ages in daily clinical practice, affecting approximately one-third of individuals during their lifetime (Dong W et al, 2015). Such pain may lead to the impairment of shoulder joint function and to severe reduction in quality of life. Shoulder impingement syndrome, which is defined as the compression of the rotator cuff and the subacromial bursa, is considered to be one of the most common causes of shoulder pain and may be cited as a contributing factor to shoulder pain in up to 65% of cases (Garving et al, 2017). Shoulder impingement is a clinical syndrome in which soft tissues become painfully entrapped in the area of the shoulder joint. Shoulder impingement has been defined as compression and mechanical abrasion of the rotator cuff structures as they pass beneath the coracoacromial arch during elevation of the arm (Dong W et al, 2015). In literature shoulder impingement syndrome (SIS) is reported to be a contributing factor between 48% and 65% of all painful shoulder conditions (Burbank et al, 2008). Different kinds of SIS are defined in literature depending on the structures involved: subacromial impingement syndrome (SAI), internal impingement

(IIM), and Sub-coracoid impingement (SC) (Garving et al, 2017).

Charles Neer described subacromial impingement as the compression and abrasion of the bursal side of the rotator cuff beneath the anterior acromion, and developed the anterior acromioplasty as a treatment (Neer, 1983). External impingement is now understood as a much broader category than that described by Neer, and could include compression or abrasion of the cuff tendons or tendon of the long head of the biceps brachii beneath any aspect of the coracoacromial arc (Neer, 1983). The coracoacromial arch includes not just the acromial undersurface, but also the coracoacromial ligament, and the undersurface of the acromioclavicular (AC) joint (Garving et al, 2017).

2 DISCUSSION

2.1 Joints

The main function of the joints of the shoulder girdle is to move the arm and hand into almost any position in relation to the body. As a consequence, the

shoulder joint is highly mobile, where stability takes second place to mobility, as is evident from the shape of the osseous structures: a large humeral head lying on an almost flat scapular surface. Stability is provided mainly by ligaments, tendons and muscles; the bones and capsule are of secondary importance. The function of the shoulder girdle requires an optimal and integrated motion of several joints. In fact, five 'joints' of importance to 'shoulder' function can be distinguished:

1. The glenohumeral joint
2. The acromioclavicular joint
3. The sternoclavicular joint
4. The subacromial joint or subacromial gliding mechanism: the space between the coracoacromial roof and the humeral head, including both tubercles. This is the location of the deep portion of the subdeltoid bursa
5. The scapulothoracic gliding mechanism: this functional joint is formed by the anterior aspect of the scapula gliding on the posterior thoracic wall. Optimal mobility also requires an intact neurological and muscular system.⁵

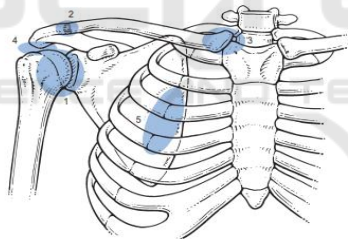


Figure 1: A global view of five joints of the shoulder girdle: 1. Glenohumeral joint; 2. Acromioclavicular joint; 3. Sternoclavicular joint; 4. Subacromial joint or subacromial gliding mechanism; 5. Scapulothoracic gliding mechanism.

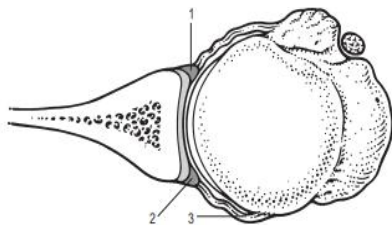


Figure 2: Shoulder (glenohumeral) joint: 1. Labrum, 2. Glenoid cartilage, 3. Shoulder

2.1.1 Glenohumeral joint

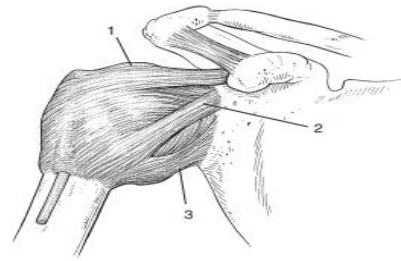


Figure 3: The glenohumeral joint capsule and ligament: 1. Superior; 2. Medial, 3. Inferior (glenohumeral)

The glenohumeral joint is a ball-and-socket between humeral head and glenoid fossa. There is a remarkable geometrical relationship between glenoid and head which is responsible for the considerable mobility of the joint but is also an important predisposing factor to glenohumeral instability. First, the large spherical head of the humerus articulates against the small shallow glenoid fossa of the scapula (only 25–30% of the humeral head is covered by the glenoid surface). Second, the bony surfaces of the joint are largely incongruent (flat glenoid and round humerus). However, the congruence is greatly restored by the difference in cartilage thickness: glenoid cartilage is found to be the thickest at the periphery and thinnest centrally, whereas humeral articular cartilage is thickest centrally and thinnest peripherally. This leads to a uniform contact between humeral head and glenoid surface throughout shoulder motion (Rockwood et al, 2009).

The labrum is a fibrous structure that forms a ring around the periphery of the glenoid. It further contributes to stability of the joint by increasing the depth of the glenoid socket, enlarging the surface area and acting as a load-bearing structure for the humeral head. The synovial membrane of the joint capsule is mainly attached to the labrum, covering its inner surface, and at the anatomical neck of the humerus. The fibrous portion of the capsule is very lax and has several recesses, depending on the position of the arm. Very often adhesions form here. The joint capsule is large, loose and redundant allowing full and free range of motion of the shoulder. The rotator cuff and labrum are the shoulder structures most vulnerable to throwing injuries (Lin DJ et al, 2018).

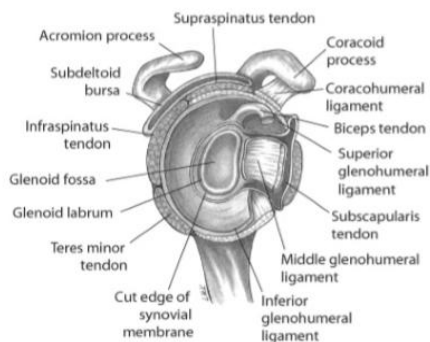


Figure 4: The glenohumeral joint.

At the anterior portion of the capsule three local reinforcements are present: the superior, medial and inferior glenohumeral ligaments. These contribute, together with the subscapularis, supraspinatus, infraspinatus and teres minor muscles, to the stability of the joint. By virtue of the blending of their tendons with the glenohumeral capsule and ligaments, selective contraction of the cuff muscles can adjust the tension in these structures, producing ‘dynamic’ ligaments (Rockwood et al, 2009).

2.1.2 Acromioclavicular joint

The acromioclavicular joint is the only articulation between the clavicle and the scapula. The anteroposterior stability of the acromioclavicular joint is controlled by the acromioclavicular ligaments and the vertical stability is controlled by coracoclavicular ligaments (conoid and trapezoid) (Rockwood et al, 2009).

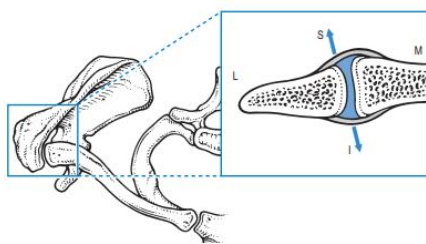


Figure 5: Acromioclavicular joint; S:Superior, I:Inferior, L:Lateral, M:Middle.

2.1.3 Sternoclavicular joint

Its the medial end of clavicle lies in contact with the superolateral angle of the sternal manubrium and with the medial part of the cartilage of the first rib to

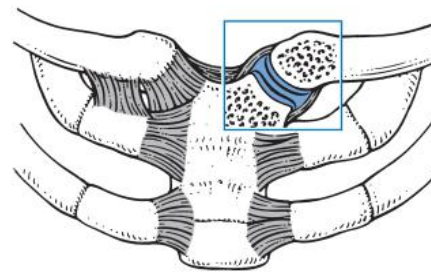


Figure 6: The sternoclavicular joint.

form the sternoclavicular joint. In both the vertical and anteroposterior dimensions, the clavicular portion is larger than the opposing manubrium and extends superiorly and posteriorly relative to the sternum. The prominence of the clavicle enables its palpation. The sternoclavicular joint is mobile along all axes and almost every movement of the scapula and the arm is associated with some movement at this joint (Rockwood et al, 2009).

2.2 Muscles

The primary muscle group that supports the shoulder joint is the rotator cuff muscles. The four rotator cuff muscles are supraspinatus, infraspinatus, teres minor, and subscapularis. Together the rotator cuff muscles form a musculotendinous cuff as they insert on the proximal humerus.

The rotator cuff muscles attach to the proximal humerus anteriorly at the greater tuberosity. The rotator cuff muscles provide considerable structural support to the glenohumeral joint and keep the humeral head in a firm position by articulating with the scapula within the glenoid cavity. The muscles of the chest also provide structural support to the shoulder joint (Eovaldi et al, 2018).

2.2.1 Supraspinatus Muscle

The supraspinatus muscle functions by abduction of the humerus up to 30 degrees, as well as to stabilize the glenohumeral joint (Burbank et al, 2008).

Approximately 70% of the muscle fibers attach to the intramuscular tendon, whereas 30% attach directly to the extra muscular tendon. This muscle is categorized as a circumpennate muscle (Eovaldi et al, 2018).

The supraspinatus is part of the force couple to stabilize the glenohumeral joint by compression and initializes elevation. Elevation in case of supraspinatus paralysis requires more deltoid force,



Figure 7. The Supraspinatus Muscle

but the other rotator cuff muscles are still able to stabilize the humeral head sufficiently for full range of motion. The suprascapular nerve (C4-6) supplies innervation (Reyes et al, 1978).

2.2.2 Infraspinatus Muscle

The infraspinatus muscle functions by externally rotating the humerus and doing horizontal abduction (Reyes et al, 1978).

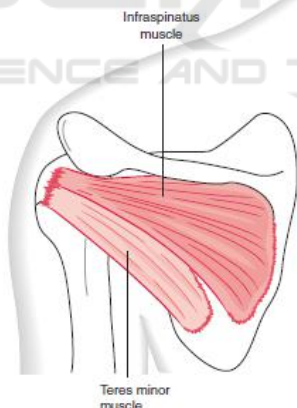


Figure 8: The Infraspinatus and Teres Minor Muscle.

2.2.3 Teres Minor Muscle

The Teres minor acts to externally rotate the humerus and assists with abduction of the humerus. Similar to the infraspinatus, this is a circumpennate muscle with a single intramuscular tendon located in the center of the muscle belly. The teres minor

muscle acts as stabilizer of the glenohumeral joint by resisting posterior and superior translation and generates 45% of the total external rotation force.

2.2.4 Subscapularis Muscle

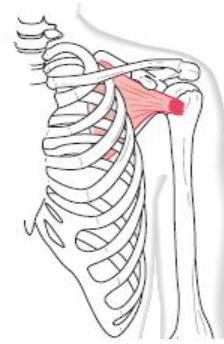


Figure 9: The Subscapularis Muscle

As the only component of the anterior rotator cuff, it stabilizes actively the glenohumeral joint by resisting anterior and inferior translation and acts as a strong internal rotator. It is considered to be a passive stabilizer too, because of the dense collagen structure of its tendon, and its fusion with the middle and inferior glenohumeral ligament (Eovaldi et al, 2018).

2.2.5 Trapezius Muscle

The only muscle which acts to depress the shoulder is the lower trapezius, which is assisted by gravity in the upright position. The function of the trapezius muscle is both elevation and depression of the shoulder depending on whether the upper or lower muscle fibers are activated. When the entire trapezius muscle contracts the fibers are geometrically opposed, and the forces are balanced resulting in no movement of the shoulder (Eovaldi et al, 2017).

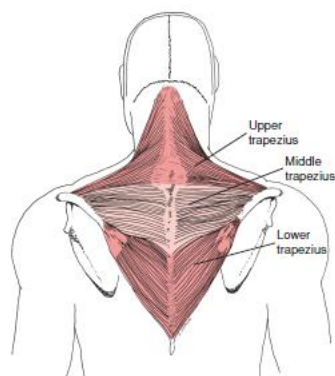


Figure 10: The Trapezius Muscle

2.2.6 Deltoid Muscle

The deltoid muscle overlies the shoulder superficially and functions to abduct the humerus. The deltoid muscle has three origins; the body of the clavicle, the spine of the scapula, and the acromion. The deltoid muscle has three origins; the body of the clavicle, the spine of the scapula, and the acromion. The deltoid muscle has its insertion on the deltoid tuberosity of the humerus. The short head of the biceps brachii originates from the coracoid process, and the long head originates from the supraglenoid tubercle, passing through the intertubercular groove of the proximal humerus. The biceps brachii is not a shoulder muscle but does originate from the shoulder. Paralysis of the deltoid results mainly in 50% loss of abduction strength. The axillary nerve (C4-5) innervates the deltoid (Lynn, 2000).

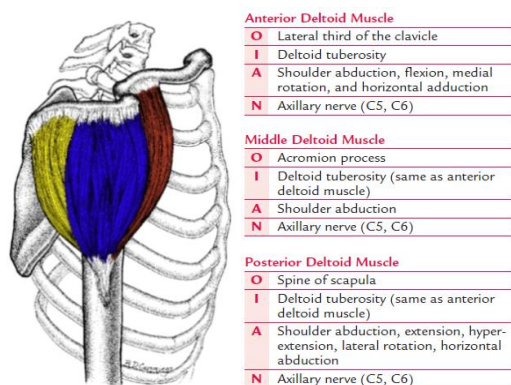


Figure 11: The Deltoid Muscle.

2.3 Etiology of SIS

Internal impingement was first described as a condition noted in overhead athletes, identified in part due to poor outcomes of acromioplasty in this population. Multiple theories exist as to the primary etiology of shoulder impingement, including anatomic abnormalities of the coracoacromial arch or humeral head “tension overload,” ischemia, or degeneration of the rotator cuff tendons; and shoulder kinematic abnormalities. Regardless of the initial etiology, inflammation in the suprahumeral space, inhibition of the rotator cuff muscles, damage to the rotator cuff tendons, and altered kinematics are believed to exacerbate the condition. Impingement is thought to be due to inadequate space for clearance of the rotator cuff tendons as the arm is elevated. Therefore, factors that further minimize this space are believed to be detrimental to the condition (Ludewig and Cook, 2000)

There are some structures that could contribute to its onset, such as the shape of the acromion, the coracoacromial ligament, the superior aspect of the glenoid fossa, hypermobility and instability of the glenohumeral joint, capsular retractions and rotator cuff tendinopathy (Lewis et al, 2005). Rotator cuff problems are thought to account for nearly one third of physician visits for shoulder pain complaints (Ludewig and Cook, 2000). The vast majority of people with impingement syndrome who are younger than 60 years of age relate their symptoms to occupational or athletic activities that involve frequent overhead use of the arm. Epidemiologic investigations have revealed a high prevalence (16%–40%) of shoulder complaints consistent with impingement in certain occupations, including assembly-line workers, welders, steelworkers, and construction workers. Frequent or sustained shoulder elevation at or above 60 degrees in any plane during occupational tasks has been identified as a risk factor for the development of shoulder tendinitis or nonspecific shoulder pain. Evidence relating occupational exposure of frequent or sustained shoulder elevation to shoulder musculoskeletal symptoms is strongest for combined exposure to multiple physical factors, such as holding a tool while working overhead.

A number of impingement categories have been identified including subacromial impingement or “external impingement”; internal impingement, which can be further divided into anterior or posterior; and coracoid impingement.¹⁴

All categories of impingement are potential mechanisms for the development or progression of rotator cuff disease, or long head biceps tendinopathy. Physical exam findings consistent with impingement can also be associated with labral tears in internal impingement (Budoff, 2003) or develop secondary to instability or as a delayed consequence of adhesive capsulitis. There are multiple mechanisms by which impingement may occur, including excess or reduced motion and abnormal patterns of motion at particular portions of the range of motion (Micahener et al, 2003). In addition, anatomic abnormalities of the humerus or acromion have been implicated in impingement. It should be noted that rotator cuff disease can develop without impingement, through tensile overload or intrinsic tissue degeneration. Regardless of the initial precipitating factor, however, impingement, abnormal shoulder motions, and associated rotator cuff disease often are found in the presence of partial or full thickness rotator cuff tears. In other words, even if rotator cuff disease or tearing did not initiate from impingement or abnormal motion, impingement and abnormal motion are likely to contribute to disease progression (Manske et al, 2014).

The subacromial impingement syndrome has both primary and secondary forms. Primary impingement is due to structural changes that mechanically narrow the subacromial space; these include bony narrowing on the cranial side (outlet impingement), bony malposition after a fracture of the greater tubercle, or an increase in the volume of the subacromial soft tissues – due, e.g., to subacromial bursitis or calcific tendinitis – on the caudal side (non-outlet impingement). Secondary impingement results from a functional disturbance of centering of the humeral head, such as muscular imbalance, leading to an abnormal displacement of the center of rotation in elevation and thereby to soft tissue entrapment (Garving et al, 2017).

2.4 Stages of SIS

Neer graded SIS into 3 different stages. In stage I, the typical characteristics are reversible lesions with edema and hemorrhage; most patients younger than 25 years are in this category. In stage II, chronic inflammation or repeated episodes of impingement lead to histomorphological changes, such as fibrosis and thickening of the supraspinatus, the long biceps tendon, and subacromial bursae. Patients in this stage are usually between 25 and 40 years of age.

In stage III, in patients more than 40 years of age, tears of the rotator cuff, rupture of the biceps tendon, and bony changes may be observed, accompanied by significant tendon degeneration following a long history of refractory tendinitis.

2.5 Normal Motion of Shoulder

During normal motion, the scapula will upwardly rotate and posteriorly tilt on the thorax during elevation of the arm in flexion, abduction, scapular plane abduction, or unrestricted overhead reaching (Ludewig and Barman, 2011). Throughout this manuscript, elevation will be used to refer the raising of the arm overhead in any of these planes. Scapulothoracic internal or external rotation is less consistent during arm elevation, differing in pattern depending on what plane the arm is elevated in, and depending on what portion of the elevation range of motion is considered (Ludewig and Reynolds, 2009). The scapula must adjust in the transverse plane for the intended plane of elevation. For flexion, the scapula will internally rotate somewhat early in the motion, whereas for coronal plane abduction, it will externally rotate at the initiation of the motion. Based on the limited end range data available, it appears some external rotation of the scapula will occur near end range for each of these planes of elevation (Ludewig and Braman, 2011).

Recent investigations have added new knowledge on how SC and AC joint motions contribute to overall ST motion. The primary clavicular motion occurring at the SC joint during active arm elevation in any plane except extension is 30° of posterior long axis rotation.¹⁷ Secondly, the clavicle will retract $w15^\circ$ at the SC joint during elevation, even with flexion. However, the clavicle also “adjusts” in the transverse plane (less retraction with flexion, more with abduction) similarly to the changes in scapular internal rotation with flexion versus abduction (Ludewig and Reynolds, 2009). Finally, a small amount of clavicular elevation will occur at the SC joint with humeral elevation in any plane. Concurrent with clavicular motion relative to the thorax, measurable motion of the scapula relative to the clavicle is also occurring at the AC joint as the humerus is elevated in any plane. Primary AC joint motions include upward rotation and posterior tilt of the scapula relative to the clavicle. Secondly the scapula will internally rotate relative to the clavicle at the AC joint, even

while abducting the arm (Ludewig and Braman, 2011).

Overall ST motion occurs either through motion of the clavicle relative to the thorax, motion of the scapula relative to the clavicle, or some combination of both. During normal arm elevation in any plane, both clavicular (SC) and scapular (AC) motions described above are contributing to the final position of the scapula on the thorax. However, the non-parallel alignment of the axes of rotation of the SC and AC joints makes their contributions to ST motion challenging to visualize (Teece et al, 2008). The AC joint axes are aligned consistently with how the axes are described for the scapula on the thorax, such that if the scapula upwardly rotates, posteriorly tilts or internally rotates relative to the clavicle, there is a 1:1 “coupling” with ST motion. In other words, 5° of scapular upward rotation relative to the clavicle would contribute to 5° of ST upward rotation. In order to understand the coupling of clavicular motion to ST motion, it is helpful to visualize an axis of rotation embedded along the long axis of the clavicle, and another embedded in the scapula from the root of the scapular spine to the AC joint. In a superior transverse plane view, first imagine a hypothetical situation where the clavicle and scapular axes are parallel. In such a hypothetical alignment, if the clavicle were elevated about its anteriorly directed axis 9° relative to the thorax, the scapula would upwardly rotate 9° on the thorax, assuming no motion of the scapula relative to the clavicle at the AC joint. If the clavicle rotated posteriorly about its long axis 30° relative to the thorax, the scapula would posteriorly tilt 30° relative to the thorax, and if the clavicle retracted 9° relative to the thorax, the scapula would externally rotate 9° relative to the thorax (Teece et al, 2008). Now consider an alternative hypothetical situation where the scapula is internally rotated 90° relative to the clavicle, such that the described axes in the transverse plane are at a 90° angle (Ludewig and Braman, 2011). In such a hypothetical alignment, if the clavicle were elevated about its anteriorly directed axis 9° relative to the thorax, the scapula would anteriorly tilt 9° on the thorax. If the clavicle rotated posteriorly about its long axis 30° relative to the thorax, the scapula would upwardly rotate 30° on the thorax, and if the

clavicle retracted 9° relative to the thorax, the scapula would externally rotate 9° on the thorax (Teece et al, 2008).

In addition to the coupling of clavicle motion to ST motion, during arm elevation in any plane, the scapula relative to the clavicle is also moving at the AC joint. These AC joint motions may increase or decrease the overall ST joint motion depending on whether they complement or offset the SC joint coupled scapular motions. So, in the example above for scapular plane abduction to 120° relative to the thorax, the 20° ST upward rotation coupled with clavicle posterior rotation on the thorax, and 3° ST upward rotation coupled with clavicle elevation on the thorax would be complemented by an average of 11° of scapular upward rotation relative to the clavicle across the same increment of scapular plane abduction. The end result would be 34° of ST upward rotation. For ST tilting, the 10° posterior tilting coupled with clavicle posterior rotation on the thorax would be reduced by 6° anterior tilting coupled with clavicle elevation on the thorax as described above. Subsequently, the clavicle overall contribution to ST posterior tilting would only be 4°.

However, the scapula relative to the clavicle is tilting posteriorly during that scapular plane abduction motion on average 16°, to result in overall ST motion of 20°. Finally the 9° of ST external rotation coupled with clavicle retraction on the thorax is offset by an average of 6° scapula internal rotation relative to the clavicle, resulting in 3° of ST external rotation. Note that final resulting scapular upward rotation motion and position on the thorax is produced by complementary motion of the clavicle relative to the thorax and scapula relative to the clavicle. ST tilting is produced almost exclusively by scapular motion relative to the clavicle as the clavicle elevation and posterior rotation motions at the SC joint are offsetting. ST external rotation is minimal due to offsetting motions of clavicle retraction relative to the thorax and scapular internal rotation relative to the clavicle (Ludewig and Braman, 2011).

2.6 Abnormal Shoulder Motion in Impingement

Recent review article identified scapular motion abnormalities in subjects with impingement or rotator cuff disease. The most frequent findings have been reduced ST posterior tilting, reduced ST upward rotation, increased ST internal rotation, or increased clavicular elevation relative to the thorax (Ludewig and Reynolds, 2009). These movement alterations are believed to increase proximity of the rotator cuff tendons

to the coracoacromial arch or glenoid rim (Ludewig and Braman, 2011).

Additionally, increased humeral head superior or anterior translation has been found in subjects with impingement (Ludewig and Braman, 2011). These directions of humeral head motion are believed to reduce the subacromial space and increase impingement risk. Biomechanical evidence also supports the idea of glenohumeral internal rotation contributing to sub-acromial impingement beneath the anterior structures (Ludewig and Braman, 2011).

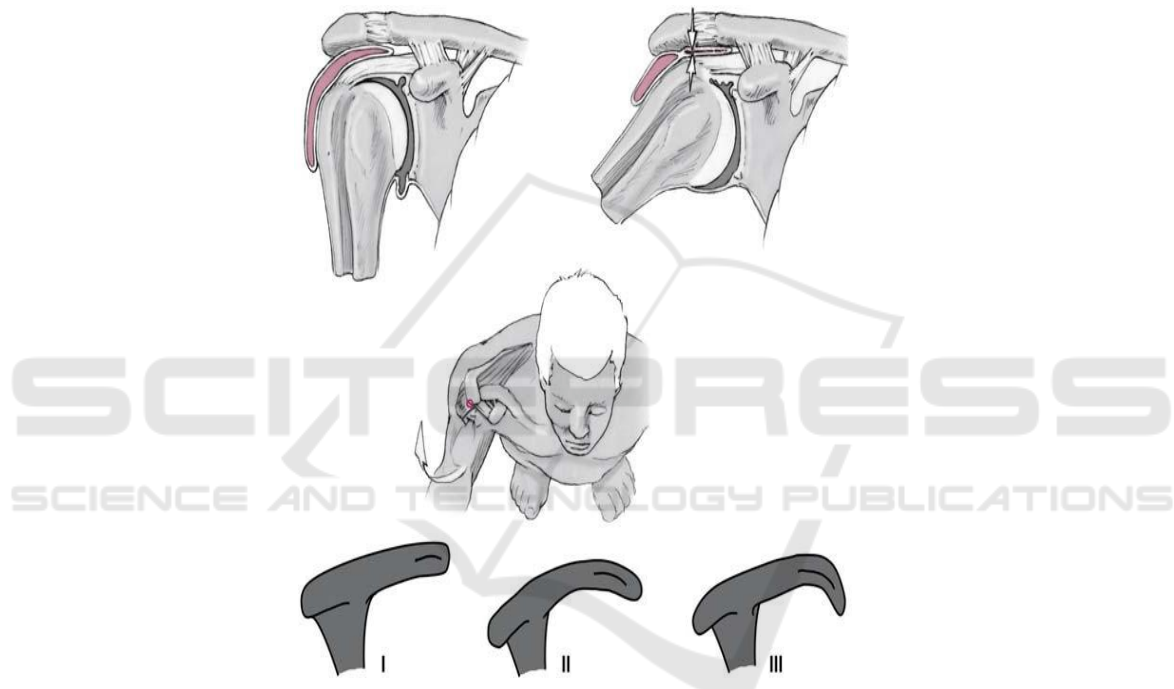


Figure 12: Anatomical overview of the shoulder (left, above), showing the mechanism of subacromial impingement with painful entrapment of soft tissues (arrows, right, above) on elevation of the arm, due to pathological contact of the humeral head with the roof of the shoulder joint, particularly the anterolateral portion of the acromion (below).

Acromial shapes as classified by Bigliani and Morrison: type I (flat), type II (curved), type III (hooked).

Recent work also demonstrates how angles of humeral elevation which minimize the subacromial space may differ from angles of humeral elevation where the rotator cuff soft tissues are at greatest risk. The subacromial space is typically described as minimized at 90° of humeral elevation in all planes. However, the portion of the humerus in closest contact at that point in the range of motion of abduction is actually the lateral aspect of the greater tuberosity, which has no rotator cuff soft tissue. The rotator cuff tendons are actually in closest proximity to

the undersurface of the acromion near 45° of humeral abduction relative to the thorax. By angles past 60° humeral abduction, the attachment sites or footprints of the cuff tendons on the greater tuberosity have rotated past the lateral acromial undersurface.²⁰ Patients may still have a painful arc of motion near 90° of humeral elevation in any plane, since this is where rotator cuff muscle forces are highest. However, pain at or above 90° of humeral elevation relative to the thorax is unlikely a direct result of a compressive subacromial impingement of the rotator cuff

tendons. Alternatively, proximity of the undersurface of the cuff tendons to the superior glenoid rim increases at higher arc (Ludewig and Braman, 2011). These same factors can influence humeral motions. In summary, there is some evidence of increased upper trapezius activation and reduced serratus anterior activation in the same subjects who have demonstrated reduced ST posterior tilting, increased internal rotation, and reduced upward rotation. There is also evidence of increased ST anterior tilting and internal rotation in subjects with a relatively short resting length of the pectoralis minor (Ludewig and Braman, 2011).

Glenohumeral internal rotation deficit and experimentally induced posterior capsule tightness have also been shown to increase ST anterior tilting and humeral anterior translations relative to the glenoid, respectively. Slouched sitting, thoracic kyphosis, and increased age have also been related to increased ST anterior tilting and internal rotation and reduced ST upward rotation (Ludewig and Braman, 2011).

Although not experimentally demonstrated, other factors including reduced rotator cuff activation and pectoralis major tightness can be biomechanically theorized to impact ST or glenohumeral kinematics in ways that are believed to increase impingement risk. Each of these factors provides additional insight in planning treatment intervention approaches targeted to specific movement deviations.

2.7 Clinical Presentation

The patient should be asked about the nature, duration, and dynamics of the pain and about any precipitating trauma (perhaps trivial trauma) or stress, as well as about analgesic use. Patients often report painful elevation and depression of the arm between 70 ° und 120 °, pain on forced movement above the head, and pain when lying on the affected shoulder. The physical examination consists of inspection, palpation, and passive and active range-of-motion testing of the shoulder, with attention to scapular dyskinesia and hyperlaxity or instability of the glenohumeral joint. Strength is tested in comparison to the opposite side. In sub-acromial impingement syndrome, weakness mainly affects abduction or external rotation. Testing includes the active and passive range of motion, isometric contraction testing for the selective determination of strength in internal and external

rotation and in abduction, and additional impingement tests. The sensitivity and specificity of such tests is low individually, but, taken together, they are indispensable for the differential diagnosis. Examining techniques are summarized in Table 1.

Although impingement symptoms may arise following trauma, the pain more typically develops insidiously over a period of weeks to months. The pain is typically localized to the anterolateral acromion and frequently radiates to the lateral mid-humerus. Patients usually complain of pain at night, exacerbated by lying on the involved shoulder, or sleeping with the arm overhead. Normal daily activities such as combing one's hair or reaching up into a cupboard become painful, and a general loss of strength may be noted. Onset of shoulder pain and weakness following a fall in an individual over 40 years of age should raise concern for a complete tear of the rotator cuff (Garving et al, 2017).

2.7.1 The tests

A thorough examination of the neck and shoulder is

Table 1: Examination for Shoulder Impingement Syndrome.

Hawkins test	Positive when pain arises on maximal internal rotation of the arm in 90° of anteversion with the elbow flexed. This narrows the subacromial space between the greater tubercle and the coracoacromial ligament, causing pain.
Neers sign	One hand fixes the scapula while the other elevates and internally rotates the arm. This causes painful contact of the greater tubercle with the roof of the shoulder joint.
Jobe test	Both of the patient's arms are held in 90° of abduction, 45° of flexion, and internal rotation. The patient tries to elevate the arms further against the examiner's marked resistance.
Painful arch	Pain on abduction, with extended elbow, in the scapular plane between 60° and 120° indicates pathology in the subacromial space.

critical to properly diagnosing SIS. Strength testing of the upper extremities as well as neck and shoulder ranges of motion should be carefully assessed. In SIS, active and passive shoulder range of motion is typically normal. The muscles of the rotator cuff are

best isolated with 3 separate maneuvers. To isolate the subscapularis, the patient places their hand behind the back and attempts to push away the examiner's hand, a maneuver called the lift-off test. Next, with the arms at the sides and the elbows flexed, the examiner resists the patient in external rotation of the shoulder. Next, to isolate the supraspinatus, which may be painful with SIS, the patient abducts the arms to 90°, forward flexes to 30°, and internally rotates each humerus so that the thumbs are pointed to the floor. A downward force is then applied to the forearms as the patient resists (Garving et al, 2017).

Two provocative examination techniques are highly sensitive but not very specific for diagnosing SIS. Neer's sign elicits pain with maximum passive shoulder elevation and internal rotation while the scapula is stabilized (Dong W et al, 2015). Hawkins sign is pain with passive forward elevation to 90° and maximum internal rotation (Garving et al, 2017). These 2 tests have a negative predictive value of greater than 90% when combined (Burbank et al, 2008). Marked rotator cuff weakness with positive impingement signs may indicate a complete cuff rupture. The Neer impingement test involves injecting the subacromial space with 10 mL of local anesthetic and observing an amelioration of pain with these provocative tests (Garving et al, 2017).

2.8 Diagnostic And Evaluation

Clinical history-taking and a thorough physical examination are the basis of the diagnostic assessment. The diagnostic sensitivity of physical examination is 90%. Imaging studies (initially, plain x-rays) are indispensable for differential diagnosis and for the exclusion of calcific tendinitis or arthritic changes. If the patient has had a circumscribed functional limitation or persistent pain for 6 weeks or more despite the usually adequate analgesia and physical therapy, further imaging studies and referral to a specialist are recommended (Garving et al, 2017).

2.8.1 Differential Diagnosis

Narrowing the etiology of shoulder pain can be difficult as a number of conditions often coexist in older individuals. The etiology of adhesive capsulitis is unknown, although thought to be inflammatory in nature. The disease is more commonly encountered among women in their 50s and 60s. It is 5 times

more likely to occur in patients with diabetes mellitus² and has been associated with hypothyroidism. Adhesive capsulitis often presents with unremitting shoulder pain at rest, and early stages of adhesive capsulitis may present much like impingement syndrome. Later, patients will develop progressive loss of motion, with loss of internal rotation an early sign of the motion loss. Patients with adhesive capsulitis will be limited in both active and passive ranges of motion, particularly in contrast to SIS, where passive motion is unrestricted (Garving et al, 2017).

Cervical radiculopathy may present with unilateral shoulder pain. This can be particularly difficult to sort out in older patients who may have both rotator cuff pathology and cervical spine osteoarthritis. The patient with shoulder pain of a cervical origin may have pain and spasm in the trapezius muscles and a limited neck range of motion. They may also experience pain, numbness, or paresthesia radiating to the arm and hand. Symptoms may be provoked by hyperextension and lateral rotation of the neck (Spurling's maneuver). A key historical detail may be that pain is alleviated when the forearm is rested above the head (Garving et al, 2017).

Degenerative changes within the acromioclavicular (AC) joint and osteolysis of the distal clavicle are often found in individuals with a history of heavy labor or weightlifting but may occur in anyone. The pain may be present over the AC joint itself or be referred to the upper shoulder and neck. Sleeping on the affected side and overhead movements exacerbate the symptoms. Physical examination typically confirms the diagnosis with marked tenderness over the AC joint and pain with compression of the joint through adduction of the elevated arm. Osteoarthritis of the glenohumeral joint presents with a painful diminished range of motion. Arthritic changes in either joint are apparent on radiographs (Garving et al, 2017).

3 CONCLUSION

The natural course of SIS is poorly described, but evidence suggests that the condition is not self-limiting (Garving et al, 2017). The initial management of shoulder impingement has traditionally included medical rehabilitation program (therapeutic exercise and modalities), nonsteroidal anti-inflammatory drugs (NSAIDs), and corticosteroid injection. Medical rehabilitation program (therapeutic exercise and modalities) is frequently implemented to lessen pain and improve function in SIS. In addition to medical rehabilitation

programs (therapeutic exercise and modalities) and medications, activity and workplace modifications must be discussed. Patients should attempt to discontinue overhead activities until symptoms diminish. It may be helpful to discuss “living within a window” in which they consciously attempt to keep their hands within an area in front of their body during activity. The “window” should be from chest to waist and 2 to 3 feet wide, allowing the patient to avoid reaching over- head, away from the body, or

behind the back, all of which will exacerbate their symptoms (Garving et al, 2017).

Bearing in mind that the literature offers few truly well-conducted trials regarding the management of SIS, we present an algorithm of our recommended management of SIS based upon a synthesis of the best available literature. Note that an MRI is not recommended until at least a 6-week therapeutic trial has been implemented unless a complete rupture is suspected (Garving et al, 2017).

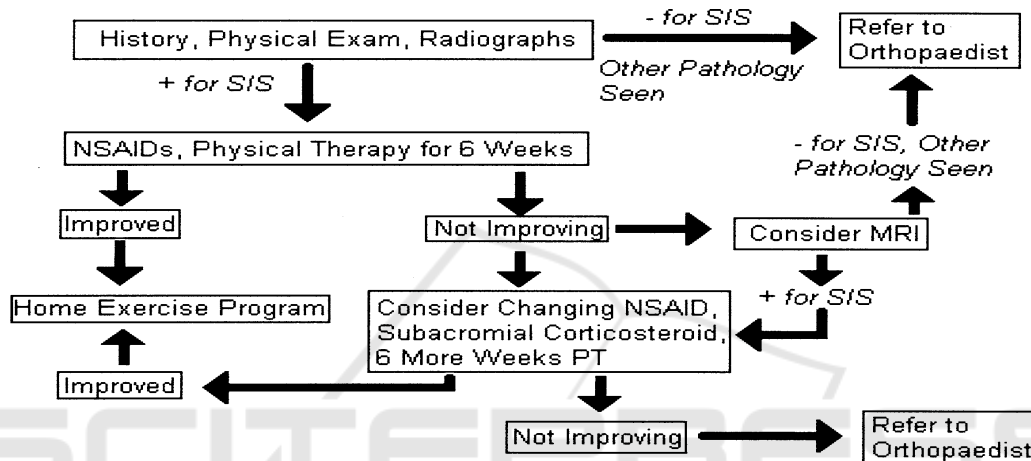


Figure 13: Treatment algorithm for subacromial impingement syndrome. SIS = subacromial impingement syndrome; NSAID = nonsteroidal anti-inflammatory drug; PT = physical therapy.

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