Subacromial Impingement Syndrome – Secondary Impingement

By: Kevin Bond

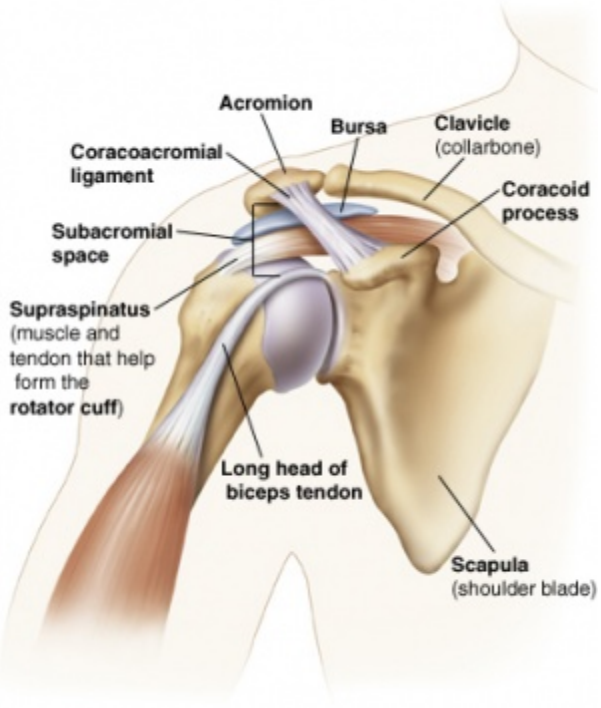
Introduction[1,2]

Subacromial impingement syndrome (SIS) is the most common pathology related to the shoulder joint.[1] In fact, 44-65% of all shoulder pain complaints are diagnosed as SIS.[1] The syndrome presents in many different ways and often has multiple and varying etiologies, making SIS difficult to diagnose. Because of the wide variety of potential mechanisms that cause SIS, the syndrome is organized into two separate categories: primary and secondary impingement.[2] Primary impingement deals with internal or external structural/anatomical deformities causing mechanical compromise of the subacromial space and its surrounding soft tissues.[2] In other words, the impingement in the subacromial space is the main problem. Examples of primary impingement include: bony deformities of the humeral head (CAM) or bony glenoid (Pincer), rotator cuff failure, calcific tendonitis, or shape of the acromion.[2] Each of these examples primarily decrease the subacromial space causing SIS. Secondary impingement indicates something is causing the impingement of the subacromial space. This can further be broken down into secondary external and internal impingement. This paper will discuss

secondary impingement and the relevant anatomy, pathomechanics, assessment, and intervention.

Anatomy[3,4]

In order to understand the pathomechanics, evaluation, and treatment for SIS, the relevant anatomical features of the shoulder need to be known. The bony structures that make up the shoulder girdle and surrounding upper quarter include: upper thoracic vertebrae, first/second ribs, manubrium, scapula (including acromion and coracoid process), clavicle, and the humerus.[3] These bones come together in different spots to create the various joints in the shoulder girdle. The glenohumeral joint (GHJ) is a ball and socket joint connecting the humerus into the bony glenoid cavity of the scapula. The glenoid cavity faces laterally, forward, and superiorly with an angulation of 75 degrees.[3] The head of the humerus is much larger than the glenoid cavity, but the fibrocartilaginous glenoid labrum deepens the cavity and creates more surface area for the head to operate.[3] Superior to the GHJ is the acromion, or



bony process stemming from the scapula. The space between the acromion and the humeral head as it begins to form the GHJ is known as the subacromial space. The distal clavicle and acromion meet together to form the planar acriomioclavicular joint (ACJ). The proximal clavicle forms the saddle sternoclavicular joint (SCJ) with the sternum.[3] The scapulothoracic joint, which is more of a mechanism than a joint, connects the scapula to the thorax. Other important joints not directly linked to the shoulder but essential in describing secondary impingement are the thoracic vertebral joints, cervical vertebral joints, and rib articulations with the sternum and spine.

Surrounding the GHJ is the joint capsule, which is a thin and lax band of ligaments surrounding the actual joint.[3] The ligaments are in a medial twist at rest given the forward facing orientation of the GHJ. Providing support to the capsular ligaments are the glenohumeral ligaments including: the superior, inferior, and middle glenohumeral ligaments.[3] The transverse humeral ligament overlays the bicipital groove just inferior to the humeral head on the humerus and creates a tunnel for the long head of the biceps. The coracoacromial ligament connects the coracoid process and the acromion superior to the GHJ.[3] The trapezoid ligament and conoid ligament connect the coracoid process to the clavicle. Surrounding the ACJ are the

ACJ capsule and ligament.[3] Sternoclavicular joint capsule and anterior ligament are the most important players at the SCJ.

The muscles surrounding the shoulder girdle are all essential for overall biomechanical function of the shoulder joint.[3] Working to stabilize the GHJ are the rotator cuff muscles including: the subscapularis, infraspinatus, supraspinatus, and teres minor.[3] These muscles all originate on the scapula and insert on the greater tuberosity of the humerus. The supraspinatus assists the humerus in abduction and its tendon travels directly through the subacromial space towards its attachment site on the humerus. The deltoid muscle covers the proximal humerus and greatly assists with flexion, extension, and abduction of the GHJ. Along with the subscapularis, powerful internal rotators of the shoulder include: the pectoralis major (horizontal adduction), latissimus dorsi, and teres major. Another muscle assisting with humeral flexion is the biceps. The trapezius muscle is a powerful controlling upper quarter muscle that works to elevate, retract, protract, and depress the scapula.[4] Other scapular stabilizers include: the serratus anterior, rhomboids (major and minor), and levator scapula. Potential players in secondary impingement pathology include the diaphragm, intercostal muscles, abdominals, and erector spinae muscles.

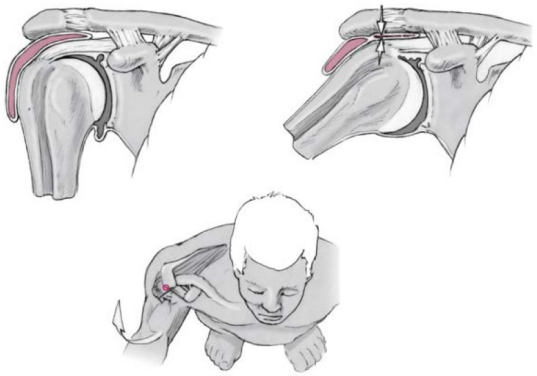
Other anatomical features that may be influencing, or influenced by, secondary impingement include: bursas, nervous tissue, and vascular tissue. A bursa lays directly underneath the acromion, sitting in the subacromial space. Nerves originating from the cervical spine innervate the muscles in the upper quarter and play a crucial role in neuromuscular control. A majority of the nerves and vascular tissue travel underneath the anterior shoulder anatomy to get out into the arm.

Pathomechanics[3,4,6,7]

Full humeral elevation at the shoulder involves four different joints working together. The process of lifting the arm up and over the head is referred to as scapulohumeral rhythm and includes the GHJ, scapulothoracic joint, ACJ, and SCJ. The GHJ is responsible for 120 degree of total scapulohumeral rhythm and is the primary mover of the first 30-60 degrees of elevation.[4] In addition, the greater tuberosity of the humerus will begin to impede on the coracoacromial arch. External rotation of the humerus, by means of the infraspinatus and teres minor, is required to clear this potential bone-on-bone contact. [4] After this point, the scapulothoracic joint is required to move in sync with the GHJ. For every 2 degrees of GHJ motion, there is 1 degree of scapulothoracic movement.[3] During this process, the scapula needs to be stabilized against the thoracic cavity by the scapular stabilizing

muscles. The scapula performs protraction and upward rotation in order to assist with the motion at the GHJ for a total of 60 degrees.[3] The coracoclavicular ligament will work to limit the amount of scapular motion during elevation. In order to achieve the full 60 degrees, the clavicle has to rotate. [3] The ACJ is directly related to scapular movement, but because of its planar joint surface, it does not have as much motion.[3] At the saddle shaped SCJ joint, more motion can occur as the clavicle can roll and slide more freely in this joint to allow the scapula to continue its upward rotation. This is an extremely delicate mechanism that can be disrupted causing pain or impingement. There are thought to be multiple mechanisms that disrupt this process, and no single etiology. Four different mechanisms that may cause SIS include: muscle imbalance/weakness, abnormal/restricted joint movement, poor posture and overuse.

Muscles perform the motions involved in scapulohumeral rhythm. The rotator cuff muscles are designed to stabilize the glenohumeral joint and perform the necessary actions to achieve elevation. In combination with the deltoid, these muscles form a force couple. [4] As the supraspinatus and deltoid muscles



abduct/flex the shoulder, the other rotator cuff muscles work to depress the humeral head and prevent superior translation into the subacromial space. [4] Weakness or imbalance of these muscles, often times seen with the external rotators, can lead to poor stability of the humeral head in any given direction. [4] As a result, impingement will occur during elevation of the humerus. The scapulothoracic joint works as a force couple with the GHJ. The scapular stabilizers need to keep the scapula fixed onto the thorax. For example, the trapezius and serratus anterior work to perform upward rotation and protraction of the scapula. Weakness of the serratus anterior can lead to poor scapular control with humeral elevation. [4] This will lead to abnormal translation of the humeral head inside the GHJ and impingement. In addition to muscles that perform the mechanism as agonists, there are muscles that act as agonists like the rhomboids and pectoralis minor in the scenario above. These muscles provide the guidance and control of the upward rotation performed by the trapezius and serratus anterior. Abnormal motion at the scapula is referred to as scapular dyskinesia and is a common with impingement. [3] Any loss or imbalance in muscle function surrounding the GHJ or scapulothoracic joint will affect all movement at the GHJ and can lead to impingement of the humeral head into the coracoacromial arch.

The muscles involved may not be weak at all, but they could be tight or shortened. For example, the shoulder external rotators are required to externally rotate the shoulder as the greater tuberosity comes in close contact with the coracoacromial arch. Tightness of the antagonist muscles, the internal rotators, could limit the ability of the external rotators to perform this necessary action.[4] Limiting this action could cause some impingement at the shoulder. Other tight soft tissues in the area could cause impingement or limited joint mobility. Certain portions of the GHJ capsule could be tight, often the posterior capsule, which would promote more motion in another direction and lead to abnormal translation of the humeral head in the GHJ.[3] Reduced movement could also be caused by poor joint mobility. There are four joints involved in humeral elevation and if any of these joints cannot provide the motion, other joints will compensate. Tight soft tissues at the SCJ would not allow the proper scapular movement to assist the GHJ, for example.

Poor posture is commonly seen in the generally population and can have an impact on shoulder impingement.[6] Some of the key features of poor posture include: kyphosis of the thoracic spine, forward head position, and rounded shoulders.[6] Kyphosis causes altered, or limited motion of the scapula because of the change in thoracic spine extension.[6] As discussed

above, anything that impedes motion at any of the joints involved in humeral elevation can lead to impingement. Forward head posture has been shown to cause a narrowing of the subacromial space.[6] The posture pulls and lengthens the trapezius muscle and scapula itself effecting the length tension relationship of the supraspinatus. As discussed, this tendon runs right through the subacromial space and is prone to inflammation and degeneration with forward head posture.[6] Rounded shoulders could be the result of tight soft tissues, such as the pectoralis minor pulling on the coracoid process. The shortened position of the pectoralis minor can create a poor length tension relationship here and on the antagonist muscles posteriorly. The external rotators, because of their attachment site, would be left in a lengthened and weakened positioned. This would cause poor control of the GHJ and superior translation of the humeral head into the subacromial space. Poor posture affects joints, muscles, and ligaments surrounding the shoulder joints and is therefore very influential to SIS.

Overuse is another cause of secondary impingement. This is typically seen with overhead athletes or more active people doing overhead activities such as painting or stocking in a warehouse. In the literature, this type of impingement is intrinsic secondary impingement, or posterior impingement.[7] It is defined as contact of the posterior glenoid with the posterior tendons of

the rotator cuff.[7] Overhead athletes are required to perform extremes of external rotation to create the joint forces to, for example, throw a 100mph fastball. Pain with this mechanism occurs during compression of the supraspinatus and infraspinatus tendons in the GHJ/subacromial space due to the relatively externally rotated greater tuberosity.[7] Because the humerus and scapula are not operating in the same plane of motion now at the late cocking phase of throwing, there will be impingement of the rotator cuff tendons shown above. This mechanism will lead to compression and fraying of the tendon causing the inflammatory process. Inflammation takes up space and will encroach on the subacromial space.[7] This is also the mechanism associated with just general overuse. Tendons of the rotator cuff put in compromising positions because of poor posture, imbalance, weakness, etc can lead to fraying/degeneration of the tendon itself. Inflammation can also occur due to the bursa being irritated or other soft tissues in the area. In general, all of the mechanisms discussed in this section will lead to inflammation of the soft tissues in the GHJ and subacromial space.[4] After inflammation is present in the space, there will be pain with overhead activity and with these secondary causes still in place, the soft tissue will not be allowed to heal properly. This can lead to fibrotic tissue and bony spurs in the area and chronic SIS.[4]

Assessment [4, 8, 5]

Proper assessment of the secondary causes of impingement is necessary by physical therapists in order to create a plan of care to address these dysfunctions. The diagnostic sensitivity for patients with SIS is 90%, as in the syndrome can be ruled OUT very well.[5] As with all diagnostic criteria, there is a gold standard. Diagnostic imaging of the subacromial space via MRI, X-ray, etc is the gold standard for detecting space narrowing.[5] It is the physical therapists job to discover what is actually causing the space narrowing, assuming no primary mechanisms are the problem. History taking and a thorough physical examination are the keys to proper assessment.[5] Useful information from the subjective portion of the assessment includes: when it started, how it started, where the pain is, what actions cause the pain, what actions ease the pain, nature of the pain, other orthopedic problems, and what they do for work.[3] Also necessary here is noting any red flags or symptoms that may not be musculoskeletal in nature. Often times, a diagnosis can be made by simply listening to the patient speak.

Next is the physical examination. As discussed in the section above, posture can be a main cause of secondary impingement. Observation of the patient is first and starts immediately upon seeing the patient and during the

interview; how are they sitting, what is their posture, etc. After is a more thorough examination of posture and anatomical features. Features like scapular winging, elevated shoulder, depressed shoulder, and asymmetry are key indicators for secondary impingement. It is this author's belief that all shoulders are considered cervical pathology until proven otherwise. Thus, a cervical screen is necessary including: active range of motion and whether neck motions affect shoulder pain. Upper quarter range of motion needs to be assessed to determine any limitations in range of motion and which motions hurt.[3] Noting the amount of motion and the quality of that motion here is indicated. It is necessary to assess the motion from all angles to observe any biomechanical problems. From a posterior view, scapular dyskinesia can be assessed during humeral elevation. If patients have extreme pain with active motions, passive range of motion in supine would be indicated in order to get a baseline range of motion measurement for all shoulder motions. For patients with SIS, passive range of motions will often be painful but not limited because no mechanisms are impeding full motion. General manual muscle testing could be used to determine: which muscles may be weak or weaker than others, if isometric contraction of that muscle/tendon unit causes pain, and if this is the pain they are coming in for. [3] The supraspinatus is often the most problematic muscle in SIS patients.

Palpation of soft tissues in the area will help reveal any tenderness indicating inflammation or irritation of that soft tissue. Palpation may be provocative of pain, but special tests are more provocative in many cases. [3]

There are many special tests for the shoulder, but there are specific tests for subacromial impingement and rotator cuff impingement/pathology. The SIS cluster of tests includes: painful arc, external rotation resisted, Hawkins Kennedy, Neer test, and empty can.[8] Each of these tests places the subacromial space in a compromised position. A positive result on at least 3/5 of these tests indicates impingement syndrome is a differential diagnosis. In addition to these special tests, there are other tests specifically for rotator cuff muscles.[8] Yokum test places the shoulder in a compromised position and can indicate impingement of the rotator cuff. Subscapularis tests include the belly press, lift off sign, and bear hug.[8] The supraspinatus tests include the drop arm test (more for rotator cuff tear), full can, and empty can. External rotation lag sign is used to assess the strength/integrity of the infraspinatus, while the horn blower sign assesses the teres minor. These tests can be used to further diagnose which tissue is the main problem. Assessing instability of the shoulder with the apprehension/relocation test, anterior load and shift test, posterior apprehension, jerk test, biceps load test (1 and 2), crank test, and O'brien test could be useful. [8] Instability of the

shoulder could cause the translation of the humeral head into the subacromial space leading to chronic inflammation of the tendons.

While special tests are useful and necessary during examination, they are not always sensitive or specific. In other words, some of these tests are not very reliable and using the other information from the examination can provide better information for treatment. The goal of the assessment is to collect objective data from the patient in order to determine a root cause. The special tests can help to rule in or out certain pathologies. Physical therapists will want to take the other aspects of examination and determine what the mechanism of injury is. All the data gathered from this detailed assessment hopefully lead to a clear diagnosis, or short differential diagnosis list, and specific mechanisms that could potentially be causing the impingement. From here, we use interventions to help address the problems.

Intervention [3,4,7,8,9,10,11,12]

A detailed assessment of the mechanisms of injury can implicate the various interventions that can be implemented to help treat SIS related to secondary mechanisms. This author follows a certain train of thought when treating musculoskeletal pathologies: pain, motion, strength, and control. First and foremost is reducing pain of the injured tissue. Avoiding the painful activities, which is typically overhead activity, is the first step. This

may mean activity modification or reduced participation in sport or work. Education on how the repetitive painful stimulus is continuously causing inflammation of the subacromial space would be necessary for these patients because they may not like the idea of reduced activity. However, this is a necessary step to allow the tissue to heal well and correctly. Immobilization is NOT advised because this can ruin the integrity/strength of the soft tissue. Poor posture could be the mechanism leading to impingement. Postural re-education exercises/stretches such as scapular retractions, cervical retractions, wall stretches for the pectoralis minor, or thoracic mobilizations are very useful interventions. [3,8] Because these exercises do not involve elevation of the GHJ to a point that produces pain, they can be done as starter exercises to help reduce pain.

In the clinic, modalities and stretching/manual therapy have been shown to be beneficial for patients with SIS.[9] Modalities include: ice, heat, low-level laser therapy, dry-needling, and taping for external support. Low-level laser therapy has been shown, in a study by Haslerud et al, to help reduce pain associated with tendinopathy in the shoulder.[10] They also stated that there are benefits in combination with other physical therapy interventions.[10] In a randomized control trial performed by Shih et al, they looked at kinesiology taping for pain control, joint position sense, and

movement control.[11] They found that there were positive benefits, less errors associated with joint position sense, after placing KT tape to the upper and lower trapezius muscles.[11] The external support of the tape enabled the shoulder to operate optimally biomechanically. As patients are working to decrease pain, using KT tape on the shoulder in this way could help to reduce some pain by repositioning the joint into its optimal position with movement. In a case study by Passigli et al, they demonstrated the effects of dry-needling on a patient with posterior impingement.[12] They found that dry-needling was immensely beneficial in reducing pain by releasing trigger points in muscles around the shoulder.[12] Although this is not the best evidence, it indicates that dry-needling is an option to help reduce pain.

Manual therapy techniques include: soft tissue massage, joint mobilizations, and stretching.[3] As discussed in the pathomechanics section, there could be restrictions from soft tissues. The assessment will help to reveal which soft tissues may be influencing scapulohumeral rhythm.[3] Stretching tight soft tissues in combination with postural re-education exercises can be beneficial for patients with impingement. Capsular stretching can be used to help free up mobility, specifically in patients with posterior impingement.[7] Releasing trigger points and tight soft tissues with manual therapy techniques could help to improve muscle kinematics and

relieve pain at the subacromial space. Cross-friction massage to the tendons in question could help to relieve built up scar tissue that may be impeding on the subacromial space. There is evidence to support all of these interventions as a way to reduce pain.[9] Joint mobilizations could refer to any joint in the upper quarter or spine. For patients with extreme thoracic kyphosis, central posterior-anterior mobilizations can help reduce the effects of kyphosis and thus decrease the space narrowing of the subacromial space.[3] Superior-inferior grade I and II mobilizations can be used for pain control at the GHJ and relieve the pressure in the subacromial space.[3]

With pain under control, the joints will begin to free up and increase range of motion at the shoulder. Continued work on range of motion through stretching and pain free joint mobilizations is necessary, but significant strengthening can begin. Based on the assessment, the physical therapist should determine which muscles are weaker or weak in the rotator cuff or scapular stabilizers. These muscles should be the primary target during initial strengthening. For example, the external rotators are often weak in patients with secondary impingement. Performing isolated external rotation exercises are beneficial for strengthening. [3] If the infraspinatus is the primary culprit of muscular imbalance, strengthening it should improve the imbalance and reduce the abnormal translation of the humeral head into the

subacromial space. Other rotator cuff exercises include: internal rotation, flexion, extension, and abduction in various positions. [3] Performing these simple motions are necessary during the initial strengthening program to help improve GHJ stability.[4] Postural strengthening exercises are all designed to help reduce the kyphosis, rounded shoulders, or forward head posture and include: upright rows, bilateral external rotation, scapular squeezes, standing rows, horizontal abduction, middle trap pull downs, etc. [3] Not all of these strengthening exercises should be performed. It depends entirely on the assessment of tissue needs the treatment or which muscle is weaker. Strengthening interventions are extremely numerous. This author suggests starting with simple strengthening exercises like the ones listed above and progressing to more complicated exercises. Beginning with 2 sets of 10 for strengthening exercises and progressing to 3 sets of 10 or 2 sets of 15 would be indicated. [3,8] As patients begin to get stronger, mobility and pain should continue to improve and decline, respectively.

Last, patients can do more complicated neuromuscular control exercises. Athletes will want to begin sport specific exercises in functional overhead positions. Challenging joint proprioception with perturbations performed by the PT, wall ABCs with a ball or theraband, or proprioceptive neuromuscular facilitation exercises would be necessary to improve the

GHJ's dynamic stability.[3] These challenge to rotator cuff to stabilize the humeral head in the joint. Performing these exercises in various functional positions is necessary. Scapular neuromuscular control exercises need to be performed to improve scapular mobility/stability during humeral elevation.[3] Planks with perturbations, wall climbs with therabands, push-up plus, or wall ABCs with a ball could be used to challenge the scapula in various functional positions.[3] These control exercises are the last aspect of intervention and should be done in the absence of pain and with full range of motion. Creativity and innovation for these exercises is endless, but the core concepts discussed in this paper are the basis for implementing intervention. The idea behind these exercises is to return patients to participation in sport, work, or activities of daily living and reduce the likelihood of re-injury.

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