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Review paper

Anatomical and biomechanical mechanisms of subacromial impingement syndrome

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Abstract

Subacromial impingement syndrome is the most common disorder of the shoulder, resulting in functional loss and disability in the patients that it affects. This musculoskeletal disorder affects the structures of the subacromial space, which are the tendons of the rotator cuff and the subacromial bursa. Subacromial impingement syndrome appears to result from a variety of factors. Evidence exists to support the presence of the anatomical factors of inflammation of the tendons and bursa, degeneration of the tendons, weak or dysfunctional rotator cuff musculature, weak or dysfunctional scapular musculature, posterior glenohumeral capsule tightness, postural dysfunctions of the spinal column and scapula and bony or soft tissue abnormalities of the borders of the subacromial outlet. These entities may lead to or cause dysfunctional glenohumeral and scapulothoracic movement patterns. These various mechanisms, singularly or in combination may cause subacromial impingement syndrome.

Keywords: Subacromial impingement syndrome; Shoulder; Review

1. Introduction

Subacromial impingement syndrome (SAIS) of the shoulder is the most common disorder of the shoulder, accounting for 44–65% of all complaints of shoulder pain during a physician's office visit (van der Windt et al., 1995, 1996; Vecchio et al., 1995). This disorder can present in many forms, ranging from inflammation to degeneration of the bursa and rotator cuff tendons of the subacromial space. SAIS may lead to a full-thickness tear of the rotator cuff tendons and degenerative joint disease of the joints of the shoulder girdle (Neer, 1972; Fu et al., 1991; Bigliani and Levine, 1997; Budoff et al., 1998). The consequences of SAIS are functional loss and disability (Vaz et al., 2000; Ludewig and Cook, 2000; Brox et al., 1999; Nordt et al., 1999; O'Connor et al., 1999; Chipchase et al., 2000; Lukasiewicz et al., 1999; Beaton and Richards, 1998; Beaton and Richards, 1996; Brox et al., 1993). The cost of care for this disorder is variable, because several treatment options are typically explored before a successful outcome is achieved (van der Windt et al., 1995, 1996; Vecchio et al., 1995). The selection of an effective treatment regimen often proves difficult, because of the multi-factorial nature of SAIS.

The subacromial space is defined by the humeral head inferiorly, the anterior edge and under surface of the anterior third of the acromion, coracoacromial ligament and the acromioclavicular joint superiorly (Neer, 1972). The tissues that occupy the subacromial space are the supraspinatus tendon, subacromial bursa, long head of the biceps brachii tendon, and the capsule of the shoulder joint. Any or all of these structures may be affected with SAIS.

SAIS is an encroachment of the subacromial tissues as a result of the narrowing of the subacromial space. There are two predominate mechanistic theories as to the cause of the space narrowing in SAIS. The first, labeled 'intrinsic impingement', theorizes that partial or full thickness tendon tears occur as a result of the degenerative process that occurs over time with overuse, tension overload, or trauma of the tendons (Budoff et al.,

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1998; Uhthoff et al., 1998). Osteophytes, acromial changes, muscle imbalances and weakness, and altered kinematics leading to impingement will subsequently follow. An alternative theory is that of 'extrinsic impingement', where inflammation and degeneration of the tendon occur as a result of mechanical compression by some structure external to the tendon (Neer, 1972; Bigliani and Levine, 1997). Potential extrinsic mechanics that may lead to SAIS are faulty posture, altered scapular or glenohumeral kinematics, posterior capsular tightness, and acromial or coracoacromial arch pathology. The question is, 'which comes first, tendon degeneration or changes external to the tendon'? By the time a patient with SAIS seeks health care, the typical examination findings reveal tendon pathology in some form and the presence of one or more extrinsic factors such as osteophytes or muscle weakness. Consequently, it is difficult to ascertain which occurred first.

The classification of SAIS was first developed based upon the degree of injury to the tissues of the subacromial space. Neer (1983) defined this disorder as a mechanical compression injury of the tissues of the subacromial space, and proposed three progressive categories. Stage I, known as the edema and hemorrhage stage is found in patients under 25 years of age with a history of overhead use in sports or work. Progression to stage II is defined by further deterioration of the tendon and bursa, and found in 25–40 years old patients. Further progression of the disorder results in the development of stage III, characterized by bone spurs and partial or full-thickness tendon rupture affecting those over 40 years of age.

The staging by described by Neer (1983) cannot classify patients into discrete categories. Other classification systems have emerged in an attempt to logically categorize the potential mechanistic factors of SAIS. The factors have been classified as direct or indirect, intrinsic or extrinsic, primary or secondary, static or dynamic (Fu et al., 1991; Bigliani and Levine, 1997). Categorization of the anatomical factors is based upon their location in the subacromial space, their ability to generate force, or whether or not there is a superimposed pathology. There is overlap with all of these systems and their terminology is not synonymous, therefore these systems cannot be used interchangeably to present the anatomical and biomechanical mechanisms of SAIS.

1.1. Glenohumeral joint kinematics

The glenohumeral joint possesses six degrees of freedom, three rotations and three translations. With simulated cadaver or active in vivo glenohumeral abduction in the scapular plane (approximately 30–40° anterior to the frontal plane), the humerus concomitantly externally rotates (Browne et al., 1990; An et al., 1991; Pearl et al., 1992). External rotation is important for clearance of the greater tuberosity and its associated tissues as it passes under the coracoacromial arch, as well as for relaxation of the capsular ligamentous constraints to allow maximum glenohumeral elevation (Browne et al., 1990; An et al., 1991). Limited glenohumeral external rotation during elevation has been hypothesized to lead to SAIS (Browne et al., 1990), however no evidence is available to support this postulate in patients with SAIS.

Translation of the humeral head in the magnitude of 1–3 mm in the superior direction, occurs in the first 30–60° of active glenohumeral scapular plane elevation (Poppen and Walker, 1976; Chen et al., 1999; Ludewig and Cook, 2002) or during simulated elevation in the scapular plane (Kelkar et al., 1992; Thompson et al., 1996). Conversely, one study demonstrated inferior translation of 0.7 mm during the 30–60° phase of glenohumeral abduction, which was performed with the subjects lying supine (on their back) and thus most likely did not similarly recruit muscle activity (Eisenhart-Rothe et al., 2002). After the initial phase of elevation in the scapular plane or frontal plane abduction, the humeral head remains somewhat centered on the glenoid cavity with fluctuations between inferior and superior translations of typically less than 1 mm (Poppen and Walker, 1976; Ludewig and Cook, 2002; Kelkar et al., 1992; Thompson et al., 1996; Eisenhart-Rothe et al., 2002; Sharkey and Marder, 1995; Deutsch et al., 1996; McMahon et al., 1995; Wuelker et al., 1994b; Paletta et al., 1997; Yamaguchi et al., 2000; Graichen et al., 2000). The glenohumeral joint demonstrates essentially ball and socket kinematics above approximately 60° of glenohumeral elevation. During passive glenohumeral motion, limited evidence demonstrates that during 30–60° superior translation occurs, and then from 60–150° inferior translation was the dominant motion (Graichen et al., 2000).

Superior humeral head translation that occurs during the initial phase of elevation may in part be due to the deltoid. With the arm at the side, the deltoid's line of pull is such that in addition to its rotational torque, it also produces a translatory force in the superior direction. Conversely, the translatory force component of the supraspinatus is compressive in nature, which helps stabilize the joint (Thompson et al., 1996; Howell et al., 1986). Therefore, the superior translation that occurs during the initial phase of elevation appears to be due in part to the cranially directed pull on the head of the humerus by the deltoid muscle (Kronberg et al., 1990).

Humeral head translations in the anterior–posterior directions have been less well investigated. Anterior humeral head translations in the magnitude of 2–5 mm have been demonstrated during passive (Harryman et al., 1990a,b, 1992) and simulated active (Wuelker et al., 1994b) glenohumeral flexion. During active glenohumeral flexion, anterior humeral head translation of less than

1 mm occurs over the course of motion (Wuelker et al., 1994b; Harryman et al., 1990a,b, 1992). Several studies have examined translations in specific phases of elevation, revealing that in the first 30–60° phase of scapular plane abduction 0.7–2.7 mm of anterior translation, 0–1.5 mm of posterior translation in the 60–90° phase, and 4.5 mm of posterior translation in the 90–120° phase of scapular plane elevation (Ludewig and Cook, 2002; Eisenhart-Rothe et al., 2002; Graichen et al., 2000). Conversely, one study demonstrated anterior translation of approximately 1 mm in the final phase of 90–120° of elevation (Graichen et al., 2000).

Excessive superior or anterior humeral head translations have been hypothesized to lead to SAIS and rotator cuff degeneration (Ludewig and Cook, 2002; Deutsch et al., 1996; Paletta et al., 1997). Limited evidence provides support for this theory. During active glenohumeral elevation, increased superior humeral head translation of 1–1.5 mm (Poppen and Walker, 1976; Deutsch et al., 1996) and increased anterior translation of approximately 3 mm (Ludewig and Cook, 2002) has been demonstrated in patients with SAIS. Increased superior humeral head translations have also been demonstrated with rotator cuff tendon degeneration during active or simulated active glenohumeral elevation of 1.5–5 mm (Poppen and Walker, 1976; Thompson et al., 1996; Deutsch et al., 1996; Paletta et al., 1997; Yamaguchi et al., 2000). Excessive superior translations were also demonstrated (Chen et al., 1999; Sharkey and Marder, 1995) with weakness or induced fatigue of the deltoid and rotator cuff in healthy subjects during abduction in the coronal or scapular plane. The amounts of excessive anterior and superior translations range from 1 to 5 mm, which would appear to be potentially insignificant due to their small magnitude. However, because the subacromial space is small in volume and contains the subacromial structures, there is little room for ''error''.

The height of the subacromial space, from the head of the humerus to the coracoacromial arch, is only 1.0–1.5 cm as seen on radiographs (Flatow et al., 1994). Changes of this space occur in subjects with healthy shoulders; a decrease in the width of the acromio–humeral interval occurs during glenohumeral abduction (Flatow et al., 1994; Graichen et al., 1999a, 2001) and an increase in the contact between the inferior acromion and underlying subacromial tissues occurs during glenohumeral abduction and flexion (Flatow et al., 1994; Brossmann et al., 1996; Solem-Bertoft et al., 1993). A decrease of 3 mm of the acromio–humeral distance was demonstrated in patients with SAIS as compared to healthy controls, at 90° of isometric glenohumeral abduction (Graichen et al., 1999b). Contact pressure and force in the subacromial space has also been demonstrated to increase during glenohumeral abduction, with the highest subacromial force and contact pressure observed in the mid-range of motion (Nordt et al., 1999; Payne et al., 1997; Wuelker et al., 1994a). Theoretically, these changes in the subacromial space would be accentuated with an increase in the normal superior and anterior humeral head translation, leading to mechanical compression of the tissues of the subacromial space during glenohumeral motion (Fu et al., 1991; Bigliani and Levine, 1997; Flatow et al., 1994; Brossmann et al., 1996).

The long head of the biceps via its attachment on the anterior superior glenoid serves to stabilize the head of the humerus anteriorly and superiorly. Contraction of the biceps muscle has been demonstrated to result in a decrease in superior humeral head translation (Pradhan et al., 2000) and anterior translation (Kumar et al., 1989), as well as a decrease the pressure in the subacromial space (Payne et al., 1997).

Glenohumeral elevation range of motion is decreased in patients with SAIS (Ludewig and Cook, 2000; Lukasiewicz et al., 1999; Greenfield et al., 1995). This may be due, in part to the pain experienced during elevation as a painful arc during glenohumeral elevation is a common finding in these patients (Neer, 1972, 1983). This complaint of pain in the mid-range of glenohumeral abduction corresponds to the highest values of subacromial pressure (Nordt et al., 1999). Additionally, the greatest contact of the rotator cuff and biceps tendon with the coracoacromial arch is in the mid-range of abduction (Flatow et al., 1994; Brossmann et al., 1996; Burns and Whipple, 1993).

1.2. Scapulothoracic articulation kinematics

The scapula and the thoracic cage form the scapulothoracic articulation. This articulation is assessed kinematically either two-dimensionally or three-dimensionally. The joint is typically described with five degrees of freedom, three rotations and two translations however there are multiple ways in which to model scapular motion. Landmark investigations by Inman et al. (1944) and Poppen and Walker (1976) provided the first studies of scapular kinematics. However, these two-dimensional studies were static in nature, asking the subjects to elevate their arm and hold that position while data regarding the position of the scapula and humerus were collected. It is unclear how accurately static data represent dynamic scapular motion. Additionally, the scapula moves in a three-dimensional fashion, so the use of two-dimensional technique cannot fully capture scapular motion.

Three-dimensional studies of scapular kinematics have recently emerged in the literature. The protocol for three-dimensional analysis of scapular motion by van der Helm and Pronk (1995) describe scapular upward rotation occurring about an anterior–posterior axis, with the inferior angle of the scapula moving laterally;

external rotation occurring about a superior–inferior axis, with the lateral border of the scapula moving posteriorly; and posterior tilt occurring about a medial–lateral axis, with the inferior angle moving anteriorly.

Studies of three-dimensional scapular kinematics in asymptomatic shoulders have utilized a variety of techniques that include radiographs, magnetic tracking devices, and electronic digitizers (Ludewig and Cook, 2000; Lukasiewicz et al., 1999; van der Helm and Pronk, 1995; Kondo et al., 1984; Hogfors et al., 1991; Johnson et al., 1993; McQuade et al., 1995; Ludewig et al., 1996; Meskers et al., 1998b; de Groot, 1999; Price et al., 2000; Karduna et al., 2001). From this body of work, a three-dimensional pattern of scapular motion has emerged. Utilizing the terminology previously described by van der Helm and Pronk (1995), the scapula demonstrates a pattern of upward rotation, external rotation, and posterior tilting during glenohumeral elevation. The predominant motion of the scapula is upward rotation, and to a lesser degree scapular external rotation and posterior tilt.

Less well examined are scapular translations, depicted as scapular positions. Scapular positions can be represented by clavicular rotations about the sternoclavicular joint in two different planes: clavicular elevation/depression for superior/inferior translation and clavicular protaction/retraction for anterior/posterior translation. The assumption is made that motion of the clavicle at the sternoclavicular joint will be in direct relationship to scapular translation, because of the interposed rigid bone (clavicle) between these two joints and the lack of significant motion occurring at the acromioclavicular joint (Karduna et al., 2001; McClure et al., 2001). During glenohumeral elevation the clavicle retracts posteriorly and elevates, putting the scapula in essentially a more superior and posterior position (van der Helm and Pronk, 1995; McClure et al., 2001; Meskers et al., 1998a).

A recent three-dimensional study assessed scapular kinematics in vivo (McClure et al., 2001) by measuring scapular movement via two 3/16 mm steel bone pins drilled directly into the scapula of eight healthy subjects, allowing for a more accurate representation of the scapular position and orientation. The results revealed a mean of 50° of scapular upward rotation, 30° of posterior tilting, and 24° of external rotation during scapular plane glenohumeral elevation. For glenohumeral flexion in the coronal plane the results revealed a mean of 46° of scapular upward rotation, 31° of posterior tilting, and 26° of external rotation. A mean of 21 and 20° of clavicular retraction and a mean of 10° and 9° of clavicular elevation was revealed during glenohumeral scapular plane elevation and flexion respectively. The results from the in vivo study (McClure et al., 2001) for scapular upward rotation were in agreement with prior studies that utilized external devices to assess scapular kinematics. However, the results for external rotation and posterior tilt were either under or overestimated by the previous studies.

Altered scapular kinematics have been demonstrated in patients with SAIS (Ludewig and Cook, 2000; Lukasiewicz et al., 1999; Warner et al., 1992; Endo et al., 2001). Warner et al. (1992) demonstrated a pattern of increased scapular winging with glenohumeral elevation, using a Moiré topography technique. This winging pattern appears to represent scapular internal rotation and anterior tilting. Recently, three-dimensional kinematic analysis has demonstrated during glenohumeral elevation decreased posterior tilt (Ludewig and Cook, 2000; Lukasiewicz et al., 1999), upward rotation (Ludewig and Cook, 2000), and external rotation (Ludewig and Cook, 2000). Radiographic assessment at multiple joint angles revealed a decrease in scapular posterior tilt and upward rotation at 90° of glenohumeral elevation, and a decrease in posterior tilt at 45° of glenohumeral elevation (Endo et al., 2001).

Scapular upward rotation results in elevation of the acromion, while posterior tilting elevates the anterior acromion. Both of these scapular motions appear to be important during glenohumeral elevation to prevent impingement at these areas of the acromion (Flatow et al., 1994). Shoulder retraction, of which scapular posterior tilting seems to be a component, has been demonstrated to increase the area of the subacromial space as compared to shoulder protraction (Solem-Bertoft et al., 1993). Because the subacromial space is relatively small (Flatow et al., 1994), even a subtle change in dimension could result in compression of the subacromial tissues (Nordt et al., 1999; Graichen et al., 1999b) during glenohumeral elevation.

Scapular kinematics can be altered by various surrounding soft tissues and bone. Weak or dysfunctional scapular musculature (Ludewig and Cook, 2000; McQuade et al., 1998; Pascoal et al., 2000), fatigue of the infraspinatus and teres minor (Tsai, 1998), and changes in thoracic and cervical spine posture (Kebaetse et al., 1999; Ludewig and Cook, 1996; Wang et al., 1999) have all demonstrated a change in scapular kinematics. Theoretically, other potential causes of altered scapular kinematics are weak or dysfunctional rotator cuff musculature, soft tissue tightness about the scapula, bony morphology or soft tissue changes at the coracoacromial arch, and shoulder pain.

1.3. Tendon and bursa pathology

SAIS involves a degree of inflammation of the tendons or bursa of the subacromial space (Fu et al., 1991; Bigliani and Levine, 1997; Ogata and Uhthoff, 1990). This inflammation will cause a decrease in the overall volume of the subacromial space, potentially leading to increased compression of the tissues against the borders

of the subacromial space. Degeneration of the tendons of the subacromial space has been demonstrated in patients with SAIS, which may result from the inflammatory process or tension overload during shoulder activities (Paletta et al., 1997; Ogata and Uhthoff, 1990; Banas et al., 1995; Tuite et al., 1995; Toivonen et al., 1995).

1.4. Acromial morphology and shape

Dimensional changes in the subacromial space can be caused by variations in the architecture of the coracoacromial arch. One factor implicated is the acromion, specifically the morphology or the presence of osteophytes on the inferior aspect of the acromion or acromioclavicular joint. The morphology or shape of the acromion has been described in various manners. A widely used classification system for acromial shape is flat (type I), curved (type II), or hooked (type III), which was developed from observations of 139 shoulder specimens (Bigliani and Levine, 1997). Two studies have demonstrated a relationship between type III acromions and the degree of rotator cuff tearing (Bigliani and Levine, 1997; Toivonen et al., 1995), while two other studies have indicated no relationship (Banas et al., 1995; Farley et al., 1994). One potential reason for these conflicting results may be the poor level of inter-rater reliability of this acromial classification system (Jacobson et al., 1995; Haygood et al., 1994).

Several other methods of assessing acromial shape and curvature have been developed and have been demonstrated to be reliable. Significant relationships have been demonstrated between acromion morphology and patient's self report shoulder function (Vaz et al., 2000) and the severity of the rotator cuff pathology (Banas et al., 1995; Tuite et al., 1995; Prato et al., 1998). Although the correlation values between these measures were statistically significant, they were small in value indicating that acromial geometry does not account for all of the change in a patient's shoulder function or disease severity. Chronological age has also been demonstrated to be associated with rotator cuff disease severity and acromial morphology (Banas et al., 1995; Farley et al., 1994; Wang and Shapiro, 1997). It is unclear as to exact nature of the relationship between acromial morphology and rotator cuff disease severity.

Acromial geometry has also been linked to changes in subacromial pressure and abnormal contact with the tissues of the subacromial space. In hooked acromions as compared to flat or curved, there is increased subacromial pressure specifically at the inferior anterior lateral aspect (Payne et al., 1997), and greater contact throughout the range of motion with the tendons of the rotator cuff (Flatow et al., 1994). In patients with SAIS, distal clavicle and acromial resection resulted in significant decreases in subacromial pressure throughout glenohumeral elevation (Nordt et al., 1999).

Another possible culprit of encroachment into the subacromial space is the coracoacromial ligament. A thickened coracoacromial ligament can directly decrease the subacromial space, thereby causing decreased space for tendon excursion. A cadaver study demonstrated that forcible internal rotation with either glenohumeral elevation or cross-body adduction resulted in impingement of the rotator cuff at the coracoacromial ligament (Burns and Whipple, 1993). Additionally, a significant relationship has been demonstrated to exist between the presence of a thickened coracoacromial ligament and the incidence of rotator cuff tears (Ogata and Uhthoff, 1990; Farley et al., 1994; Soslowsky et al., 1996).

Other potential factors of the coracoacromial arch that may lead to subacromial impingement are the coracoid process and an unfused distal acromial epiphysis, or os acromiale. A deformity of the coracoid process that results in encroachment into the subacromial space can cause impingement. Impingement syndrome has also been demonstrated to be associated with the presence of an os acromiale (Hutchinson and Veenstra, 1993).

These findings suggest that the morphology or changes in the coracoacromial arch may result in compression of the structures of the subacromial space, thus contributing to SAIS. However, the question of whether degenerative changes in these tissues produce impingement or impingement produces degenerative changes remains to be answered. In either case, treatment aimed at removing these structures may reduce the impingement and produce a favorable outcome. However, it has been demonstrated that acromial decompression surgical intervention does not consistently produce a successful outcome in patients with SAIS (Neer, 1972; Gartsman, 1990). Additionally, non-operative treatment has been demonstrated to produce a comparable level of successful outcome as decompression surgery (Brox et al., 1993, 1999). This suggests that direct encroachment of the subacromial space by the coracoacromial arch soft tissue or bony changes is not be the only mechanism of impingement.

1.5. Posture: spine, shoulder, and scapula

Position and mobility of the thoracic spine can directly influence scapulothoracic and glenohumeral kinematics and thereby lead to impingement. A relatively small increase in thoracic spine flexion has resulted in a more elevated and anteriorly tilted scapula at rest, and less upward rotation and posterior tilt during glenohumeral elevation (Kebaetse et al., 1999; Culham and Peat, 1993). An increase in thoracic spine flexion has also resulted in a decrease in the amount of elevation of the glenohumeral joint (Kebaetse et al., 1999; Culham and Peat, 1993), and a decrease in the amount of force

generated at 90° of glenohumeral scapular plane abduction (Kebaetse et al., 1999).

Position and mobility of the cervical spine can also influence scapular and glenohumeral kinematics. Cervical spine flexion of 25° has been demonstrated to cause an increase in scapular upward rotation and a decrease in posterior tilting during glenohumeral elevation in healthy subjects (Ludewig and Cook, 1996). Patients diagnosed with overuse of their shoulder (indicative of SAIS) have demonstrated an increased forward head posture (increased upper cervical spine extension and lower cervical spine flexion), but no change in thoracic spine posture as compared to healthy subjects (Greenfield et al., 1995).

Forward shoulder posture is defined by Kendall et al. (1993) as a position of abduction and elevation of the scapula, which may appear as winging of the scapula, and medial rotation of the humerus (Kendall et al., 1993). In a lateral view plumb line analysis, the acromion process lies anterior to the plumb line, which is referenced by aligning it with the lobe of the ear. Theoretically, this posture may produce or result from soft tissue tightness anteriorly of the serratus anterior, pectoralis minor and upper trapezius, as well as muscular weakness of the middle and lower trapezius (Kendall et al., 1993). Soft tissue tightness and muscle weakness that occur with forward shoulder posture have been implicated as contributing factors to SAIS (Fu et al., 1991).

Alterations in scapular resting posture have been demonstrated in patients with SAIS of greater scapular anterior tilt (Lukasiewicz et al., 1999) and increased scapular winging and elevation (Warner et al., 1992) as compared to healthy controls. The description of the scapular winging appears to indicate a position of scapular internal rotation and anterior tilt. Scapular protraction, which also appears to be a combination of scapular internal rotation and anterior tilt, has demonstrated to be greater in patients with overuse syndrome as compared to healthy controls (Greenfield et al., 1997). Scapular protraction results in a reduction of the subacromial space, as compared to a retracted position, which is associated with non-slouched posture (Solem-Bertoft et al., 1993). It is noted that in one study, scapular resting posture in a group of construction workers with SAIS was not significantly different from healthy controls (Ludewig and Cook, 2000). However, spinal postural was not controlled for during measurements of scapular posture, which may explain why the groups did not differ in scapular resting posture.

Thoracic and cervical spine, shoulder and scapular posture may be linked together and described as upper quarter posture. The term "slouched posture" is used to describe the upper quadrant posture of increased thoracic spine flexion, forward head posture, and forward shoulder posture. Slouched posture may result in or from shortness of the tissues of the anterior shoulder and posterior upper cervical spine, and weakness of the posterior lower cervical spine and thoracic spine. This slouched posture can alter scapular and glenohumeral kinematics, potentially leading to abnormal subacromial pressure and dimensional changes of the space (Solem-Bertoft et al., 1993; Kebaetse et al., 1999; Ludewig and Cook, 1996; Culham and Peat, 1993).

1.6. Posterior capsule

Posterior capsular tightness may cause changes in glenohumeral kinematics leading to SAIS. When posterior capsular tightness was surgically induced in cadavers, there was an in increase in superior and anterior humeral head translations during passive glenohumeral flexion (Harryman et al., 1990a). Excessive superior and anterior humeral head translations can decrease the size of the subacromial space, leading to increased mechanical compression of the subacromial structures (Flatow et al., 1994; Brossmann et al., 1996).

One method to clinically assess posterior capsular tightness is to examine the degree of cross-body horizontal glenohumeral abduction. Harryman et al. (1990a) demonstrated that with this maneuver, there was an increase in anterior humeral head translation. It is important to note that these results were demonstrated during passive glenohumeral motion in a cadaver model, which may not apply to patients with posterior capsule tightness during active motion. However, these same authors followed up this study with one in which they placed intracortical pins into the humerus and scapula of two healthy subjects without signs of posterior capsular tightness, and the same pattern of motion was demonstrated as that with the cadavers (Harryman et al., 1990b). These studies may help to explain why patients have pain with this cross-body abduction maneuver, which is also used clinically to test the length of or stretch the posterior capsule.

An assessment of posterior capsular tightness is difficult, secondary to the inability to selectively isolate the posterior capsule from the posterior rotator cuff and deltoid. Warner et al. (1990) assessed tightness by measuring cross-body horizontal glenohumeral adduction, while Tyler et al. (1999) made this assessment by measuring glenohumeral horizontal adduction with the scapula manually stabilized. In either case, the posterior capsule was not isolated. In studies utilizing one of these techniques, tightness of the posterior capsular was demonstrated in patients with SAIS when compared to healthy controls (Warner et al., 1990; Tyler et al., 2000).

1.7. Rotator cuff musculature

The supraspinatus along with the other rotator cuff muscles of teres minor, infraspinatus, and subscapularis serve to maintain the congruent contact between the

humeral head and the glenoid fossa by producing a compressive force during glenohumeral movements. The latissimus dorsi and teres major, and to a lesser degree the rotator cuff musculature of the infraspinatus and the subscapularis impart an inferior translatory force to the head of the humerus to depress the humeral head (Halder et al., 2001). These secondary movers of the glenohumeral joint are critically important for the production of a smooth, coordinated movement of glenohumeral elevation. The rotator cuff also functions with the deltoid muscles to produce a smooth trajectory of the humerus during all phases of glenohumeral elevation (McMahon et al., 1995; Inman et al., 1944; Alpert et al., 2000). However, after the initial phase of elevation of approximately the first 30–60°, the rotary contribution of the supraspinatus declines significantly (Reddy et al., 2000). This may be due to a change in the length–tension relationship and a decrease in the moment arm of the supraspinatus with increased elevation (Reddy et al., 2000; Kuechle et al., 1997).

Dysfunctional or weak rotator cuff musculature has been well documented in patients with subacromial impingement (Brox et al., 1993, 1999; Warner et al., 1990; Reddy et al., 2000; Hawkins and Dunlop, 1995; Leroux et al., 1994; Bartolozzi et al., 1994). With a decrease in the contribution of the rotator cuff during glenohumeral elevation, the deltoid will be required to increase its contribution (Payne et al., 1997). An artificially induced disruption in the force-couple of the deltoid and supraspinatus has resulted in increased superior translation of the humeral head (Chen et al., 1999; Thompson et al., 1996; Sharkey and Marder, 1995; Deutsch et al., 1996; Paletta et al., 1997). A naturally occurring state of a dysfunctional rotator cuff, degeneration or tears of the rotator cuff tendons, has also resulted in increased superior humeral head translation (Poppen and Walker, 1976; Deutsch et al., 1996; Paletta et al., 1997; Yamaguchi et al., 2000; Pradhan et al., 2000). Rotator cuff muscle dysfunction in the form of fatigue can also lead to changes in scapular kinematics. Fatigue of the infraspinatus and teres minor has resulted in less scapular posterior tilt in healthy individuals (Tsai, 1998).

In patients with SAIS, a decrease in electromyographic activity of the infraspinatus and subscapularis during glenohumeral elevation from 30° to 60° was demonstrated as compared to healthy subjects (Reddy et al., 2000). In this range of glenohumeral motion, the rotator cuff musculature normally provides an inferiorly directed force to control the superior humeral translation that is occurring (Halder et al., 2001). Excessive superior translation of the humeral head resulting from rotator cuff weakness can theoretically lead to a decrease in the subacromial space during elevation, and thus increased mechanical compression of the subacromial contents. Evidence for this theory has been demonstrated with the opposite activity. With increased muscle activity of the rotator cuff or deltoid during glenohumeral elevation, healthy subjects demonstrated an increase in the acromio–humeral distance at 60° and 90° of glenohumeral elevation (Graichen et al., 1999b). In a dynamic shoulder model, a decrease in the subacromial pressure was observed with increased simulated supraspinatus activity (Payne et al., 1997).

Weakness or dysfunctional rotator cuff musculature can lead to changes in glenohumeral and scapulothoracic kinematics (Poppen and Walker, 1976; Chen et al., 1999; Thompson et al., 1996; Sharkey and Marder, 1995; Deutsch et al., 1996; Paletta et al., 1997; Yamaguchi et al., 2000; Pradhan et al., 2000; Tsai, 1998; Halder et al., 2001). These changes may result in increased mechanical compression of the structures of the subacromial space. It is uncertain if impingement syndrome causes dysfunctional muscle performance secondary to subacromial compression, or if the weakness causes the impingement syndrome to develop.

1.8. Scapular musculature

The scapulothoracic articulation is controlled by the musculature that is attached to the scapula, humerus, thoracic cage and spinal column. Scapular upward rotation is produced by the upper trapezius and lower serratus acting as a force couple during the initial phase of glenohumeral elevation (Bagg and Forrest, 1986; Wadsworth and Bullock-Saxton, 1997; Filho et al., 1991). In the middle phase of glenohumeral elevation, the lower trapezius increases its contribution (Bagg and Forrest, 1988); while in the final phase of glenohumeral elevation the lower and upper trapezius and the lower serratus are approximately equally active (Bagg and Forrest, 1986; Wadsworth and Bullock-Saxton, 1997). Production and control of scapular posterior tilting and external rotation have not been investigated.

An important role of the scapular musculature is to stabilize the scapula to support the base of the glenohumeral joint. With a decrease in the stabilization of the scapula by the surrounding musculature, a change in scapular position or motion may result (Ludewig and Cook, 2000; McQuade et al., 1998; Pascoal et al., 2000). The altered scapular position can change the length–tension relationship of the muscles attached to the scapula, specifically the rotator cuff. Theoretically, a dysfunctional rotator cuff can therefore result from alteration in the scapular position and scapular muscle strength.

Swimmers with impingement syndrome have demonstrated an increased variability in the onset of recruitment of the lower and upper trapezius and serratus anterior during glenohumeral elevation (Wadsworth and Bullock-Saxton, 1997). In construction workers with impingement syndrome, the upper and lower trapezius has demonstrated increased activity while the

serratus anterior has demonstrated decreased activity, and concurrent scapular kinematic changes of decreased upward rotation and increased anterior tilting and internal rotation during glenohumeral elevation (Ludewig and Cook, 2000). During glenohumeral elevation, the serratus anterior is required to work in concert with the trapezius to upwardly rotate the scapula to allow free movement of the subacromial structures under the coracoacromial arch (McQuade et al., 1998). Fatigue of the serratus anterior has resulted in an altered pattern of scapulohumeral rhythm in the range of 60–150° of glenohumeral motion (McQuade et al., 1998). Proper scapulohumeral rhythm is critical in this mid-range of glenohumeral motion, because that is the range in which impingement of the structures of the subacromial space will occur (Flatow et al., 1994; Brossmann et al., 1996). Changes in the timing and function of the upper and lower trapezius as well as the serratus anterior lead to changes in scapular kinematics, and thus most likely alter glenohumeral kinematics as well.

2. Conclusions

SAIS is the most common cause of shoulder pain, causing or resulting from multiple factors. The evidence indicates that glenohumeral and scapular kinematics are altered; increased anterior and superior humeral head translations and decreased posterior tipping, external rotation and upward rotation. Weakness or fatigue of the muscles that control these articulations and increased thoracic spine and cervical spine flexion and alterations of the shoulder girdle posture have also been demonstrated to be present in patients with SAIS. Postural, kinematic, and muscle changes have all been demonstrated to directly or indirectly alter the subacromial space dimension and relationships to the structures within the subacromial space. Changes in these relationships can also be brought about by architectural deviations of the subacromial space boundaries. These multiple factors are typically present in some combination, as opposed to a single factor presenting individually. A comprehensive assessment of all anatomical and biomechanical factors should be performed for all patients with SAIS, in order to design a treatment program that will have the greatest chance of a successful outcome. Future research is needed to further elucidate the mechanisms of SAIS and the relationships between the multiple factors implicated in this disorder.

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